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Prevention and management of iatrogenic flatback deformity

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Current Concepts Review

Prevention and Management of Iatrogenic Flatback Deformity

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- The most common cause of iatrogenic flatback syndrome is Harrington distraction instrumentation extending into the lower lumbar spine.
- Other common causes and exacerbating factors include failure to enhance regional lordosis during lumbar fusion for degenerative spondylosis, development of pseudarthrosis or postoperative loss of correction, development of kyphosis at the thoracolumbar junction, development of degeneration and decompensation cephalad or caudad to a prior fusion, and hip flexion contractures.
- Prevention of flatback syndrome involves preoperative assessment of sagittal balance, avoidance of distraction instrumentation and extension of long fusions into the lower lumbar spine, enhancement of physiologic lordosis during lumbar fusions, and intraoperative positioning with the hips extended.
- Treatment of flatback syndrome involves corrective pedicle subtraction or Smith-Petersen osteotomies with segmental instrumentation.
- Polysegmental osteotomies and vertebral column resection may be utilized in cases of sloping global sagittal imbalance and related severe coronal imbalance, respectively.
- Following surgical treatment, sagittal balance is generally improved with fair-to-good clinical outcomes, high patient satisfaction, and moderately high perioperative complication rates.

In 1973, Doherty[1] described a symptomatic fixed forward inclination of the trunk due to loss of normal lumbar lordosis following posterior spinal fusion for scoliosis, and he treated his patient with bilateral pelvic osteotomy. Shortly thereafter, Moe and Denis[2] coined the term "flatback syndrome" and reported their early results after treatment of that syndrome with vertebral extension osteotomies. Grobler et al.[3] subsequently defined the associated symptom complex and reported good short-term results following extension osteotomy in these same patients and thirteen others. Since then, flatback syndrome, which is also commonly known as fixed sagittal imbalance, has become a well-recognized entity. The etiology of flatback syndrome may be multifactorial, but the most common cause is iatrogenic loss of lumbar lordosis secondary to Harrington distraction instrumentation[4-10].

Farcy and Schwab[11,12] described a subgroup of similar patients who had what they termed "kyphotic decompensation syndrome" and "flat buttock syndrome," a fixed positive sagittal imbalance due to malalignment at the site of a spinal fusion to the sacrum performed with distraction instrumentation for etiologies other than scoliosis. However, the clinical presentation, root cause, and treatment of this entity are identical to those of the previously described classic flatback syndrome. Similarly, Booth et al.[13] divided their series of patients with flatback into a group with segmental (Type-I) loss of lumbar lordosis or lumbar kyphosis with maintenance of normal sagittal balance and a group with global (Type-II), or classic, flatback syndrome with a substantial fixed positive sagittal imbalance. For the purposes of this review, we will define flatback syndrome as a symptomatic postfusion condition attributable to severe loss of lumbar lordosis of any etiology. Ankylosing spondylitis, while not discussed in detail, will also be included, as many of the techniques utilized in the operative correction of flatback syndrome were originally described for the treatment of the progressive fixed kyphosis of Marie-Strümpell disease.

Etiology of Iatrogenic Flatback

The most common cause of loss of lumbar lordosis is degenerative disc disease due to aging. The most common reported cause of flatback syndrome is the extension of distraction instrumentation into the lower lumbar spine or sacrum[4-10,14-16]. It has also been reported following the use of anterior thoracolumbar compression instrumentation without structural graft support, but this is a rare occurrence[17]. The iatrogenic loss of lumbar lordosis was not a problem following fusion for the treatment of scoliosis prior to the advent of corrective sur-

TABLE I Etiology of Flatback Deformity

Distraction (Harrington) instrumentation to lower lumbar spine or sacrum
Hypolordotic lumbar fusion for degenerative spondylosis
Other malaligned fusion
Pseudarthrosis with progression of deformity or loss of correction
Kyphosis at thoracolumbar junction
Decompensation of inferior or superior adjacent segments secondary to inadequate scoliosis fusion or segmental degeneration
Hip flexion contractures or extensor weakness

gical instrumentation[18]. With Harrington instrumentation, the combination of a straight rod and distractive forces causes an obligatory loss of lumbar lordosis with subsequent anterior translation of the vertical axis and the body's center of gravity. As the resulting imbalance is typically rigid as a result of the fusion mass and supporting instrumentation, the patient attempts to compensate locally by hyperextending any segments not included in the instrumentation as well as the cervical spine and by flexing the hips and knees in order to stand upright or see straight ahead.

The loss of lordosis correlates directly with more caudad extension of instrumentation. In a study of ninety-six patients with scoliosis who were treated with Harrington instrumentation, Aaro and Ohlen[4] found the lumbar lordosis to average 38° when the implant stopped at T12 but only 21° when the fusion was to L4 and 16° when it was to L5. Other authors have reported similar findings[8,14]. In addition, LaGrone et al.[6] noted decreased lordosis in patients with a fusion to the sacrum and no problems in those with instrumentation cephalad to L3. Kostuik and Hall[19] found that twenty-two (49%) of forty-five adult patients with scoliosis who had a fusion to the sacrum with distraction instrumentation manifested a substantial loss of lumbar lordosis, and thirteen of the twenty-two (29% of the total number of patients) required one or more corrective procedures. van Dam et al.[10] reviewed the results in ninety-one adult patients treated with posterior spinal fusion with Harrington instrumentation and noted that 43% of those with instrumentation to L4 or L5 demonstrated marked loss of lordosis but that symptomatic sagittal imbalance developed in only two patients. Thus, the prevalence of flatback syndrome increases with more caudad instrumentation, and its frequency depends on the severity of the loss of lumbar lordosis.

While distraction instrumentation is classically the primary cause of sagittal imbalance, several other important contributing factors have been identified (Table I). With the increasing performance of lumbar fusions for the treatment of degenerative spondylolisthesis, scoliosis, and stenosis with instability, a different type of flatback with fixed sagittal imbalance is becoming more common[13,20-22]. Failure to maintain (or ideally to enhance) lumbar lordosis during a fusion of a degenerated spine can result in an inability to compensate locally, with accelerated adjacent degeneration and loss of sagittal balance[23-27]. As these patients lose additional lordosis as a consequence of degeneration and aging, kyphotic decompensation may occur, presenting with fixed forward inclination and symptoms attributable to flatback syndrome. Degenerative loss of lumbar lordosis and flatback syndrome in the absence of prior instrumentation or surgery, termed "lumbar degenerative kyphosis," has been reported to be a common problem in Asian populations[28,29]. Greater attention to sagittal alignment and enhancement of lumbar lordosis are therefore warranted during all lumbar fusions.

Pseudarthrosis following posterior spinal fusion is also a common etiologic factor[6,7,20,30]. LaGrone et al.[6] noted that pseudarthrosis contributed to the deformity in eleven of fifty-five patients treated for symptomatic flatback syndrome. Similarly, Cummine et al.[30] found pseudarthrosis in all five patients with loss of lumbar lordosis in their series of fifty-nine adult patients treated with revision Harrington fusion for adult scoliosis. Furthermore, particularly large cantilever loads are transmitted to the lumbar and sacral instrumentation following fusion to caudad levels, and an increased pseudarthrosis rate is noted after such fusions[9,31]. These biomechanical stresses and the risk of subsequent pseudarthrosis may be increased with progressive loss of sagittal balance; thus, pseudarthrosis may be both a causative factor and a complication of flatback syndrome, especially when lordosis is not restored with operative treatment.

Progressive or fixed thoracic and lumbar kyphosis may also contribute to iatrogenic flatback deformity. The thoracolumbar junction may be susceptible as a result of the anatomic alignment transition from the costally supported thoracic vertebrae to the unsupported lumbar spine, and from the regional thoracic kyphosis to the lumbar lordosis. In addition, 80% to 90% of the normal standing axial load in the lumbar spine is transmitted by the anterior column[32]. The magnitude and moment arm of these stresses increase with underlying fixed positive sagittal imbalance. Thoracolumbar kyphosis may be preexisting as a result of thoracolumbar or lumbar scoliosis or a post-traumatic etiology. In scoliotic deformities, thoracic or thoracolumbar hyperkyphosis often develops secondary to lumbar hyperlordosis. In such a setting, even small decreases in lumbar lordosis may affect sagittal balance[7,21]. Alternatively, thoracolumbar kyphosis may be caused by poor contouring of the rod[6]. Inadequate lordotic compensation of any remaining mobile lumbar segments has also been observed in patients with moderately decreased lumbar lordosis following treatment with Harrington instrumentation[8].

Another etiologic factor contributing to the development of flatback syndrome is decompensation by adjacent segments cephalad or caudad to a fusion mass[20,21,33]. This may occur because of a poor selection of fusion levels or increased stresses on the adjacent segments. Similarly, patients with a fusion extending into the upper thoracic spine are unable to compensate for decreased lumbar lordosis with thoracic hypokyphosis[7].

