

Flatback Syndrome

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Doherty [1] was the first to describe this phenomenon. In 1973, he described a postural inclination of the trunk with loss of normal lumbar lordosis observed in patients with thoracolumbar scoliosis treated with posterior spinal fusion and placement of Harrington instrumentation (Figs. 1 and 2).

In 1977, Moe and Denis [2] reported on a series of 16 patients with loss of lumbar lordosis after thoracolumbar fusions and coined the term *flatback syndrome*. Symptoms reported included muscular pain in the upper back and lower cervical area, knee pain, and inability to stand upright. In evaluation of this spinal deformity, a full-length standing lateral radiograph was obtained. A plumb line was dropped from the center of C-7 vertebra, and the distance of this line was measured to the sacral promontory; a distance of less than 2 cm was considered to be normal. In these patients, Moe and Denis [2] used extension osteotomies to correct the lumbar hypolordosis. Good short-term results were observed in these patients. Since then, flatback syndrome, also known as “fixed sagittal imbalance,” has become a well-known entity. The etiology of flatback syndrome may be multiple; however, the most common cause is iatrogenic secondary to Harrington rod instrumentation [3–9].

In a slight variation in etiology, Farcy and Schwab [10,11] described a group of patients with “kyphotic decompensation syndrome” and “flat buttock syndrome,” which was described to be a fixed positive-sagittal imbalance attributable to malalignment at the site of spinal fusion to

the sacrum performed with a distraction instrument in treatment of disease other than scoliosis.

Flatback syndrome has also been divided into separate subgroups: segmental (type 1) loss of lumbar lordosis or lumbar kyphosis with preservation of normal sagittal balance and global (type 2), or classic, flatback syndrome with significant fixed positive sagittal imbalance [12]. Since then, flatback syndrome has been generally defined as an iatrogenic symptomatic condition attributable to spinal instrumentation or fusion that causes loss of lumbar lordosis [13].

Iatrogenic causes of flatback syndrome

A broad spectrum of causes leads to flatback syndrome. The identified factors that contribute to this include placement of distraction instrumentation in the lower lumbar spine or sacrum, pseudoarthrosis resulting in loss of sagittal plane correction, fixed thoracic hyperkyphosis, hip flexion contractures, and preexisting thoracolumbar kyphosis [2]. The most commonly reported cause of flatback syndrome is extension of distraction instrumentation into the lower lumbar spine or sacrum [3–5,7–9,14–16]. The advent of Harrington rod instrumentation for scoliosis correction increased the incidence of flatback syndrome. Conversely, Moskowitz and colleagues [17] reported on patients who had undergone posterior spinal fusion without instrumentation for scoliosis with a 20-year follow-up; none had flatback syndrome.

Harrington instrumentation or distraction instrumentation allowed substantial correction in the coronal plane, and little thought was given to the effects on the sagittal plane. This technique often led to the loss of lumbar lordosis, with anterior translation of the vertical axis and even

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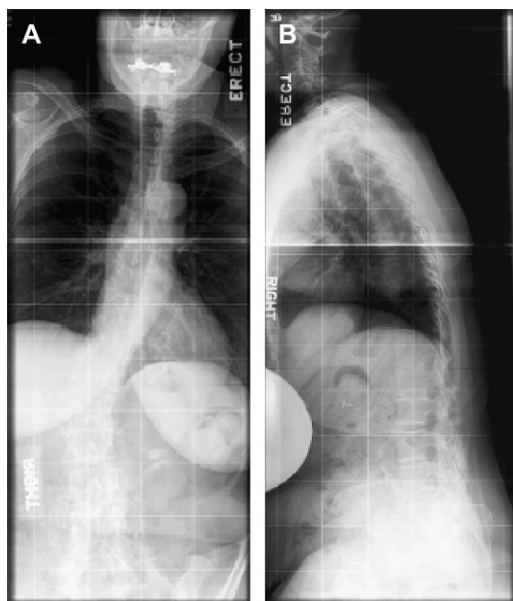


Fig. 1. (A) Anterior-posterior radiograph of a 72-year-old woman with previous lumbar fusions 14 years before presentation. The patient presented with back pain and an inability to stand up straight. (B) Sagittal standing films show the patient's loss of lumbar lordosis and positive sagittal balance.

