Degenerative lumbar scoliosis associated with spinal stenosis
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Abstract
BACKGROUND CONTEXT: Degenerative de novo scoliosis is commonly present in older adult patients with spinal pain. The degenerative process including disc bulging, facet arthritis, and ligamentum flavum hypertrophy contributes to the appearance of symptoms of spinal stenosis in these patients.

PURPOSE: The etiology, prevalence, biomechanics, classification, symptomatology, and treatment of degenerative lumbar scoliosis in association with spinal stenosis are reviewed.

STUDY DESIGN: Review study.

METHODS: Retrospective analysis of studies focused on all parameters concerning degenerative scoliosis associated with stenosis.

RESULTS: There is a variety of treatment methods of degenerative scoliosis based on symptomatology and radiologic measurements of scoliosis and stenosis. Satisfactory clinical results reported in relevant retrospective studies after operative treatment range from 83% to 96% but with increased percentage of complications. An algorithm for operative treatment corresponding to a newly proposed classification system of degenerative lumbar scoliosis with associated canal stenosis is presented.

CONCLUSIONS: There is an increasing prevalence of degenerative scoliosis in the aged population. Even though the exact percentage of patients with symptomatology of spinal stenosis is not known, the main goal is to provide pain relief and improved functional lifestyle with minimum intervention.

Keywords: Degenerative scoliosis; Spinal stenosis; Decompression; Fusion

Introduction
Adult scoliosis is defined any curvature of the spine more than 10° in a skeletally mature individual. The term degenerative de novo scoliosis (DDS) refers to scoliotic curves developing after skeletal maturity without previous history of scoliosis. Scoliosis occurs de novo in later life and is associated not only with severe back or leg pain but also with complicated surgical outcomes [1].

Degenerative spondylolisthesis is very common in cases of degenerative scoliosis. In a study of patients with degenerative scoliosis, 55% of them had additional degenerative spondylolisthesis [2]. Rotatory olisthesis coexists in 13% to 34% of adult scoliosis cases [3,4]. It is a triaxial deformity consisting of axial rotation on the vertical axis, lateral translation toward the convexity of the curve, and anterior translation in the sagittal axis. Trammell et al. [4] originally described rotatory olisthesis as the apparent lateral subluxation of one vertebral body upon another seen in the anteroposterior films. Other terms used in the past for the same pathology are lateral spondylolisthesis, translatory shift, lateral olisthesis, and lateral subluxation. It occurs most frequently at the L3–L4 level, in women, and increases with age and magnitude of the curve.

Lumbar spinal stenosis is a common problem in the older adult population. It is defined as a pathologic condition in which the neural elements are compressed by bone, soft tissue, or both resulting in ischemia of nerve roots [5]. It is distinguished in central stenosis, when there is abnormal narrowing of the spinal canal, and lateral stenosis, when there is lateral recess (subarticular stenosis) or foraminal (foraminal stenosis) narrowing [6]. Symptoms of neurogenic claudication and radiculopathy predominate. Frequently, it is accompanied by degenerative olisthesis and...
degenerative scoliosis. The adjuvant scoliosis complicates neural compression and makes surgical treatment more difficult [7].

Aging affects bony structures, discs, ligaments, facet joints, and muscles. This causes wedging of both vertebral bodies and discs and may ultimately lead to a domino-like effect of rotation and translation into the upper lumbar and sometimes thoracolumbar spine. Degenerative scoliosis, reduced lumbar lordosis, and neural element compression is the final end result. Also, the combination of ligamentum flavum hypertrophy, facet hypertrophy, disc collapse, and olisthesis cause neural compression.

The purpose of this article is the in-depth analysis of degenerative scoliosis associated with symptoms of canal stenosis. A comprehensive and simple classification system with corresponding algorithm for operative treatment for degenerative lumbar scoliosis associated with spinal stenosis is presented.