Finally, hip extensor weakness and/or flexion contractures may contribute to flatback deformity, as hip hyperexten-

sion is the physiologic method of compensation for lumbar hypolordosis[15]. Hip extensor weakness, as seen in patients treated for postpoliomyelitic or myelomeningocele spinal deformities, may compromise this compensatory function and contribute to flatback syndrome[16]. Hip flexion contracture, however, is more likely the result, rather than the cause, of flatback syndrome, as patients must walk with both hips and knees flexed in order to remain upright[21,34]. Nonetheless, failure of the surgeon to appreciate and address the magnitude or severity of a hip flexion contracture or of increased pelvic tilt preoperatively may compromise the results following an otherwise successful operative correction of fixed sagittal imbalance[35,36].

Clinical Presentation

Patients with flatback syndrome typically present with painful loss of lumbar lordosis resulting in forward inclination of the trunk with difficulty standing erect with the knees extended (Fig. 1). The pain is generally associated with fatigue and may affect the cervical, upper thoracic, or lower lumbar regions. Often, there is a history of multiple spinal operations. Physical examination reliably demonstrates obligatory flattening of the lumbar region and forward tilting of the trunk. In an effort to compensate for this fixed sagittal imbalance, the patient flexes the knees and attempts to hyperextend the cervical spine and any remaining mobile vertebral segments in the thoracic and lumbar spine. Biomechanical studies have demonstrated that increased paraspinal muscular forces are required to maintain an erect posture when there is loss of normal lumbar or cervical lordosis, which may contribute to the fatigue-related etiology of the symptoms[37,38]. Furthermore, attempts to compensate and maintain a horizontal gaze may result in increased strain, pain, and degenerative changes within the cervical spine or unfused lower lumbar discs, which may require operative treatment. Degenerative cervical changes have been documented in >50% of patients with long-term follow-up after surgery for adolescent idiopathic scoliosis[14]. Pain or tenderness in the fused lumbar region or at the thoracolumbar junction of the spine may indicate a pseudarthrosis[7].

Hip flexion contractures are frequently apparent on examination, and pelvic tilt may be abnormal[15,35]. The pelvic tilt is in the sagittal plane, with either anterior or posterior pelvic inclination. Patients with increased anterior pelvic tilt may have a less satisfactory result of operative treatment, with persistent stooping despite the restoration of adequate lumbar lordosis[35]. Sarwahi et al.[34] performed gait analysis on twenty-one patients with flatback syndrome and found decreased step and stride length as well as gait velocity. Cadence was maintained compared with that of normal controls, whereas the stance phase and hip and knee flexion were increased. Patients may have knee or quadriceps pain and fatigue. Patients with severe progression of thoracolumbar or thoracic kyphosis following a lumbar fusion with distraction may manifest or report cardiopulmonary or digestive abnormalities; however, this is unusual in patients with iatrogenic flatback syndrome. True tension signs (e.g., ipsilateral or crossed straight-leg raise) and radicular pain are also unusual, but they may occur in the setting of fixed sagittal imbalance associated with or due to spinal degeneration with stenosis or degeneration of adjacent segments below a caudad fusion[20,35].

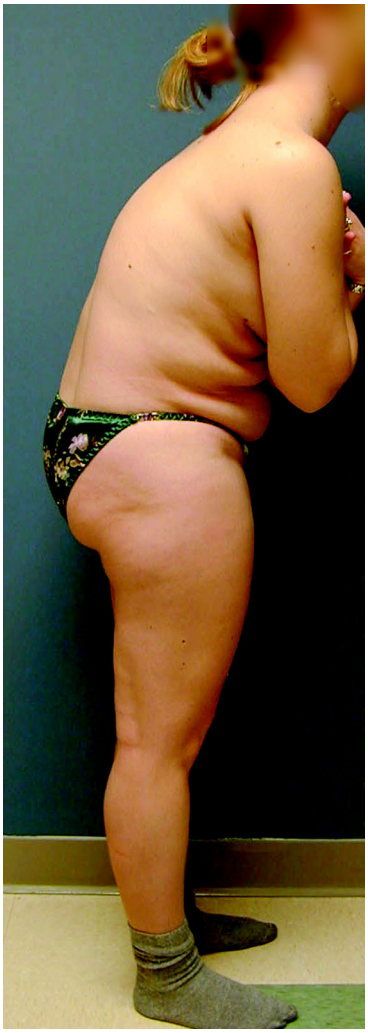

Fig. 1
Lateral clinical photograph of a patient with flatback syndrome. Note the total loss of normal lordosis and the fixed forward inclination of the trunk.

Radiographic Evaluation

Normal Sagittal Alignment

Discussion and assessment of pathologic sagittal imbalance requires a clear understanding of normal sagittal alignment. Several investigators have evaluated normal sagittal curvature in the thoracic and lumbar spine as well as at the thoracolumbar junction. Reported mean values have ranged from 37° to 42° for normal thoracic kyphosis (as measured from the superior end plate of T3 to the inferior end plate of T12 with the Cobb method[39]) and 50° to 75° for normal lumbar lordosis (as measured from the superior end plate of L1 to the superior end plate of S1 with the Cobb method)[40-43]. However, normal functional ranges vary widely, and these ranges (rather than averages) should be considered during the radiographic evaluation

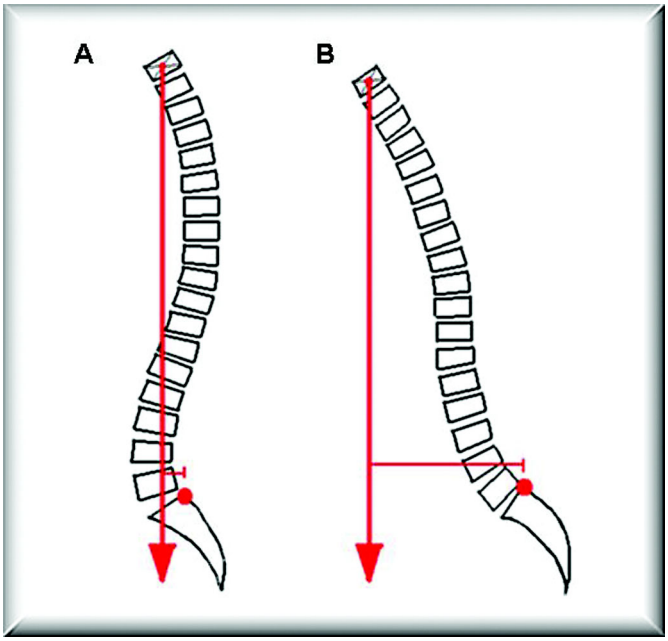

Fig. 2

Schematic representation demonstrating the technique for assessing the sagittal vertical axis in a spine with normal alignment (A) and in a patient with fixed sagittal imbalance (B). A plumb line is dropped vertically from the center of the C7 body, and the horizontal linear distance between the line and the posterior-superior part of the sacral end plate is measured. A normal value is a distance of ±3 cm.

of sagittal plane deformity. It is generally accepted that normal thoracic kyphosis ranges from 20° to 50° and normal lumbar lordosis ranges from 20° to 65° with increasing fractional lordosis in the caudad segments[40,43]. Some authors have advocated measuring lumbar lordosis from the superior end plate of L1 to the inferior end plate of L5 because of the wide variations in sacral inclination and the difficulties of accurately assessing the S1 end plate radiographically, whereas other authorities have recommended measuring from the superior end plate of T12 to S1[40,41]. However, as up to 60% of the segmental lordosis occurs in the caudad two motion segments, measurement from the sacrum is more appropriate[41]. The thoracolumbar junction should be nearly straight. Decreases in normal lumbar lordosis of up to 20° have been recognized as a consequence of aging and degeneration[41,44]. Smaller decreases in lumbar lordosis have been observed in patients with low-back pain[45].

More critical than absolute measurements in the assessment of sagittal alignment is the overall sagittal balance, particularly given the wide range of normal values for both thoracic kyphosis and lumbar lordosis. The sagittal vertical axis is best assessed by dropping a plumb line from the center of the C7 body to the sacrum. Normally, this axis should fall through the S1 disc space, preferably at the posterior-superior aspect of the S1 end plate (Fig. 2)[41,45,46]. Measurements of the total sagittal vertical axis, or the sagittal plumb line, should be performed from the posterior-superior aspect of S1. A plumb line falling posterior to S1 is considered to represent negative sagittal balance, whereas an anteriorly shifted line is considered to represent positive sagittal balance. Vedantam et al.[47] demonstrated a 4-mm posterior shift of the sagittal vertical axis with the arms raised horizontally with the shoulders in 90° of flexion, as opposed to 30° of forward flexion; those authors therefore recommended positioning the shoulders at 30° of flexion to prevent this negative shift when the sagittal vertical axis is assessed radiographically.