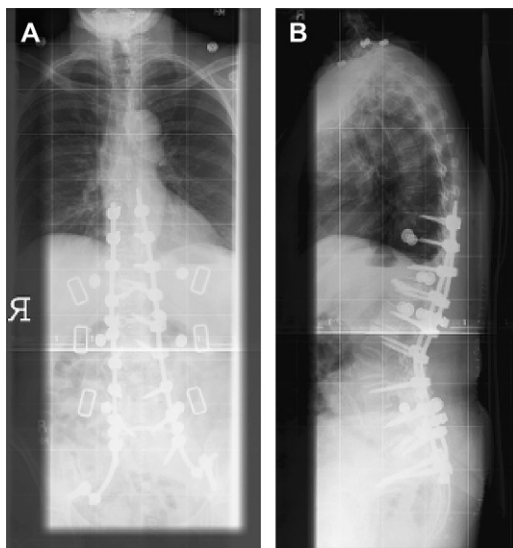


Fig. 2. (A) Anterior-posterior radiograph demonstrates coronal correction of scoliosis. (B) Sagittal radiograph after L3 pedicle subtraction osteotomy was performed with posterior fixation from T9 to the ilium.

kyphosis. The loss of lordosis increased the more distally the instrumentation was placed. This was confirmed by a study of 96 patients with Harrington distraction instrumentation conducted by Aaro and Ohlen [4]. They recommended avoiding fusion below L-3 if possible. Patients who had instrumentation placed down to T-12 had lumbar lordosis of -38° , but patients who had instrumentation placed down to L-5 had -16° of lordosis [3,14].

The incidence of flatback syndrome varies and depends on the distal level of distraction instrumentation. In 1981, Swank and colleagues [8] reported a 5% incidence of symptomatic loss of lumbar lordosis. In 1983, Kostuik and Hall [18] reported a 49% incidence of loss of lumbar lordosis in patients who underwent distraction instrumentation and fusion to the sacrum for scoliosis. Twenty-nine percent of these patients underwent corrective osteotomies with improvement in pain.

Clinical presentation

Flatback syndrome is characterized by forward inclination of trunk, difficulty in standing erect with the knees fully extended, and pain as a result of loss of lumbar lordosis. To compensate for the loss of lumbar lordosis and to stand erect, the patient flexes the hips and knees, with cervical extension to maintain an upright horizontal gaze. This abnormal posture puts stress on the cervical, thoracic, and lumbar spine, resulting in fatigue and pain. Physical examination reveals flattening of the lumbar region and forward tilting of the trunk when the patient is standing erect with the knees fully extended.

Studies have shown that paraspinal muscles in the cervical and lumbar areas are used to maintain an erect posture in the setting of loss of lumbar lordosis; this contributes to increased pain and degenerative changes. Degenerative spondylosis in the cervical spine has been seen in patients with corrective surgery for adolescent idiopathic scoliosis with Harrington rod distraction [14]. Disc and facet joint degeneration has been seen at the L4-L5 level; this has been attributed to ending Harrington distraction instrumentation at L-4 [19].

Radiographic evaluation

Sagittal curvature in the thoracic and lumbar spine has also been used to assess pathologic

imbalance. Normal functional ranges vary widely; however, it is generally accepted that normal thoracic kyphosis ranges from 20° to 50° and that normal lumbar lordosis ranges from 20° to 65°. Additionally, the thoracolumbar junction should be straight [20,21].

Formally, the workup for flatback syndrome should include a standing full-length, 36-in lateral radiograph with extension of the knees. Visualization of the C-7 vertebral body and the hip joint is critical. To assess pathologic global sagittal imbalance, a plumb line is dropped from the center of C-7 to the pelvis. Neutral sagittal balance occurs if the plumb line intersects the posterosuperior corner of the S-1 vertebral body. If the plumb line falls behind the lumbosacral disc, there is negative balance; likewise, if the plumb line falls in front of the lumbosacral disc, there is positive balance. If the plumb line falls within 2 cm of the anterior aspect of the sacrum, this is considered normal in patients without scoliosis or in those with idiopathic scoliosis who have not undergone corrective surgery [2].