Etiology

Decreased bone density was initially considered to be the cause of degenerative scoliosis [8,9], but this has changed after finding that no significant difference exists in the bone density between patients with degenerative scoliosis and patients with adolescent scoliosis of the adult [10]. Actually, degenerative changes, especially of the disc, vertebral body wedging, and facet joint arthrosis are responsible for the appearance of “de novo” scoliosis and degenerative rotatory scoliosis [11,12].

A cadaver study of 19 lumbar spines with degenerative olisthesis without scoliosis showed it was mostly associated to disc degeneration and osteophytesis of apophyseal joints and less to fractured endplates. The joint capsules become attenuated as a new false articular surface is created on the lamina next to the inferior articular process. In the same study, canal constriction was not found because of olisthesis. In the same study, canal constriction was not found because of olisthesis. Therefore, the vertebra above in the spinal unit progress gradually to forward olisthesis and rotation that leads to rotatory scoliosis and stenotic symptoms caused by the kinking of the neural elements by the malrotated pedicles [13–15].

Prevalence and natural history

The prevalence of degenerative scoliosis is reported to be from 6% to 68% [8,11,16,17] and increases with age [2]. Pritchett and Bortel [2] studied 200 patients more than 50 years old (70% women) with late-onset scoliosis. They found that most of the lumbar degenerative curves did not exceed 60° and progressed an average 3° per year. Factors for curve progression were Nash-Moe grade 3 apical rotation, a Cobb angle more than 30°, lateral vertebral translation of 6 mm or more, and the prominence of the L5 vertebra in relation to the intercrest line. On the contrary, Murata et al. [12] showed in the early phase of degenerative curves they might also regress after consequential wedging in the disc space or the vertebral bodies for compensating imbalance.

The mean age of patients with degenerative scoliosis was less than 60 years in 38% of Perennnu’s study and 33% in Kostuik’s study, whereas 41% of scoliotic patients were younger than 64 years old in Robin’s study. In Trammell’s study, the average age of patients with rotatory scoliosis was 41 years. Right-sided degenerative scolioses worsen with aging twice as much than left-sided scoliosis [3,17]. This is associated also with deterioration of rotatory olisthesis.

There are no studies estimating what percentage of patients with degenerative scoliosis will need evaluation and treatment for their symptoms, axial back pain, or symptomatology of spinal stenosis. It is also unknown what is the natural history of these symptoms without treatment. However, the symptoms of spinal stenosis irrespective of scoliosis have been found to be improving over time in 15% of cases, deteriorating during the initial 2 to 3 years of follow-up requiring surgical intervention in 40% of the patients and maintaining their status over long-term in the rest 45% of the cases [6,18].

Biomechanics

It is known that the kinematics and the coupling pattern are different from one spinal region to another. The coupling pattern of lateral bending and axial rotation is converging in the lumbosacral junction meaning that right lateral bending coincides with right axial rotation of L5 vertebra on the sacrum. However, in the lumbar spine, the coupling pattern of lateral flexion and axial rotation is
diverging; that means right axial rotation results in left lateral bending [19,20].

In scoliosis, thoracic spine motion is balanced but the coupling mechanism is abnormal. Instead of axial rotation to be toward the concavity, it is occurs into the convexity of the curve [21]. It is unknown what the etiology is of this abnormal coupling motion pattern.

On the contrary, Veldhuizen and Scholten [15] found that the coupling mechanism of axial rotation and lateral bending does not differ in normal and scoliotic spines. However, they supported that there exists a special coupling mechanism between sagittal plane motion and axial rotation in the scoliotic spine.

Disc and facet degeneration leads to increased axial rotation of the functional spinal unit. The increase in axial rotation caused by spinal degeneration was more pronounced than the increase of motion in flexion-extension [20]. Degenerative scoliosis is also followed by decreased lumbar lordosis and, subsequently, low back pain [2,12].

