



Focal disorders of the spine with compensatory deformities: how to define them

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Abstract

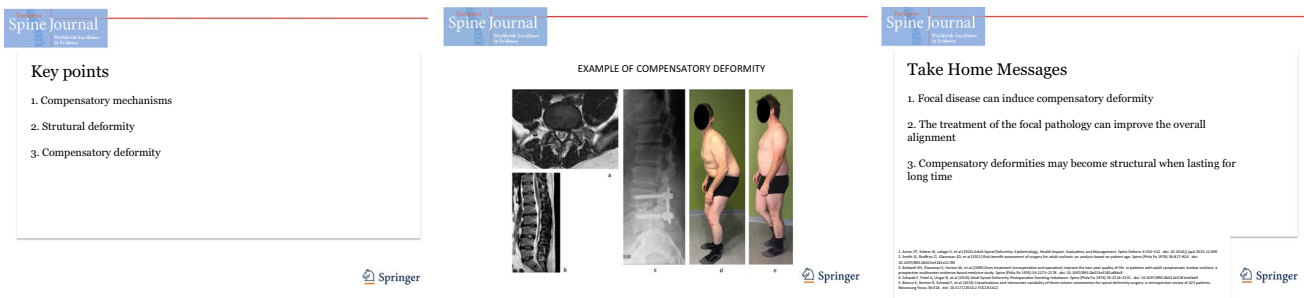
Purpose In this paper, the authors propose classifying the epiphenomenon of spinal deformity in two different categories: structural deformity, when the main driver of the observed deformity is a fixed and stiff alteration of the spinal segments, and compensatory deformity, which includes cases where the observed deformity is due to focal abnormalities. This last category comprises, but is not limited to, spinal stenosis, spondylolisthesis, disc herniation, infection or tumor, hip disease or neurological disease (such as Parkinson's disease).

Method Narrative review article.

Results We analyzed the focal diseases of the spine that may cause a compensatory deformity inducing adaptation in the unaffected part of the spine.

Conclusion The compensatory mechanisms involved in adaptive deformity represent an attempt to maintain a global alignment, to escape from pain or to control body posture.

Graphical abstract These slides can be retrieved under Electronic Supplementary material.



Keywords Adult spine deformity · Sagittal balance · Structural deformity · Compensatory deformity · Hip–spine syndrome

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Introduction

In the last 20 years, adult spinal deformity (AD) has been one of the hot topics in the field of Spine Surgery. Thanks to more profound knowledge of the disease, better diagnostic tools and technological improvement in instrumentation and perioperative management the number of surgical procedures for this disease has dramatically increased [1]. Surgical correction of adult spinal deformity has proven to be effective, leading to superior clinical and radiographic outcomes compared to non-operative treatment, especially

when proper alignment is restored [2–4]. Nevertheless, surgical treatment of adult deformity remains challenging and demanding as demonstrated by high complication rates (8.4–42%) [5, 6] and revisions (9–17.6%) [7, 8]. Moreover, surgery is able to achieve the proper correction in only 32% of cases, while 68% of patients remain under- or overcorrected [9]. These data may explain why the rate of revisions increases progressively over years [10] and can even reach 30% of cases [8]. Furthermore, there is still some controversy on how to measure the ideal lordosis and the ideal PT [11]. Some authors suggest that the treatment should be tailored on each patient [12]. Other have observed that pelvic incidence, the cornerstone of Sagittal Balance, can vary with age [13] and sometimes after surgery [14].

A balanced spine is defined by the ability of a subject to maintain the standing position with the minimal muscular effort. This definition implies that every disease that alters the normal equilibrium of the spine can be defined as a cause of sagittal imbalance. However, sagittal imbalance and even the worst cases of deformity can be perceived by the patients as a normal and asymptomatic aging process [1]. When the original shape of the spine changes because of focal degenerative disease, trauma, infection or tumors, subjects adapt their position to restore a global alignment. As a consequence, the main characteristic of adult spinal deformity is the presence of compensatory mechanisms that rely on additional muscular effort and that can become painful and lead to disability over time. The compensatory mechanisms are well known and include the possibility to increase lordosis or decrease kyphosis of the different portions of the spine.

The pelvis is a key factor in the study of sagittal alignment and is responsible for retroversion, the strongest compensatory mechanism. Pelvic retroversion consists of the posterior rotation of the pelvis around the femoral heads, and at its extreme range, leads to complete hip extension. Considering the relation $PI = PT + SS$, the amount of pelvic retroversion can reach at most the value of PI when the sacral endplate is horizontal ($SS = 0^\circ$) and for this reason individuals with high PI have wider range of adaptation. This movement is very effective in compensating the anterior translation of the gravity line and corresponds to an increase of PT that is correlated to back pain and disability [15]. When maximum retroversion of the pelvis is reached, the next step is knee flexion and ankle extension to obtain a more efficient position of the gravity line [16].

Lamartina and Berjano proposed in 2014 a classification of sagittal deformity based on segments involved in the deformity and on compensatory mechanism acting on the spine [17]. This classification is helpful in recognizing the segments of the spine affected by the deformity and the portions of the spine that adapt in response to deformity to treat only the pathology and not the compensation [18].

Structural or compensatory deformity?