Nonsurgical treatment of flatback syndrome

Exercises to increase hip and back extension, pain medication, and bracing are all used as first-line nonoperative treatment options. In the only study of nonoperative treatment of flatback syndrome, only 27% of patients were ultimately considered to have a long-term successful result of nonoperative management [10]. This percentage is probably lower, however, considering that the mean sagittal imbalance in this group of nonoperative patients was only 3.4 cm. Conservative medical management is often not useful once a patient becomes symptomatic, and surgical treatment is often indicated [6]. Surgical treatment of flatback deformity can be a highly morbid and complex procedure with perioperative complications in upward of 60% of cases [2]. Most patients improve symptomatically; however, almost none are completely pain-free [6]. Therefore, prevention of flatback syndrome is of the utmost importance in preoperative evaluation of spinal fusion procedures. Before a patient undergoes a long-fusion procedure, preoperative radiographic evaluation should be performed with plain radiographs to evaluate sagittal curvature and balance, including rotational and coronal plane deformities. Care should be taken to preserve or correct thoracic and lumbar curvature and, most importantly,

sagittal balance, as previously described. In short-segment degenerative cases, lumbar lordosis should at least be maintained if not amplified in anticipation of further degeneration and loss of curvature [22–25]. Investigators have often terminated instrumentation at or above L3 to prevent future sagittal imbalance [3–7]. In addition to decreasing the risk of sagittal imbalance, preserving the caudal lumbar spine may decrease the risk of retrolisthesis, pseudoarthrosis, and adjacent level disease caudal to fusion [8,14,17,26].

Treatment

The goal of corrective surgery is to restore physiologic lordosis and sagittal balance so that the plumb line intersects the posterosuperior aspect of the sacrum. This should allow the patient to stand erect without compensatory flexion of the knee and hyperextension of the hip. The decision regarding placement of the osteotomy depends on the site of deformity. In general, corrective osteotomies should be performed at the site of maximal deformity. Patients with flattening of the lumbar spine without thoracolumbar involvement can be treated at L2 or caudad, reducing the risk of conus medullaris or spinal cord injury. If there is coexisting thoracolumbar kyphosis, however, osteotomies may have to be extended higher.

Several types of osteotomies have been described to correct sagittal imbalance. Extension (Smith-Petersen) osteotomy, pedicle subtraction osteotomy, and vertebral column resection have all been described to treat fixed sagittal deformities.

Extension (Smith-Petersen) osteotomy

In 1945, Smith-Petersen and colleagues [27] described a posterior osteotomy for correction of fixed sagittal deformity in patients with rheumatoid arthritis. This procedure involved resecting the posterior elements, undercutting the adjacent spinous processes, and closing the osteotomy. The name of this technique originates from the closure of this osteotomy by creating an opening of the spine (extension) anteriorly into the disc space, with the posterior aspect of the disc space as the axis of rotation. Sagittal correction is achieved through posterior compression instrumentation and creates hyperextension by closing posterior elements and opening the anterior

elements. An important drawback of this procedure is that it lengthens the anterior column and may destabilize the spine if there is pseudoarthrosis. Because of this, in 1946, La Chapelle [28] described a modification of this technique by adding an anterior release. This modification includes an anterior release, discectomy, or osteotomy and structural grafting in addition to posterior Smith-Petersen osteotomies.

Although this technique can correct deformity, there is substantial risk of morbidity and mortality. By lengthening the anterior column, there is risk to anterior vascular structures and neural elements. In 1959, Law [29] reported a 10% mortality rate related to this procedure. Others have reported neurologic complications in up to 30% of cases [30].

In general, 1° of correction can be obtained with each millimeter of posterior bone resection. The reported average correction in most series is approximately 10° to 15° [6,10,18,30–32]. In a large series, LaGrone and associates [6] studied the results of 55 patients with symptomatic flatback treated by 66 Smith-Petersen type osteotomies done through the fusion mass or pseudoarthrosis. They described an average correction of 22° in lumbar lordosis and 9° in kyphosis at the thoracolumbar junction, with 8.1 cm improvement in the sagittal plane. There is a high rate of complications reported, however, including pseudoarthrosis or implant failure with loss of correction at follow-up, which required repeat operations in 26 patients. Notably, 47% of patients still had inadequate correction at follow-up at 6 years. In summary, extension osteotomy can correct sagittal imbalance through lengthening the anterior spine, which can place the anterior vascular and neural structures at risk; furthermore, the destabilizing nature of this procedure can increase the risk of pseudoarthrosis.

Pedicle subtraction osteotomy

In contrast to the extension osteotomy, the pedicle subtraction osteotomy is a procedure that corrects deformity without lengthening the anterior spine. Pedicle subtraction osteotomy is a transpedicular cortical decancellation procedure. It achieves correction by a three-column posterior closing wedge osteotomy hinging on the anterior cortex and has been attributed first to Thomasen [33]. This procedure involves removal of posterior elements, including the pedicle, transverse process, and superior and inferior adjacent

facet joints. A posterior wedge of cancellous bone is removed from the vertebral body to achieve the desired correction, with or without removal of the entire posterior and lateral vertebral body walls. The osteotomy is closed by compression of instrumentation or by extending the patient's operative position.