There have been described various methods for determination of vertebral rotation [22–24]. The most widely used method of radiographic determination of vertebral rotation is the pedicle method (grade 0–IV) introduced by Nash and Moe [22]. Grade 0 defines the neutral vertebra when there is no asymmetry between the anteroposterior pedicle projection in the convex and the concave side. Grade IV is the total disappearance of the pedicle in the concave side, whereas the pedicle on the convex side migrates past midline.

Clinical presentation

The incidence of low back pain in patients with degenerative scoliosis is not significantly more common than in the general population. Paraspinal muscle electrodiagnostic examination has helped to define the association between radiological spinal stenosis and back pain [25]. Lumbar scoliosis with degenerative changes most prominent at the sciotic apex can produce lumbar stenosis resulting in disabling low back pain and leg pain in a neurogenic claudication mode [26]. However, sometimes leg symptoms appear from the contralateral side because of the lumbosacral curve [27,28]. Liu et al. [29], in their study of patients with degenerative lumbar scoliosis and stenosis, concluded that L3 and L4 nerve roots are compressed in the foramen or extraforaminally on the concave side of the main curve, whereas the L5 and S1 roots are affected more by lateral recess stenosis at the convex of the lumbosacral fractional curve.

However, rotatory olisthesis does correlate with back pain associated with idiopathic scoliosis. It is significantly associated with increasing age and magnitude of the curve [4]. Smaller Cobb angles and lateral and rotatory olisthesis are more frequently seen in degenerative scoliosis than in adult idiopathic scoliosis.

On the contrary, Perennou et al. [3] showed that there is definitely correlation between anterior femoral radicular pain and degenerative rotatory olisthesis especially at L3–L4 and L4–L5 levels. However, the mean age of patients was 62 years in Perennou’s study and 23 years in Trammel’s study.

Velis and Thorne [30] and Kostuik et al. [31] described lateral olisthesis as an important prognostic factor for the development of back pain and curve deterioration after skeletal maturity. Sewab et al. [32] comparing radiographic parameters with VAS scores in adult scoliotic patients found that pain correlates with L3 and/or L4 obliquity, lateral rotatory olisthesis, lumbar lordosis, and thoracolumbar kyphosis. Curve magnitude and number of levels involved in the curvature were unrelated to pain. Degenerative instability is the cause of pain and a common pathway in the progression of all types of adult scolioses.

The etiology of the pain might be mechanical or neurogenic [33]. Commonly, mechanical back pain presents in cases with longstanding idiopathic scoliosis. It appears with muscle pain because of fatigue of spinal muscles (along the whole length of the spine and specifically in the convex side of the curve) and is attributed to the loss of lumbar lordosis. The pain in the concavity of the scoliosis is caused by facet arthritis and degenerative changes of the disc spaces (disc changes, ligament tension and tears, spurs, and so on).

The sagittal gravity line passes in front of the S1 vertebral body and back musculature is over functioning for keeping an upright position. The notion that clinical symptoms of back pain increase linearly with the degree of positive sagittal imbalance is supported by the findings of Glassman et al. [34] in a retrospective review of 752 patients with adult scoliosis.

Neurogenic pain is mainly a feature of patients with de novo scoliosis. It is possible, although, to be present in cases of adult idiopathic scoliosis. Neurogenic pain is caused by either foraminal nerve root or dorsal root ganglion compression (concave side of the curve) or central spinal stenosis leading to neurogenic claudication.

It is imperative to distinguish the neurogenic claudication symptoms from the pain caused from vascular claudication. The pain in neurogenic claudication is reduced if the patient acquires a forward flexed (like bicycling) or sitting position. The pain in vascular claudication is relieved by standing still and accompanied with peripheral vascular loss and skin changes.

The accompanying medical comorbidities in these elderly patients should not be overlooked. It should be kept in mind that the patient is being operated on is a whole person, not just a spine. Keeping the surgical intervention as minimal as possible should be the ideal goal.