Flatback Syndrome

Radiographic evaluation of a patient with fixed sagittal imbalance and suspected flatback syndrome should begin with a full-length standing lateral radiograph of the spine made with the patient's hips and knees extended[7]. Patients may have difficulty standing upright in this position and may require support, but failure to extend the knees may result in artificial translation of the sagittal vertical axis. The normal sagittal vertical axis should ideally fall within 2.5 cm of the anterior aspect of the sacrum[11,41]. Patients with flatback syndrome may be seen to have substantial flattening or even kyphosis of the lumbar segments (Figs. 3-A and 3-B). Preoperative sagittal imbalance as great as 29.5 cm has been reported, with averages ranging from 4.3 to 14.5 cm in patients with flatback[13,22,33,48,49].

Flexion and extension radiographs may be helpful in the assessment of the mobility of the remaining, unfused segments in the thoracic and lumbar spine, but compensation through these segments is, by definition, inadequate in patients with flatback syndrome. Supine oblique radiographs and computed tomography may be useful for assessing and confirming the presence and location of a pseudarthrosis as a factor contributing to the pain and deformity. Finally, full-length standing anteroposterior radiographs of the spine should be made to look for any associated coronal plane imbalance, so that this can also be addressed at the time of surgery if necessary. Awareness of the preoperative coronal status is critical, as this deformity can worsen as a result of corrective osteotomies addressing sagittal imbalance[20].

Prevention of Iatrogenic Flatback Syndrome

Preoperative Planning

With complication rates after revision spinal fusion reported to be as high as 60% (thirty-three of fifty-five patients) and with up to 47% of these patients having residual sagittal deformity at the time of follow-up, treatment of iatrogenic flatback must begin with prevention (Table II)[6]. Preoperative assessment aimed at the prevention of postoperative flatback defor-

TABLE II Prevention of Flatback Syndrome

Appropriate preoperative assessment of sagittal alignment
Limitation of caudad extent of fusion when practical
Utilization of segmental instrumentation/avoidance of distraction with maintenance or enhancement of physiologic lumbar lordosis and sagittal balance
Intraoperative positioning with hips extended

mity is logical and relatively straightforward. Before a patient undergoes a long spinal fusion, he or she should have adequate and accurate radiographic assessment of the preoperative sagittal curvature and balance as well as of rotational and coronal plane deformities. The operation should be planned so that the magnitude of the existing thoracic and lumbar curves is maintained or corrected to the greatest degree possible and sagittal balance is either maintained or restored. In short-segment fusions for treatment of degenerative lumbar conditions, the focus should be on increasing, rather than maintaining, lumbar lordosis in anticipation of further degeneration and loss of regional curvature[23-26]. For patients with scoliosis, obtaining normal postoperative sagittal balance may be more critical functionally than is restoring so-called normal thoracic kyphosis and lumbar lordosis. Caudally, the fusion should be stopped at or cephalad to L3 except when a more caudad level is absolutely required to prevent curve progression or decompensation[4-8]. In addition to decreasing the risk of postoperative loss of lumbar lordosis, saving caudad fusion segments in the lumbar spine may decrease the risks of pseudarthrosis and late low-back pain, retrolisthesis, and degeneration of adjacent segments caudad to the fusion mass[9,14,18,50,51]. At the time of a ten-year follow-up, Cochran et al.[14] noted subjacent retrolisthesis in fifteen (63%) of twenty-four patients with a fusion to L4 or L5; all fifteen had low-back pain, and eleven had degenerative changes.

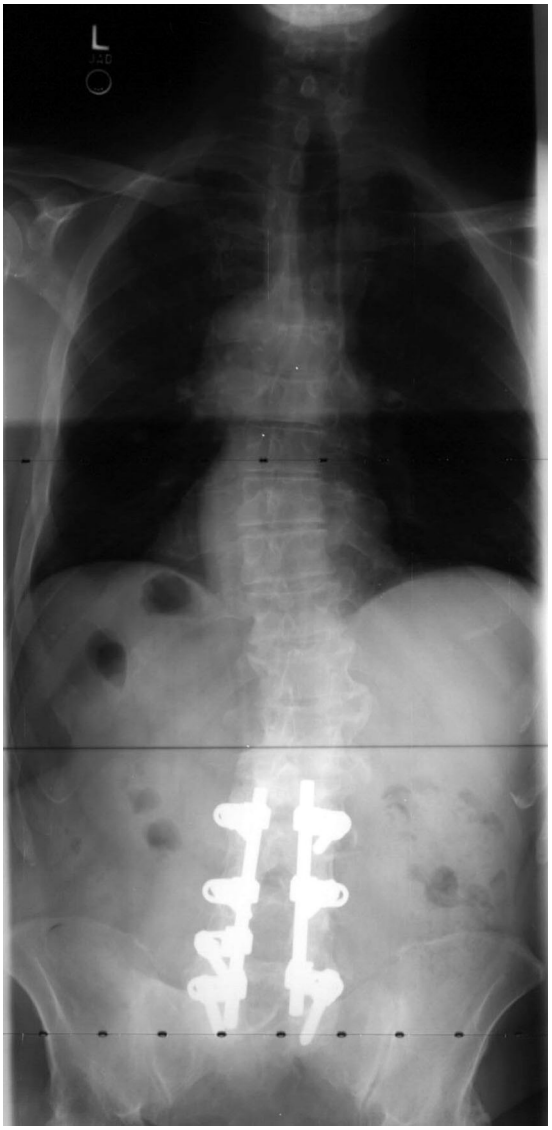

Fig. 3-A

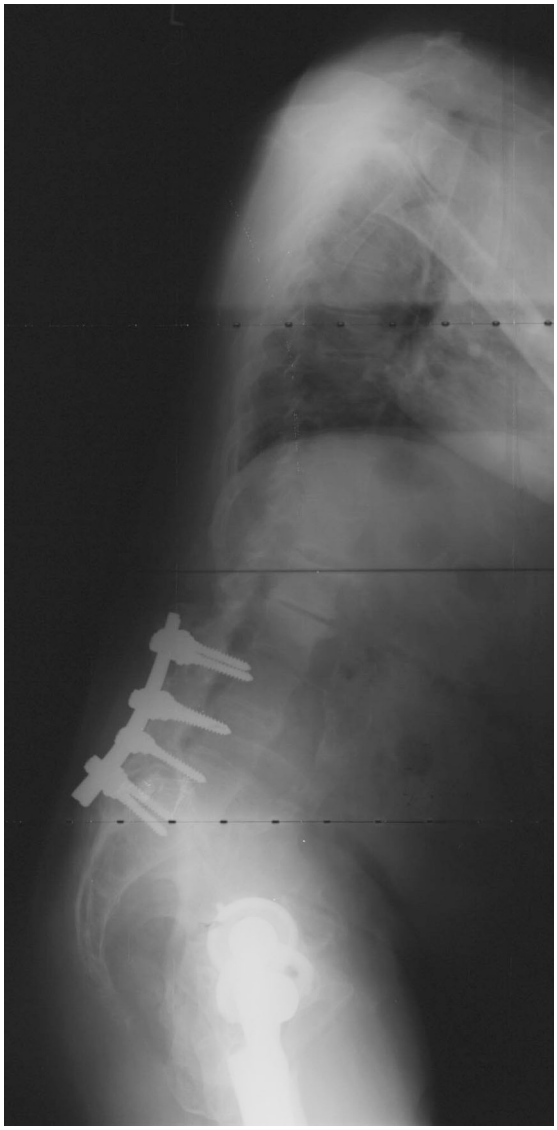

Fig. 3-B

Preoperative anteroposterior (Fig. 3-A) and lateral (Fig. 3-B) radiographs of a patient with flatback syndrome due to a malaligned lumbar fusion. Note the mild coronal imbalance on the anteroposterior radiograph and the severe sagittal imbalance on the lateral radiograph. The deviation of the sagittal vertical axis on the lateral radiograph measures 17 cm, demonstrating profound imbalance.

Intraoperative Positioning

Surgical positioning is critical to maintaining lumbar lordosis and preventing flatback syndrome following long spinal fusion. If the patient is positioned in a manner that decreases lumbar lordosis or kyphosis, it may be difficult or impossible to restore lordosis adequately through rod contouring or implant manipulation intraoperatively. Several authors have addressed the effect on lumbar lordosis of positioning on various types of surgical frames and tables. Benfanti and Geissele[52] demonstrated, in a study of thirteen anesthetized patients, that 95% of the lordosis was maintained when the patient was positioned on a Wilson frame with the hips in full extension and that there was a 26% decrease in the lordosis when the hips were flexed an average of 33°. In a study of 101 patients who had a mean preoperative standing lordosis of 45.2° before undergoing spinal surgery, Guanciale et al.[53] found that fifty-one patients who were positioned on an Andrews table with the hips flexed 90° had a significant decrease in lordosis ($p < 0.005$), to an average of 32.8°, whereas fifty patients who were positioned on a four-poster frame demonstrated a mean lordosis of 47.7°, a slight increase compared with the preoperative value.