With this procedure, removal of up to 6 cm of bone is possible with sagittal correction of up to 30°. Additionally, flexibility is present to correct for coronal plane deformity by performing asymmetric removal of posterior elements [34]. Furthermore, the anterior spine is not lengthened, thereby avoiding the vascular and abdominal complications associated with extension osteotomies. Finally, the bone surface for fusion is large, and instrumentation is used to maintain alignment rather than creating alignment, as in the case of extension osteotomy.

There are risks of this procedure. It is technically demanding, and substantial blood may be lost through the epidural venous plexus and cancellous bone itself. With the removal of pedicles bilaterally, two nerve roots now share an enlarged foramen with the superior adjacent nerve root. Care must be taken so that these nerve roots are not compressed or damaged, especially on closure of the osteotomy.

Since the original description, pedicle subtraction osteotomy has been applied to fixed sagittal plane deformities of different etiologies ranging from ankylosing spondylitis to trauma to flatback syndrome. Bridwell [34] reported on 27 patients with fixed sagittal imbalance, using this technique on patients with idiopathic scoliosis, degenerative scoliosis, traumatic spondylitis, and ankylosing spondylitis. The average correction was 34.5° at 2 years of follow-up. Similar results have been found in patients with flatback syndrome. Noun and colleagues [35] reported on 10 patients with 34° of initial correction and 31° of maintained correction at 1 year of follow-up. There was no incidence of pseudoarthrosis and no neurologic complications. Berven and colleagues [36] also reported on their series of 8 patients with flatback syndrome. They reported 30° of restored lordosis at 2 years of follow-up, confirming the results of previous studies.

Polysegmental osteotomies

The previously described complications in extension osteotomy can be avoided by

polysegmental osteotomies. This technique was first reported by Wilson and Turkell [37] for correction of sagittal balance in a patient with ankylosing spondylitis and involves removing the facet joints at several levels and then compressing the posterior elements to create lordosis. In contrast to extension osteotomy, this correction is obtained through deformation of disc spaces without rupture of the anterior longitudinal ligament. Additionally, this procedure allows restoration of physiologic harmonious curvature across multiple vertebral segments, making it suitable for patients with flatback syndrome with kyphosis cephalad to instrumentation.

There has been no application of this technique to flatback syndrome to the authors' knowledge; however, this technique can theoretically be applied to patients with flatback syndrome. The largest reported series of patients with ankylosing spondylitis managed by this method was by Hehne and colleagues [38] with 177 patients. The average correction was 44° or 9.5° per segment. This is associated with a 2.3% risk of death, a 2.3% risk of irreversible neurologic deficit, and an 18% risk of reversible neurologic injury. There was a 20% loss of correction at 36 months, with implant breakage in 4 patients.

Comparison of techniques

Each of the previously described surgical techniques addressing sagittal imbalance has its advantages and disadvantages. In 1999, Van Royen and De Gast [39] performed a meta-analysis of polysegmental, pedicle subtraction, and Smith-Petersen osteotomies in the treatment of ankylosing spondylitis. Their review included 16 studies with a total of 523 patients. Pedicle subtraction osteotomy provided more correction than polysegmental and Smith-Petersen osteotomies, and complication rates tended to be lower with pedicle subtraction osteotomy. Additionally, the perioperative mortality rate was highest in Smith-Petersen osteotomies (5.8%) and lowest in pedicle subtraction osteotomies (1.3%). Furthermore, polysegmental osteotomies carried a higher rate of implant breakage and greater likelihood of loss of correction (6%) compared with pedicle subtraction osteotomies (2.7%). Despite the appearance of pedicle subtraction osteotomy as a safer and more reliable procedure, the authors decided that there was insufficient evidence to conclude that one surgical technique is preferable

to the other because of insufficient data and poorly described surgical indications in the reports reviewed.

Summary

Recently, iatrogenic flatback syndrome has increased because of the trend of increased long-segment thoracolumbar instrumentation and fusion. Patients present with difficulty in standing erect without flexing the knees or hyperextension of the hips, and this often leads to chronic back pain that is refractory to conservative medical therapy. The initial workup must include a 36-in standing lateral spine radiograph with the patient standing erect with the hips and knees extended. The exact surgical technique depends on the disease anatomy and experience of the surgeon. Surgical correction can include extension (Smith-Petersen) osteotomy, polysegmental osteotomies, or pedicle subtraction osteotomy.

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