Lumbar spinal stenosis

Spinal stenosis is the narrowing of the spinal canal and can be local, segmental, or generalized. It is divided into the constrictive stenosis caused by bony or ligamentous reduction in
the size of the canal and the restrictive stenosis, which is attributed to intracanal fibrosis (i.e., arachnoiditis) restricting the mobility of neural elements [35]. Usually, it is acquired as in degenerative diseases or spondylolisthesis and less commonly is congenital. The degenerative disease of the spine leads to constrictive-compressive type stenosis. It reduces substantially the spinal reserve capacity; this is the ratio between the horizontal distance of posterior middle of the lamina from the posterior end of the vertebral body and the anteroposterior diameter of neural canal measured in CT transverse sections. Spinal reserve capacity is actually a functional factor of spinal canal and is better than other static measurements like the AP canal diameter [35]. It is commonly admitted that absolute stenosis exist when AP canal diameter is less than 11 mm [36]. However, the use of subjective criteria to characterize spinal stenosis by radiologists and relevant clinical physicians (spine surgeons, neurologists, and physiatrists) led to high variability of results for the same patients [37]. Radiologists specialized in spine imaging subjectively grade the stenosis as mild, moderate, and severe, according to the degree.

The criteria for central and lateral stenosis are difficult to define. Verbiest [38] was the first to introduce the terms of absolute and relative value of spinal stenosis. The shape of the spinal canal, the facet joints, and the measured spinal dimensions (interpedicular distance, sagittal diameter of the dural sac, and pedicle length) are some of the measurements evaluating spinal stenosis [39]. Hypertrophy of the ascending facet is the main cause of lateral stenosis [7]. Biomechanical studies of the foramen of the lumbar spine proved that during flexion and contralateral lateral bending of the spine the foraminal cross sectional area increases, whereas in extension or ipsilateral lateral bending the foraminal area decreases. Distraction may increase the foraminal cross-sectional area, whereas in extension or ipsilateral lateral bending the foraminal cross-sectional area decreases. Distraction may increase the foraminal cross-sectional area, whereas in extension or ipsilateral lateral bending the foraminal area decreases. Distraction may increase the foraminal cross-sectional area, whereas in extension or ipsilateral lateral bending the foraminal area decreases.

Mechanical studies of the neural foramen, it was found that the nerve root and ganglion are within the upper pole of the foramen above the disc and never occupy more than 35% of the cross-sectional area. In foraminal morphometric studies of cadaveric spines, it was found that the foraminal variable size between 40 and 160 mm² and defined the normal limits of foraminal cross-sectional area in the sagittal plane [41]. Measurement of vertebral foramina using calipers is superior to that with CT scan [42].

Often, the radiological findings are more severe than the patients’ symptoms. Studies of patients with lumbar spinal stenosis have shown that there is no statistically significant correlation between the intensity of pain and the degree of stenosis. Most commonly, the radiologic image of stenosis was more extensive than expected from the clinical picture [39]. Therefore, spinal stenosis can be either radiographic-only spinal stenosis or symptomatic spinal stenosis with a combination of clinical and radiographic characteristics.

It is mentioned earlier in the etiology of rotatory scoliosis that there is compression of the dura and traction of the nerve roots because of vertebral rotation and subluxation. Additionally, hypertrophy of ligamentum flavum, disc degeneration and protrusion, facet joint osteophytosis, and cysts of the facet joints are significantly decreasing the functional capacity of the spinal canal.

Classification

The only classification system that exists for degenerative lumbar scoliosis addresses only the existence of rotation or/and lumbar lordosis loss. It does not include rotatory lumbar scoliosis or stenosis. Simmons characterized as type I the degenerative scoliosis with no or minimal rotation, whereas type II was defined as the degenerative scoliosis with rotational deformity and reduced lumbar lordosis [1,43,44].

Schwab et al. [32] recently published a classification of adult scoliosis based on radiographic measurements in anteroposterior and lateral standing radiographs. They measured the lordosis L1–S1 and the obliquity of L3 vertebral body and divided the population in three types of severity with decreasing lordosis and increasing L3 obliquity [32].