Peterson et al.[54] compared twenty patients who were positioned with the hips flexed 90° on a Hastings frame with twenty others who were positioned with the hips extended on a Jackson table. They found that hip flexion resulted in considerable decreases (35%) in both segmental and total lumbar lordosis, whereas the prone position with the hips extended increased segmental lordosis by 22% at the L5-S1 levels and preserved the total lordosis and segmental lordosis at all other levels. Finally, Stephens et al.[55] assessed the lordosis of ten volunteers while they were standing, while they were prone on a Jackson table with the hips in extension, and while they were positioned on an Andrews table with the hips flexed both 60° and 90°. Positioning on the Jackson table with the hips extended resulted in a small increase in lumbar lordosis, whereas the position on the Andrews table produced decreases of 67% and 47% with the hips flexed 90° and 60°, respectively. Therefore, to preserve physiologic lumbar lordosis, positioning with the hips in extension should be an important consideration in all lumbar fusion surgery.

Posterior Spinal Fusion

As noted, the primary cause of the vast majority of cases of iatrogenic flatback syndrome is loss of lumbar lordosis secondary to the use of distraction (Harrington) instrumentation in the lumbar spine[1-10,14-16]. Following the initial recognition of this complication, there were several attempts to modify the Harrington instrumentation, such as with the use of a Moe squared-end rod to prevent rotation following contouring, use of Harrington compression instrumentation on the convex side of scoliotic curves, and the "rod-long, fuse-short"[15] technique. These modifications have been proven to be inadequate[4,5,10,15,56]. Therefore, the use of distraction instrumentation caudad to the level of L1 or L2 is still highly inadvisable, even with the implant modifications, as a result of the substantial risk of reducing the lumbar lordosis with variable production of a symptomatic flatback deformity.

Fortunately, there have been substantial advancements in spinal implants in the last two decades. Foremost among these was the advent of segmental spinal instrumentation. As early as 1982, biomechanical analysis demonstrated that, compared with Harrington instrumentation, Luque instrumentation with segmental sublaminar wiring provided superior fixation, less risk of loss of lumbar lordosis and thoracic kyphosis, and superior rotational control and resistance against construct failure[57]. In the same year, Luque[58] reported his initial results of treatment of sixty-five consecutive patients with scoliosis and noted improved curve correction. Although Luque did not present significant outcome data on sagittal plane correction and balance, he hypothesized that segmental instrumentation of this type may help to improve the magnitude of correction and the rigidity of the construct while preserving sagittal curvature. This was subsequently confirmed by Kostuik and Hall[19] as well as by Phillips and DeWald[59], who noted better preservation of lumbar lordosis with Luque instrumentation.

Segmental hook instrumentation has also been shown to be highly beneficial, and superior radiographically, in this regard. In a study of 160 patients with scoliosis treated with Cotrel-Dubousset instrumentation, Bridwell et al.[44] noted preservation of lumbar lordosis following fusion surgery, with preoperative values averaging 44° and 34° and postoperative values averaging 46° and 33° for children and adults, respectively. The authors concluded that Cotrel-Dubousset instrumentation can preserve and potentially enhance lumbar lordosis. Similarly, Takahashi et al.[51] reported no differences in the lumbar lordosis seen preoperatively, postoperatively, and at the time of follow-up in thirty patients with adolescent idiopathic scoliosis treated with segmental hook instrumentation. Finally, de Jonge et al.[60] reported the outcomes in a large series of 306 patients with adolescent idiopathic scoliosis who had undergone surgical correction with Cotrel-Dubousset instrumentation. They reported that normal lumbar lordosis was preserved in 97.9% of the patients with normal preoperative lordosis and in 94.4% of those with preoperative hypolordosis. However, it should be noted that mere maintenance of lumbar lordosis may be inadequate; enhancement of the lumbar lordosis may be necessary to restore sagittal balance in patients with adolescent idiopathic scoliosis in whom thoracic hypokyphosis has also been surgically corrected.

Anterior spinal fusion is an alternative method of preserving motion segments and avoiding distraction across the lumbar spine to maintain lordosis. Rodts and DeWald[61] reported the findings on follow-up in eighteen of fifty-four patients in whom idiopathic thoracolumbar and lumbar scoliosis had been treated with anterior Zielke instrumentation. They noted an 18% improvement in lumbar lordosis, with the patients with an anterior arthrodesis requiring fusion of an average of 4.7 vertebrae compared with an average of nine fused vertebrae in similar patients who had required posterior arthrodesis. When anterior fusion techniques are used, care must be taken to ensure appropriate structural graft support

to avoid loss of lordosis through compression and collapse of the anterior column[46].

More recently, segmental pedicle-screw instrumentation has gained popularity because of its potential for achieving solid three-column fixation and improving rotational control of the scoliotic spine. In a study of thirty-two consecutive patients with adolescent idiopathic scoliosis, Liljenqvist et al.[62] compared the results of treatment with mainly hook constructs with the results of treatment with mainly screw constructs. They found improved curve correction and sagittal alignment with the predominantly screw constructs. Moreover, Suk et al.[63] compared the results in seventy-eight patients with adolescent idiopathic scoliosis who had been treated with hooks, hook-patterned pedicle screws, or segmental screws. They reported significantly greater curve correction, rotational improvement, and sagittal alignment (all $p \leq 0.01$) with the pedicle-screw constructs and the best radiographic results with the segmental screws. In both of these series, pedicle-screw instrumentation was found to be superior to hooks and to be safe in both the thoracic and the lumbar spine with a low prevalence of complications.

Prevention of flatback syndrome when spinal instrumentation is placed for long fusions extending into the lumbar spine depends on adequate preoperative assessment and planning, appropriate positioning that maintains lordosis through hip extension, and placement of appropriately contoured segmental instrumentation. The evolution of posterior instrumentation has progressed from Harrington instrumentation and modifications of that technique to Luque segmental wiring and ultimately to Cotrel-Dubousset segmental hooks and segmental pedicle screws. Each iteration of implant development has produced greater curve correction, construct rigidity, and maintenance of sagittal balance. We currently recommend placement of segmental pedicle screws in the lumbar spine to achieve optimal lordosis and construct strength caudally and placement of segmental screws or hooks in the thoracic spine; interbody spacers can also be used at the caudad construct in long fusions if necessary. Furthermore, thoracic pedicle screws are rapidly gaining popularity as a result of their demonstrated efficacy and safety.

Nonoperative Treatment of Flatback Syndrome

In general, nonoperative treatment of flatback syndrome has been disappointing. A trial of hip-extension, trunk-stabilization, and back-extension exercises supplemented with bracing and nonsteroidal anti-inflammatory medications may improve the functional condition of the patient preoperatively. However, these measures have generally proven to be of little lasting benefit[7,11,12].

Farcy and Schwab[11,12] conducted the only structured study of nonoperative treatment of flatback syndrome of which we are aware. Of forty-eight patients with symptomatic flatback who were initially managed with intensive physical therapy, twenty-eight (58%) ultimately required osteotomies for operative correction of fixed deformities and sixteen of the remaining twenty patients underwent implant removal without decompression or operative correction because it was suspected that the implants were causing symptoms. Only thirteen (27%) of the forty-eight patients were ultimately considered to have had a long-term successful result of management without operative realignment. The mean sagittal imbalance in the twenty patients in whom operative correction with osteotomies was not undertaken was only 3.4 cm, slightly greater than the normal range of the sagittal vertical axis reported by Gelb et al.[41]. These patients might therefore be better classified as having symptomatic loss of lumbar lordosis without true fixed sagittal imbalance. Alternatively, patients with a good response to nonoperative treatment may be similar to patients with Type-I flatback deformity, with substantial loss of segmental lordosis but retention of sagittal balance, as described by Booth et al.[13]. The challenge when nonoperative management fails for patients with Type-I, or segmental, flatback deformity is restoration of the normal spinal curvature while maintaining the normal preoperative sagittal balance. Regardless, the results of nonoperative management of flatback syndrome are frequently disappointing, and once nonoperative treatment has failed or the deformity has progressed, operative correction is indicated.

Operative Treatment of Flatback Syndrome

Preoperative Planning

The goal of corrective surgery in the treatment of flatback syndrome is to restore physiologic lordosis and sagittal balance such that the sagittal vertical axis intersects the posterior aspect of the sacrum. This permits standing and walking with the hips and knees in a physiologic posture, improves function, and reduces fatigue-associated back and neck pain. Preexisting pseudarthrosis may compromise the results of corrective surgery because of persistent pain or loss of correction and should be addressed during the same procedure. For patients with a segmental-type flatback deformity and a normal preoperative sagittal vertical axis, the challenge is to restore lordosis and relieve pain while maintaining sagittal balance[13].