There is need for a classification system that it will address the segmental type of deformity, the sagittal spinal contour, and the symptomatology. The proposed classification system is shown in the Table 1. However, the validity and reliability of this system are still under investigation, and this is a best-guess approach based on our experience.

Radiographic evaluation

Plain full-spine standing AP and lateral radiographs are necessary to evaluate case of degenerative scoliosis associated with rotatory olisthesis. In the coronal plane, useful parameters are the following: 1) C7PL (plumbline from the center of C7 vertebral body) horizontal distance from CSVL (central sacral line), 2) scoliosis Cobb angle (major, hemicurve, and proximal thoracic), 3) proximal stable and distal stable vertebra of the major curve, 4) L3 endplate obliquity, 5) L4 endplate obliquity, 6) maximal lateral obli-thesis (mm), and 7) maximal vertebral rotation (Nash-Moe grades).

In the sagittal plane, parameters to be measured are the following: 1) sagittal Cobb angle (thoracic [T1–T12], thoracolumbar [T10–L2], and lumbar [T12–S1], 2) PLC7-S

<table>
<thead>
<tr>
<th>Type I: minimal or no rotation</th>
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<tr>
<td>Type II: rotatory olisthesis (intersegmental rotation and translation)</td>
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<tr>
<td>Type III: rotatory olisthesis and structural coronal (&gt;4cm distance from C7 plumbline) or positive sagittal imbalance (&gt;2cm from anterior sacral corner)</td>
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<tr>
<td>A: back pain without radicular sx</td>
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<tr>
<td>B: sciatic pain (from the lumbosacral hemicurve) ± back pain</td>
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<tr>
<td>C: femoral pain (from the major curve) ± back pain</td>
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Table 1

horizontal distance of the C7 plumb line starting in the center of C7 body and the anterosuperior corner of S1, 3) maximal anteroposteriorolisthesis (mm), and 4) sagittal pelvic tilt index [32].

The most predictive radiologic parameters for the associate clinical presentation of the patients with adult scoliosis are the upper endplate obliquities L3 and L4, lateral olisthesis, lumbar lordosis, and thoracolumbar kyphosis [32]. The positive sagittal balance has been shown to correlate statistically significantly with the clinical presentation, preoperatively and postoperatively [34].

In order to assess the degree of spinal stenosis it is imperative to review a CT scan or MRI of the lumbar spine. Especially the T2 sequence of the MRI shows the size of the spinal canal and the existence or intrathecal fluid in the transverse sections, whereas the patency of the foramen is better shown in the sagittal views. The myelography is not used so often nowadays because of its side effects; however, the CT myelogram gives a profound demonstration of those levels with spinal stenosis (Figs. 1–3).

Nonoperative treatment

Requirement for surgical treatment of degenerative scoliosis is the application and failure of nonoperative treatment. The first step of pain management is the use of nonsteroidal antiinflammatory drugs and nonnarcotic analgesics as well as the avoidance strenuous spinal motions. A session of oral administration of methylprednisolone is prescribed in cases of acute onset severe pain. Tricyclic antidepressants (amitriptylin) and anticonvulsion (carbapentin) drugs are preserved for cases of chronic pain. In case of symptoms persistence, the use of custom-made spine immobilization and vertebral joints’ injections follow. Epidural steroid injections may provide short- or long-term relief of symptoms. They are performed mainly for radiculopathy but also for back pain with radiographic spinal stenosis. Translaminar injections should be done always under fluoroscopy in order to avoid missing the target. Transforaminal epidural injections are indicated for one-level stenosis with unilateral symptoms and are certainly more selective to the location of spinal stenosis than translaminar injections. The