Decision-making regarding the type and location of corrective osteotomies depends on the site of the deformity and the presence and location of the pseudarthrosis. Although a preoperative pseudarthrosis may predispose a patient to postoperative recurrence of deformity at the same site[20], it makes intuitive sense to obtain correction at this site and increase the compressive forces across it with instrumentation to enhance osseous union. In general, corrective osteotomies should be performed at the site of maximal deformity. In the lumbar spine, the osteotomy typically can be carried out at L2 or caudad, where physiologic lordosis is increased, reducing the risk of conus medullaris or spinal cord injury. For patients with preexisting thoracolumbar kyphosis or decompensation of adjacent cephalad segments and rigid deformities, osteotomy may be required at higher levels. However, if the deformity is flexible, correction should be obtained caudally and the instrumentation and fusion should be extended cephalad to achieve sagittal balance.

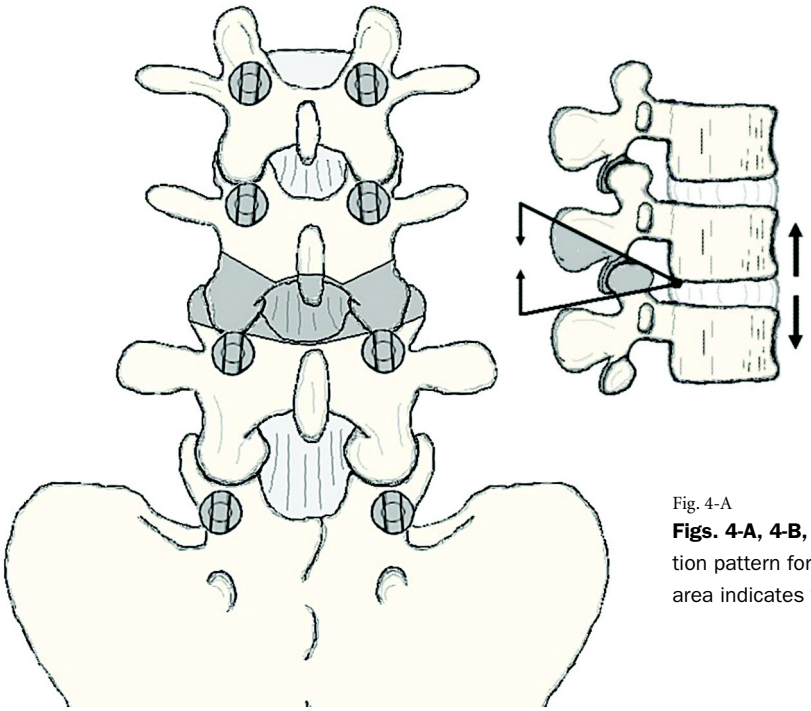

Fig. 4-A

Figs. 4-A, 4-B, and 4-C Schematics demonstrating the bone resection pattern for a Smith-Petersen osteotomy. **Fig. 4-A** The shaded area indicates the site for the osteotomy.

Extension (Smith-Petersen) Osteotomy

In 1945, Smith-Petersen et al.[64] described and performed the first posterior spinal osteotomy for correction of sagittal deformity (Figs. 4-A, 4-B, and 4-C). This procedure, which was modified by Law[65] and others[21,36,66-68], entails resection of the posterior elements at the desired level of correction with undercutting of the adjacent spinous processes. Sagittal correction is then achieved through posterior compression with

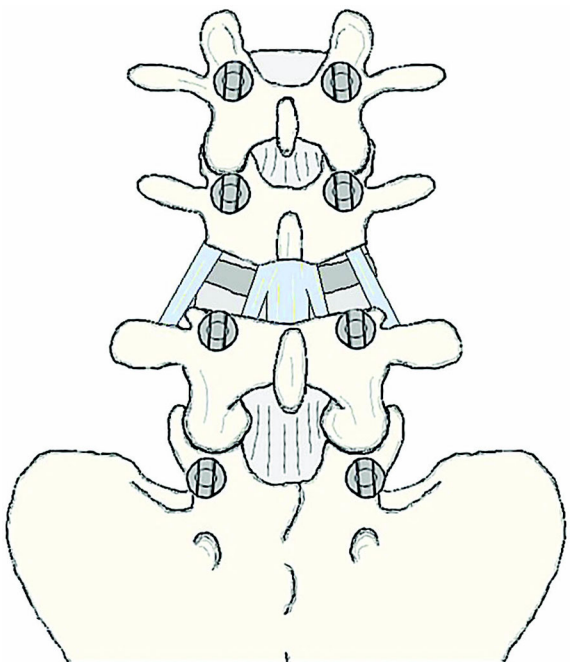

Fig. 4-B

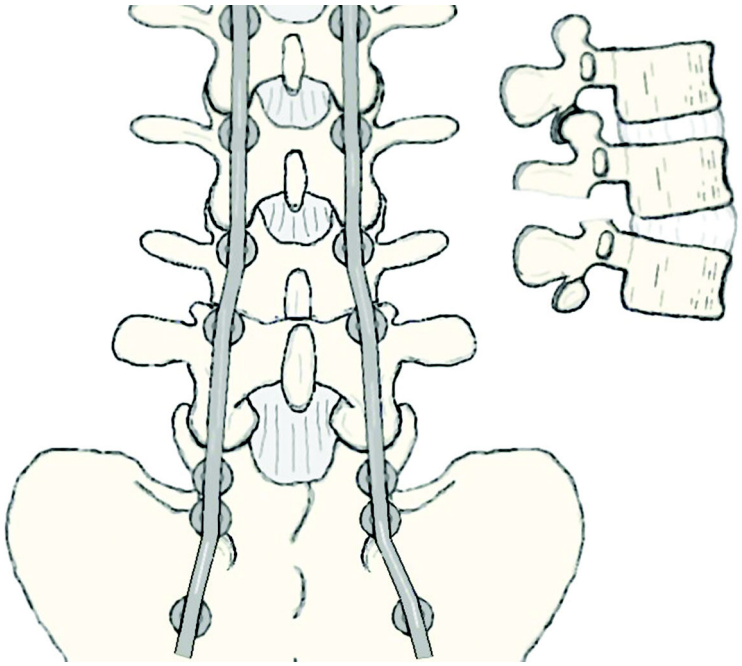

Fig. 4-C

Fig. 4-B Note the oblique nature of the bone resection following the initial osteotomy. Following this resection, undercutting of the inferior and superior adjacent laminae and wide foraminal decompression is performed in order to ensure adequate space for the neural elements prior to closure of the osteotomy. **Fig. 4-C** After final placement of the instrumentation and closure of the osteotomy, the anterior column is lengthened as a result of disc distraction, osteoclasis, or combined anterior release and osteotomy in order to obtain sufficient correction.

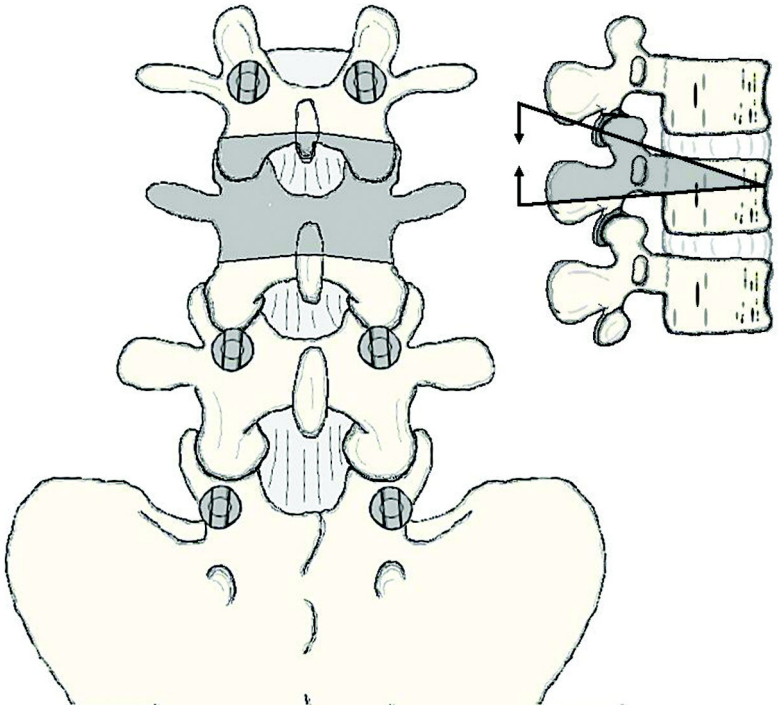

Fig. 5-A

Figs. 5-A, 5-B, and 5-C Schematics demonstrating the bone resection pattern for a pedicle subtraction osteotomy. **Fig. 5-A** The shaded area indicates the site for the osteotomy.