Fig. 1. (A) Anteroposterior and (B) lateral view full spine of a patient with degenerative rotary scoliosis with the apex to the left. Note the lateral olisthesis at L2–L3 and L3–L4 and the positive sagittal imbalance.
The time delay between epidural injections should be at least 3 weeks, and the total number of injections allowed is no more than three to four within a 6- to 12-month interval. In a recent randomized prospective study to assess the efficacy of epidural steroid injections versus intramuscular steroid and anesthetic for patients with lumbar stenosis, it was found that even though there was a significant reduction of pain in the short-term the long-term results did not show any difference in the number of patients that needed surgery. Furthermore, the systematic side effects (gastrointestinal, circulatory, haematological, and renal) from the prolonged use of nonsteroidal anti-inflammatory drugs should be kept in mind and their prescription be limited only for short-term use.

Operative treatment

All nonoperative ways of treatment should be exhausted before considering surgical treatment. The aim of the surgery is to decompress the comprised neural elements, in case of symptomatic spinal stenosis and to end with a balanced and stable spine in the coronal and sagittal plane, when there is imbalance. However, these patients are elderly patients with other comorbidities, and surgical treatment should be performed in a safe and effective way. The ideal is to proceed with only the least aggressive procedure, usually posterior only, that would involve both decompression and stabilization, if needed, of the spine.

The most frequent indication of surgical treatment is pain. However, the degree of any individual patient is very objective and cannot be measured independently. Usually, the most important question that a patient has to answer is how much his/her symptoms affect his/her life. The patients who present with back pain only should be warned that they would not be completely pain free, especially after a fusion surgery.

Progressive neurologic deficit is another indication for surgery. Increasing leg weakness and paresthesias that are not caused by vascular pathology should make the surgeon to suspect spinal stenosis. Symptoms originating from spinal stenosis typically appear between the 5th and 7th decade of life (40–60 years). Blood loss should be controlled to minimum amounts. Finally, bone quality should be examined before undertaking a surgical procedure, especially in aged patients, in case of instrumented fusion.

Patients who present with symptoms of spinal stenosis and have degenerative scoliosis less than 20° and without instability could be treated with spinal decompression only. Especially, male patients with large vertebral structures and stabilizing osteophytes can tolerate more than 2-level laminectomy without fusion.

Otherwise, patients with degenerative scoliosis more than 15° to 20°, lateral subluxation, or dynamic instability should be treated with decompression and fusion. Simmons selects different fusion strategies according to his classification’s types of degenerative scoliosis. For type I, he prefers the short instrumented fusions with distraction forces applied to the concavity of the curve in order to decompress the nerve roots. For type II, he prefers the long instrumentation and fusion with a derotation maneuver for scoliosis reduction. In situ rod contouring into lordosis and proper patient bed positioning are methods for restoring better mechanical alignment and lordosis. A less extensive fusion surgery can be preferable even though the upper and lower end of the fusion are not parallel to the sacrum.
Pedicle screws are considered the most appropriate fixation method for the aged osteoporotic bone with absent posterior elements after decompression [55]. In case of significant decompensation in the sagittal and coronal plane, it is often imperative to extend the fusion to the sacrum. Additional fixation points in the sacrum and the pelvis reduce the increased strains to the implants and help the healing of the fusion.

In Table 2, an algorithm of treatment of patients with degenerative scoliosis and stenosis is presented based on the classification system presented in Table 1. An extensive study of our patients treated using the aforementioned algorithm is under publication and showed statistically significantly improved outcomes (measured by SF-36, Oswestry, and VAS scores) postoperatively.

Results of surgical treatment

The degree of pain relief after adult scoliosis surgery is debatable. There are not many studies assessing the results of surgical treatment for adult patients with scoliosis and stenosis. Simmons and Simmons [43] studied 40 patients with 44 months average follow-up treated with decompression and fusion. Eighty-three percent of the patients reported severe pain before surgery, and 93% reported mild or no pain postoperatively. Scoliosis was reduced from 37° to 18° at the latest follow-up. Shapiro et al. [56] reported 94% satisfaction rate at 2 years postoperatively in 16 patients treated with anterior and posterior surgical reconstruction for adult idiopathic scoliosis and stenosis. The Oswestry Disability Index improved from 44.3 and preoperatively to 26.4 at the most recent follow-up, and the curve correction reached 50.4%. Hansraj et al. [51] in a study involving 47 patients with complex lumbar stenosis only (without scoliosis) and decompression surgery reported 89% to 98% satisfaction rates in different functional outcomes (ability to walk, pain, muscle strength and balance) with 92% to 94% chance of not needing revision surgery average 39 months postoperatively.

Frazier et al. [57] reported 15 patients with scoliosis and stenosis treated with lumbar decompression only. All the patients had postoperatively increased VAS score compared with preoperatively, even though the improvement of the scoliotic curve did not correlate with the outcomes.

Fig. 3. (A) Anteroposterior and (B) lateral view full spine after anterior interbody fusion L1–L4, posterior decompression L3–L5, and posterior instrumented fusion T9–S2.
The procedure can involve posterior only or combined anterior and posterior approach. Marchesi and Aebi [55] treated 27 patients with an average age of 60 and curves from 22° to 82° with decompression and posterior segmental instrumented fusion. In 4.5 years of average follow-up, 86% of the patients were satisfied with pain relief and increased walking ability. The average curve correction was 50%, and lumbar lordosis increased from 45° to 54° [55]. Grubb et al. [58] reported 40% pseudoarthrosis rate in patients with degenerative scoliosis treated with posterior-only fusions to the sacrum, whereas all the cases treated with anteroposterior approach fused uneventfully. A comprehensive and long follow-up study of the outcomes (including VAS, Oswestry Disability Index, or functional scores) of operative treatment in a large cohort of patients with degenerative scoliosis and stenosis is lacking.

### Complications of surgical treatment

The complications of the surgical treatment of degenerative scoliosis with stenosis can be multiple and risky [44,48,59–61]. With reference to age of patients who had anterior fusion for scoliosis, McDonnell and associates [62] noted a 41% (14% major and 27% minor) incidence for ages 41 to 60 years and 64% (24% major and 40% minor) for ages 61 to 85 years. The most common category of major complication was the pulmonary and the most common minor the genitourinary. Age more than 60 year was a risk factor for perioperative complication [62].

Patient positioning and procedure length should be taken into serious consideration for complication’s prevention. Surgeons should not hesitate to terminate an operation when some goals have been achieved and there is risk for the patient if the operation continues [60]. Surgery can be resumed later as a second stage.

In the literature, there is a controversy in the question if comorbidities increase the risk for postoperative complications [60,61]. Elderly patients could safely have spine surgery with an outcome similar to younger patients, if we deemed that they do not have increased comorbidities [61].

The most important complications that can happen are serious blood loss, blindness, cerebral infraction, myocardial infarction, paralysis, neurological deficit, infection, sepsis, failure or breakage of the implants, pseudoarthrosis, pneumonia, deep vein thrombosis, and death [59]. A special report should be performed regarding lumbar lordosis loss or flat-back syndrome [63]. It is caused by distraction of the lower lumbar spine during fusion or translational kyphosis of thoracolumbar spine cranially to the upper end of fusion. It leads to sagittal plane imbalance of the spine, chronic low back pain, and inability of keeping erect posture. Possible iatrogenic aetiology of flat back syndrome should be avoided during thoracolumbar scoliosis surgery.

### Conclusions

Degenerative scoliosis is appearing more frequently because of increased life duration or iatrogenic instability. Nonoperative treatment should be exhausted before proceeding with surgical treatment, especially in cases needing major reconstructive procedures. The optimal is to manage the whole pathology with only one operation. Lumbar decompression, fusion, and instrumentation are appropriate for most of the patients with degenerative lumbar scoliosis and stenosis. The main goal is to provide pain relief and improved functional lifestyle with minimum intervention.

### References