instrumentation, resulting in anterior osteoclasis through the vertebral body or distraction through the anterior longitudinal ligament and disc space. An important drawback of this procedure is that it lengthens the anterior column and may destabilize the spine if the instrumentation fails prior to fusion. For this reason, La Chapelle[66], Herbert[68], and others[11,12,21,67] described anterior release, discectomy, or osteotomy and structural grafting in conjunction with posterior Smith-Petersen osteotomies. Additionally, lengthening of the anterior column may result in traction injury of one or more of the great ves-

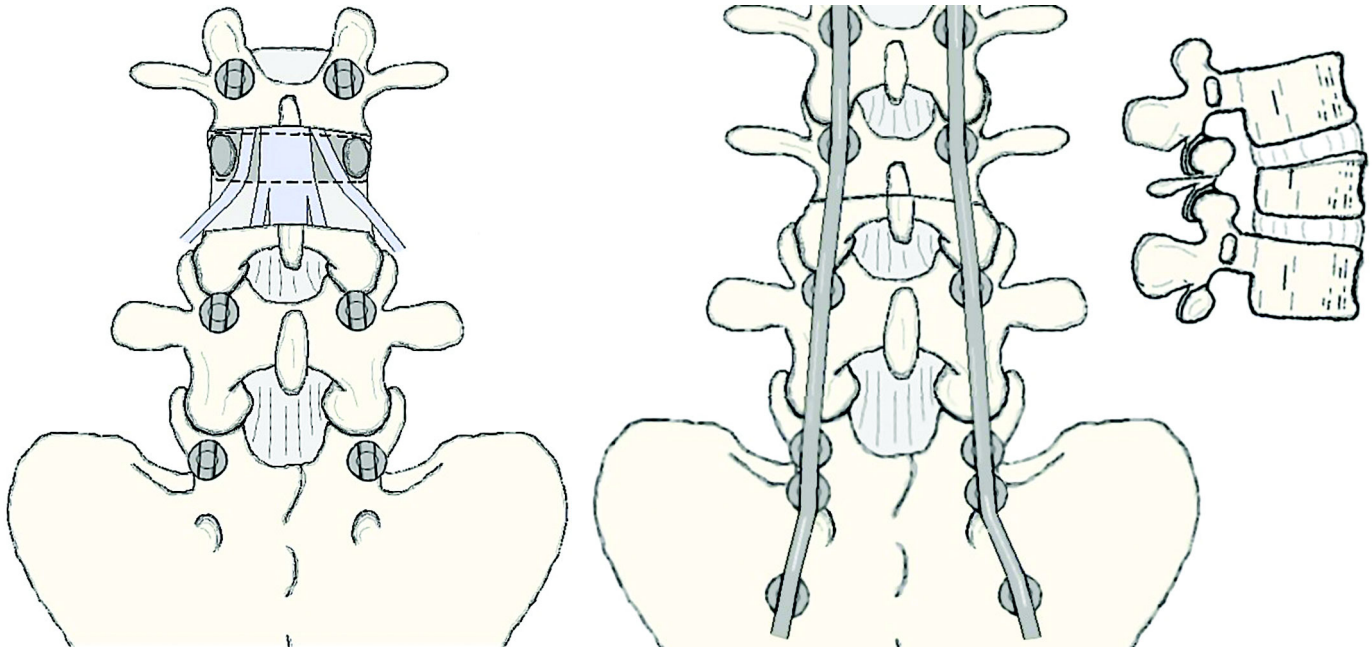

Fig. 5-B

Fig. 5-C

Fig. 5-B Following removal of the posterior elements, the pedicles are isolated but the medial wall is preserved initially to protect the neural elements and dura during the vertebral body decancellation. **Fig. 5-C** After the instrumentation has been placed securely, the pedicle and vertebral wall resections are completed and the osteotomy is closed in a controlled fashion. Note the absence of anterior column lengthening as the correction rotates about the middle column, hinging on the anterior cortex.

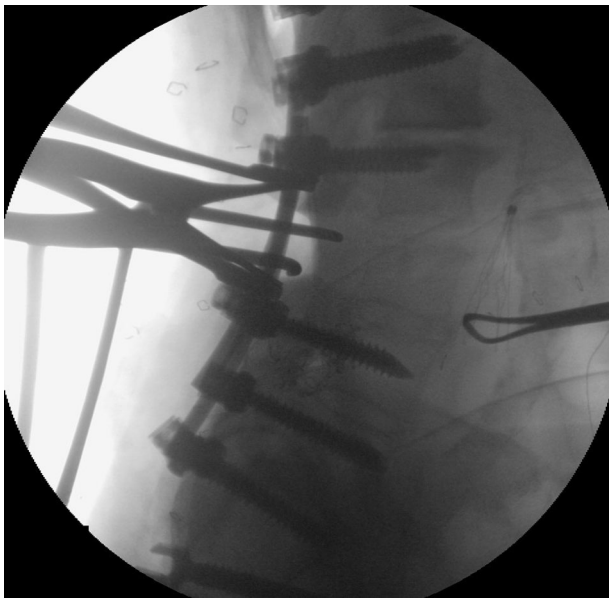

Fig. 6-A

Figs. 6-A, 6-B, and 6-C Intraoperative lateral fluoroscopic views obtained during a pedicle subtraction osteotomy. **Fig. 6-A** View following bone resection.

sels. Most vascular injuries associated with this procedure have been reported in patients with ankylosing spondylitis and presumed calcification of the great vessels[69-72]. However, vascular complications have also been reported in patients with iatrogenic flatback deformity, and they should be given careful consideration when corrective surgery is performed in older individuals, as they are frequently fatal[21]. Additionally, as the procedure lengthens the anterior column, superior mesenteric artery syndrome can result[73,74].

As a general rule, 1° of correction can be expected for each millimeter of posterior bone resection[20]. In patients treated for ankylosing spondylitis, the average correction has ranged from 33° to 40°[69,70,73]. Chang[67] reported an average of 37.8° of correction in seventeen patients in whom post-traumatic thoracolumbar kyphosis was treated with anterior discectomy and posterior osteotomy, but he performed multiple osteotomies in twelve patients.

Kostuik et al.[21] treated fifty-four patients with flatback syndrome with single-stage anterior opening and posterior closing osteotomies and reported a mean correction of 29°. Booth et al.[13] reported 25° and 30° of correction in five patients with segmental decompensation and twenty-three patients with global decompensation, respectively. The average improvement of the sagittal vertical axis was 6.4 cm in patients with sagittal imbalance. Kostuik et al. noted that 86% of the patients were satisfied with the procedure and that function was notably improved in 50%. In their landmark series, LaGrone et al.[6] evaluated the results of sixty-six Smith-Petersen-type osteotomies done through the pseudarthroses and fusion mass in fifty-five patients with symptomatic flatback. They noted an average initial correction of 22° in lumbar lordosis and 9° in kyphosis at the thoracolumbar junction, with an 8.1-cm improvement in the sagittal vertical axis. The authors also reported a high rate of complications, including pseudarthrosis and implant failure with substantial loss of correction at the time of follow-up. As a result, repeat operations were required in twenty-six patients, and 47% were still leaning forward and had inadequate correction at the time of follow-up.

Pedicle Subtraction Osteotomy

The pedicle subtraction osteotomy, or transpedicular cortical

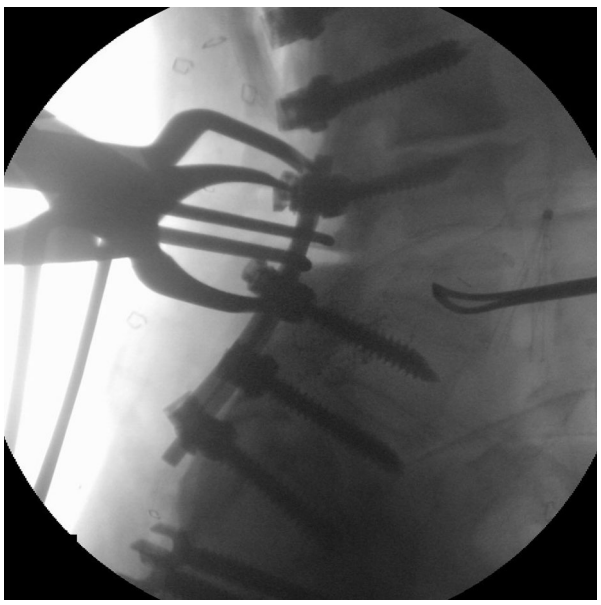

Fig. 6-B

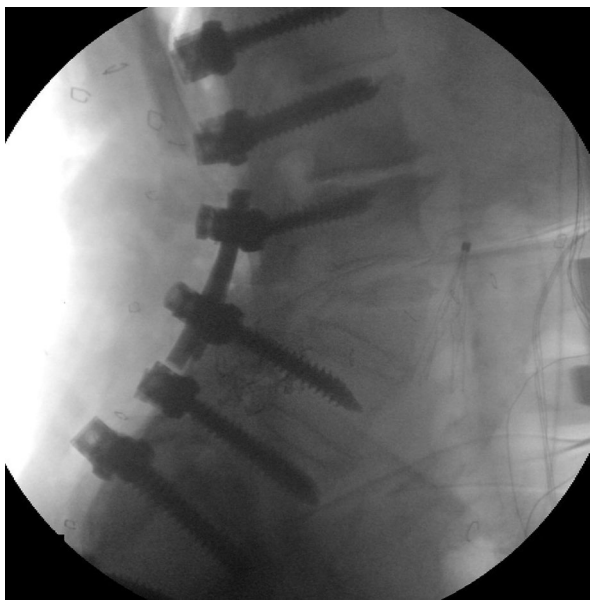

Fig. 6-C

Fig. 6-B View during compression of instrumentation to facilitate closure. **Fig. 6-C** View after completion and closure of the osteotomy.

decancellation procedure, is a three-column posterior closing wedge osteotomy hinging on the anterior cortex that has been frequently attributed to Thomasen[75], although an earlier variant was described by Leong et al.[76]. A so-called eggshell variation of this procedure was described by Heinig[77] and has also been utilized by several other authors[78,79]. The operative technique involves removal of all posterior elements at the level of the correction including the pedicles and the superior and inferior adjacent facet joints. A posterior wedge of cancellous bone is then removed from the vertebral body in order to allow the desired correction, with or without (the eggshell modification) removal of the entire posterior and lateral vertebral body walls (Figs. 5-A, 5-B, and 5-C). The osteotomy is then closed through compression of instrumentation or by extending the patient's position on the operative frame (Figs. 6-A, 6-B, and 6-C)[80]. Care must be taken to ensure that the neural elements are not compressed, and the exiting nerve root now shares an enlarged foramen with the superior adjacent root. Advantages of the procedure include the sagittal correction achievable at a single level, the ability to achieve coronal cor-

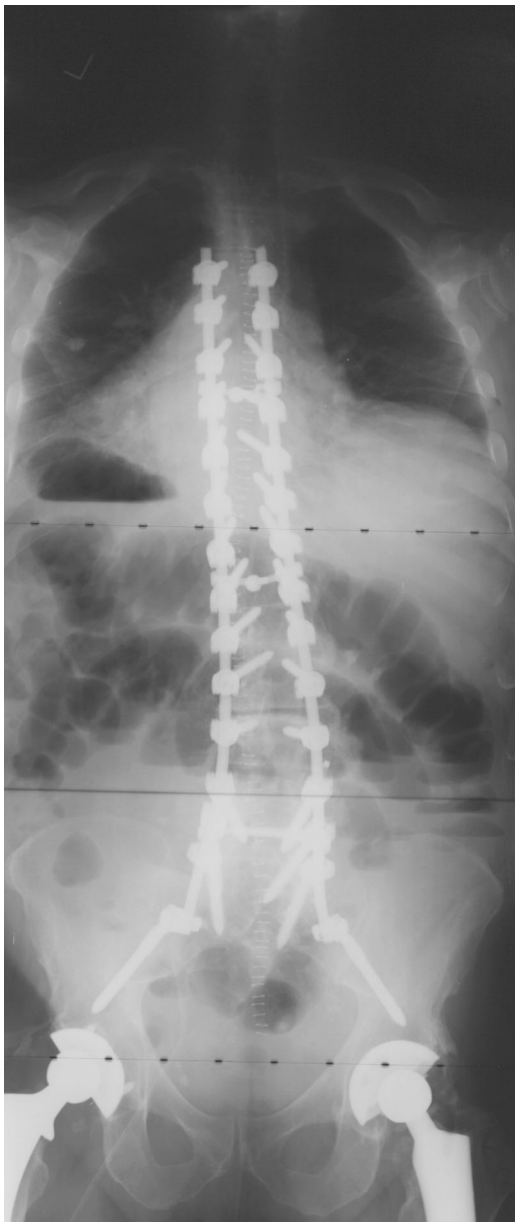

Fig. 7-A

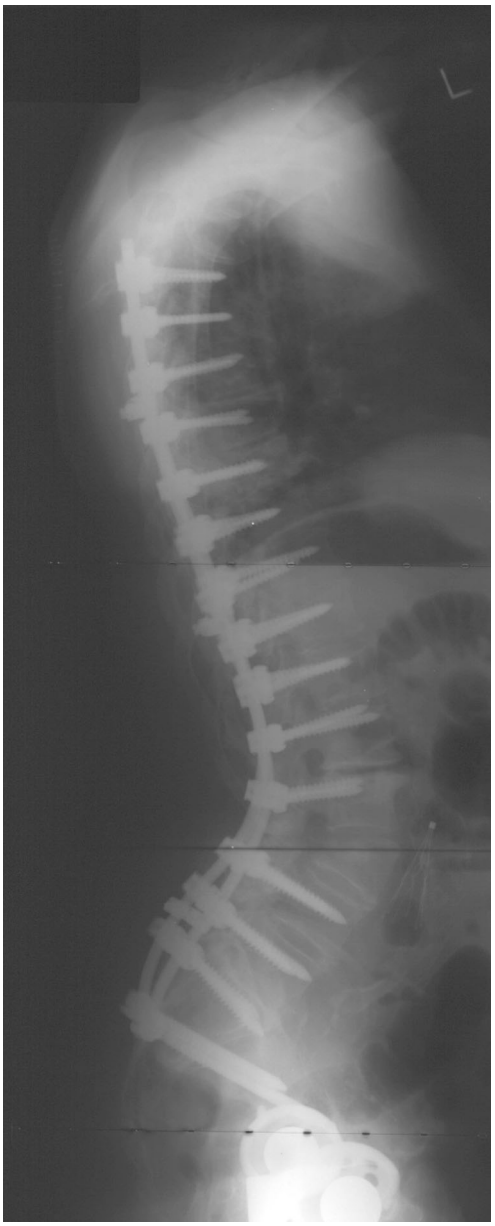

Fig. 7-B

Immediate postoperative anteroposterior (Fig. 7-A) and lateral (Fig. 7-B) radiographs following a pedicle subtraction osteotomy at the L3 fusion mass in a patient with flatback. Note the improved lumbar lordosis and sagittal balance (a change in the deviation of the sagittal vertical axis from 17 cm to 0.5 cm) compared with those seen on the preoperative radiographs (Figs. 3-A and 3-B).

rection by asymmetric resection of the vertebral body and cortex, preservation of the length of the anterior column, and the excellent potential for union provided by abundant cancellous bone contact (Figs. 7-A and 7-B). Drawbacks include the technically demanding nature of the procedure and the substantial bleeding frequently encountered as a result of the epidural venous plexus and the bleeding cancellous bone[20]. Contraindications to pedicle subtraction osteotomy include an anterior pseudarthrosis (unless a separate anterior procedure is performed) or previously placed anterior instrumentation across the correction level.

In the treatment of ankylosing spondylitis, the average achievable correction with single-level pedicle subtraction osteotomy has ranged from 32° to 36°[78,81-84]. Wu et al.[85] reported a mean of 38.8° of correction in thirteen patients in whom the operation was performed for a rigid post-traumatic kyphosis. Wu et al. noted only 2.3° of lost correction at two years postoperatively and no cases of pseudarthrosis. Murrey et al.[79] treated fifty-nine patients, including thirty-seven who had rigid adult deformity, with pedicle subtraction osteotomy. In forty-two patients followed for an average of 4.5 years, the average postoperative correction was 26°, with 5.5° of correction lost by the time of follow-up.

Similar results of pedicle subtraction osteotomy have been documented in patients with flatback syndrome. Noun et al.[22] reported 34° of initial correction and 31° of maintained correction at one year after the operation in ten patients with flatback syndrome. They found no cases of pseudarthrosis and minimal perioperative complications. Berven et al.[33] utilized pedicle subtraction osteotomy in thirteen patients with sagittal imbalance, including eight with flatback syndrome. They reported 30° of restored lordosis at the time of a two-year follow-up; correction of the sagittal vertical axis was 71% initially and 63% at the time of follow-up. However, although the authors noted no loss of correction in three patients with ankylosing spondylitis, four of their other ten patients lost >2 cm of correction of the sagittal vertical axis.

Polysegmental Wedge Osteotomy

Posterior closing polysegmental wedge osteotomy was introduced by Püschel and Zielke[86] in 1982 as a technique to restore physiologic sagittal alignment. The technique has been utilized in the management of fixed sagittal imbalance secondary to ankylosing spondylitis[70,87], but we are not aware of any reports of its use in patients with flatback deformity. Nonetheless, polysegmental wedge osteotomy deserves mention here because of its potential utility in the management of flatback syndrome. The technique is similar to the Smith-Petersen osteotomy, except that slightly less bone is resected posteriorly and less correction is attempted with each osteotomy. Advantages of the polysegmental technique include its relative safety throughout the thoracic spine and the reduced focal anterior column distraction, which decreases the need for concomitant anterior release, osteotomy, or fusion. Additionally, polysegmental wedge osteotomy may allow restoration of a physiologic, harmonious curvature across multiple vertebral segments, making the procedure well-suited for patients with ankylosing spondylitis or flatback with junctional or degenerative kyphosis cephalad to instrumentation, as opposed to achieving more dramatic angular correction at a single level.

To our knowledge, the largest published series of patients with ankylosing spondylitis managed with polysegmental correction was reported by Hehne et al.[87]. They described the operative complications and results in 177 patients treated with polysegmental wedge osteotomy and pedicle screw fixation. The average correction was 9.5° per level, for a total average correction of 43° per patient. Follow-up at eighteen and thirty-six months demonstrated losses of correction of 15% and 18%, respectively.

Vertebral Column Resection

Vertebral column resection, or vertebrectomy, and its modifications have been utilized for multiple indications since the original description by MacLennan[88] in 1922. Bradford et al.[48,89,90] popularized the utilization of vertebral column resection in the treatment of fixed or progressive sagittal plane imbalance. Vertebral column resection should not be performed for the treatment of isolated flatback syndrome, but it can be useful in patients with fixed sagittal imbalance in addition to degenerative or adult scoliosis with severe coronal plane deformities. As described by Bradford[90], the procedure consists of anterior vertebral body resection with maintenance of a cortical and periosteal flap through a convex-sided approach; posterior element resection is then performed in a sequential or staged fashion, followed by correction with instrumentation, with multiple wake-up tests. Advantages of the procedure include the dramatic correction possible at a single level and the overall shortening of the vertebral column, which relieves tension on anterior neurovascular structures; however, vertebral column resection is extremely technically demanding and can be associated with considerable perioperative morbidity.

Boachie-Adjei and Bradford[89] performed vertebral column resection in sixteen patients who had progressive loss of coronal and sagittal balance of various etiologies. They reported preservation of normal lumbar lordosis, when it had been present, and physiologic alignment in the sagittal plane in all patients at the time of follow-up, at an average of three years postoperatively. More recently, Bradford and Tribus[48] utilized vertebral column resection to manage twenty-four patients with progressive rigid coronal and sagittal decompensation. They noted correction of 82% in the coronal plane and improvement in sagittal balance of 87%. The complication rate was 43% in the study by Boachie-Adjei and Bradford and 58% in the study by Bradford and Tribus.

Comparison of Techniques and Complications

Each osteotomy technique for correction of sagittal deformity offers specific advantages and disadvantages as well as potential complications (Table III). Van Royen and De Gast[70] performed a

TABLE III Osteotomies for Correction of Flatback Deformity and Fixed Sagittal Imbalance

Type of Osteotomy	Correction per Segment	Benefits	Drawbacks	Relative Contraindications
Smith-Petersen	5°-20°, or 1°/mm of resection	Familiarity to surgeon; optimum for posterior-only approach when disc height maintained	Lengthens anterior column; frequent need for anterior release/fusion/osteotomy; higher reported pseudarthrosis rates	Ankylosing spondylitis; calcification of great vessels; or anterior instrumentation at same level
Pedicle subtraction	25°-35°	Sagittal and coronal correction possible; high union rates; posterior only	Greater blood loss; technically demanding; theoretical risk of devastating neurologic injury	Anterior pseudarthrosis or instrumentation at same level
Polysegmental posterior	9°-10°	Harmonious, sloping correction; posterior only	Multiple levels required for substantial correction; potential for loss of correction	Local stenosis; substantial single-level deformity; or anterior instrumentation at same level
Vertebral column resection	Variable	Greatest potential correction; sagittal and coronal correction possible; shortens spinal column, relieving neurovascular tension	Technically demanding; anterior procedure required; increased complication rates	

meta-analysis comparing the results of polysegmental, closing wedge (pedicle subtraction), and opening wedge (Smith-Petersen) osteotomies for the treatment of ankylosing spondylitis. Their review included sixteen studies with a total of 523 patients. Although pedicle subtraction provided, on the average, slightly less correction than did polysegmental and opening wedge osteotomies (35° compared with 40°), complication rates and loss of correction tended to be lower with pedicle subtraction osteotomy. In addition, Van Royen and De Gast reported higher mortality rates with Smith-Petersen-type osteotomies and more implant breakage and a greater potential for insufficient correction with polysegmental osteotomies. Although the authors noted insufficient presentation of data and poorly outlined surgical indications in most reports, they concluded that pedicle subtraction osteotomy appeared to be a safer and more reliable procedure.

Overall complication rates following operative correction of flatback syndrome have ranged from 20% to 60%[6,13,21,22,33,48,89]. Pseudarthrosis has not been reported following pedicle subtraction osteotomy, to our knowledge, but it developed after 38% (twenty-one) of fifty-five Smith-Petersen procedures in one report[6] and 19% (three) of sixteen vertebral column resections in another[89]. This is suspected to be due to the large surface area of cancellous bone available for healing following pedicle subtraction osteotomy[20]. Nerve root injuries may occur after all techniques, but they are generally transient. Specifically, transient radiculopathies have occurred following 0% to 13% of Smith-Petersen procedures[6,13,19] and 0% to 31% of pedicle subtraction osteotomies[22,33]. Cauda equina syndrome due to epidural hematoma formation has been reported following pedicle subtraction osteotomy[91,92]. Vascular injuries appear to be more common after Smith-Petersen osteotomies, particularly when a combined anterior-posterior procedure is performed[21].

In patients with iatrogenic flatback deformity, the average amounts of operative correction maintained following Smith-Petersen and pedicle subtraction osteotomies reportedly have been comparable, ranging from 16° to 30° and 30° to 31°, respectively[6,13,21,22,33]. To our knowledge, the outcomes of polysegmental osteotomies for the treatment of sagittal imbalance have been reported only for patients with ankylosing spondylitis, and, in what we believe to be the largest series of such patients, Hehne et al.[87] reported correction of 9.5° per level. There are also no available data on the results of vertebral column resection for flatback syndrome, to our knowledge. In two series in which that procedure was performed in patients with complex spinal deformity, Bradford et al. reported 87% correction of the sagittal vertical axis[48] and physiologic sagittal alignment[89].

Instrumentation

The wide spectrum of preoperative deformity and variations in operative techniques preclude a detailed analysis of the instrumentation utilized for the correction of fixed sagittal plane deformity in patients with flatback syndrome. However, several points deserve mention. In early series, Harrington compression instrumentation was associated with loss of correction and frequent implant failure[2,3,6,7]. At a minimum, segmental instrumentation should be utilized in order to maximize stability of the construct and osteotomy sites and to achieve bilateral compression and aid bone-healing across osteotomy sites. There should be at least four—and preferably six, seven, or eight—points of fixation on each side of an osteotomy. Most modern instrumentation allows virtually any combination of

screws and hooks, as dictated by the surgeon's preference.

Segmental pedicle screw instrumentation is often preferred in order to achieve rigid three-column fixation and control of the deformity. Several authors have found pedicle screws to be useful in the management of fixed sagittal imbalance[33,67,79,85], and use of such screws may also help to prevent loss of correction and lower the prevalence of implant failure[71]. However, when pedicle screw placement is not feasible because the prior fusion mass obscures landmarks, segmental hooks, particularly with a so-called claw construct[93], provide stable fixation and excellent compression. There may be better maintenance of restored lordosis if anterior fusion is also performed[6], although the use of pedicle subtraction osteotomy may obviate the need for anterior procedures.

A detailed discussion of sacropelvic fixation is beyond the scope of this discussion. However, when a fusion is being revised because of nonunion or when a previous lumbar fusion is extended to the sacrum in the management of flatback syndrome, supplemental fixation should be considered. At a minimum, bicortical or so-called tricortical screw fixation should be performed at S1, with the screws directed medially into the sacral promontory[94]. Additionally, supplementation with iliac screws, Luque-Galveston rods, and/or an anterior interbody fusion should be strongly considered to minimize the substantial risks of nonunion and caudad construct failure[95-98].

Overview

The management of iatrogenic flatback syndrome is difficult and complex, with a high rate of operative morbidity. Thus, attention should be focused on the prevention of this deformity through preoperative assessment and planning of the fusion, physiologic surgical positioning, and appropriate segmental instrumentation with maintenance or restoration of lumbar lordosis and sagittal alignment. Once flatback syndrome is established, appropriate operative treatment can result in excellent correction of deformity and a high degree of patient satisfaction despite high short-term complication rates. The exact technique of operative correction depends on the patient's pathoanatomy and the operating surgeon's experience. However, for most patients, pedicle subtraction osteotomy appears to be the optimal surgical technique; it provides excellent restoration of sagittal alignment and the potential to address coronal alignment with a decreased rate of complications, particularly pseudarthrosis and loss of correction. The instrumentation used following operative correction should be segmental and compressive, and pedicle screws appear to be ideal for this task.

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