Clinical observation suggests that the same compensatory mechanism involved in deformities can be found in frequent degenerative spinal disorders and other orthopedic and neurological conditions. Thus, we can divide spinal deformity in two different categories: structural deformity on one side and on the other all the diseases that act like a deformity inducing adaptation in the spine, pelvis and legs but that are reversible and so can be considered compensatory deformities due to focal pathologies. The first group often requires correction of the deformity with tricolumnar osteotomy [19] or less aggressive procedures such as multiple approach surgeries [20] to restore the ideal balance between spine and pelvis. Lafage and Schwab observed that a strong correlation exists between HRQOL and PI-LL mismatch, SVA and PT [15] and suggested that the objectives of the surgery for sagittal deformity should be $SVA < 50$ mm, $PT < 20^\circ$ and $PI-LL < 10^\circ$ [4], although the ideal goal of surgery is still debated.

On the contrary, an adaptive spinal pathology has similar presentation of deformity but the basic concept is that the treatment of the focal pathology can restore the sagittal and/or frontal balance without any further measures, but just by removing the focal pathology. Structural and compensatory deformities may therefore be clinically similar, but their treatments significantly different [21].

Clinical presentation among the two groups is similar and thus cannot be diriment for a correct diagnosis. Conversely, radiological imaging is very useful to differentiate a structural and fixed deformity from a compensatory one. As we are going to see in the next paragraphs, symptoms related to focal disease underlying a compensatory deformity often exacerbate in standing positioning and improve when laying down. For this reason there can be significant difference in spinal alignment between standing X-rays and non weight-bearing imaging such as MR and CT scan. Moreover, the number of segments altered by the disease is generally larger in structural deformity while compensatory deformities derive from a focal disease that involves usually involves the single vertebra or disc. Unfortunately, this is not always the case, especially in elderly people with multilevel spondylosis that can make the diagnostic process more intricate.

Finally, we must remember that an adaptive deformity may become structural when lasting sufficiently long to determine degenerative alterations even in the compensatory parts of the spine. In this scenario, compensatory mechanisms turn into structural deformity and they cannot be reverted by the treatment of the focal pathology that originated them. A good example is a relative leg length discrepancy, which may lead to a lumbar compensatory

scoliosis. If such a deformity persists for years, then the compensatory scoliosis becomes a structural curve, which can no longer be corrected equalizing the leg length [22].

Focal pathologies, which may lead to a compensatory spinal imbalance

Degenerative spondylolisthesis

Degenerative spondylolisthesis (DS), one of the most frequent causes of pain and disability in adult population, has a strong correlation with spino-pelvic parameters: some authors found that high value of PI is a predisposing factor for the development of degenerative spondylolisthesis [23].

Whether the focal disc disease with segmental kyphosis leads to compensatory malalignment or whether it is the local pain to induce antalgic secondary alterations of the whole spine is not entirely clear. Nor it is clearly established whether the degenerative spondylolisthesis itself is already per se the result of a long lasting compensatory mechanism for sagittal alignment restoration.

A recent paper showed that upper lumbar segments are more extended in patients affected by degenerative spondylolisthesis [24]. Hyperextension of adjacent segments is a very common local compensatory mechanism to limit the consequences of local kyphosis due to listhesis. Local extension can be efficient but leads to retrolisthesis, disc disease and increase the stress over facet joints [16]. Liu et al. observed that this kind of compensation occurs with healthy adjacent disc while in case of degenerative adjacent segments pelvic retroversion is involved as a further step of adaptation [25]. There is still large debate about the right treatment for degenerative spondylolisthesis. While some research shows that the clinical results with decompression alone are satisfactory [26, 27], the same studies do not explore the specific reciprocal contribution of surgical re-alignment to clinical results and of decompression to re-alignment. At least one study has suggested that surgical maneuvers resulting in focal increase of lordosis in the treatment of degenerative spondylolisthesis resulted in additional improvement in clinical outcomes [28].

Lumbar spinal stenosis

Lumbar spinal stenosis (LSS) is the most common indication for surgery in people older than 65 and is often associated with degenerative spondylolisthesis [29, 30]. Claudication, pain, numbness and weakness in the buttocks and legs are caused by standing and walking and relieved by sitting and rest. Symptoms are often posture dependent: they appear with lumbar extension and improve with forward flexion. As a consequence people affected by LSS can take severely stooped posture aiming pain relief. Typically spinal stenosis

patients, who have difficulties to walk, can easily ride a bicycle without presenting leg symptoms with exercise. Shin et al. showed that simple decompression surgery is able to restore good alignment in 70% of patients affected by sagittal imbalance caused by LSS [31]. Similar results were reported by Fuji et al., who observed how surgical decompression in stenotic patients was followed by improvement in alignment parameters (reduction of SVA and pelvic tilt, increase of lumbar lordosis) [32] (Fig. 1).

While stenosis can behave as a focal cause of deformity, it can be present in association with true structural deformity. How to distinguish between these two subsets of patients is not clear. Dohzono et al. studied how the magnitude of preoperative sagittal misalignment influenced the final result after laminectomy for stenosis. They found that patients with greater than 50 mm preoperative SVA had more pain post-operatively than those with smaller than 50 mm preoperative SVA, though both groups had similar percentage of improvement [33].

Similarly, Hikata found that patients with severe preoperative sagittal imbalance (SVA > 80 mm) had significant residual sagittal imbalance after decompressive surgery [34].

A different study [35] found that when mild degree of sagittal malalignment was present, patients with structural deformity had increased pelvic tilt while patients with stenosis who presented in a forward standing position to relieve compression (that is, with adaptive deformity) had increased SVA with normal pelvic tilt. This observation matches the “pelvic kyphosis” sagittal deformity category described by Lamartina and Berjano [17], who hypothesized that increased SVA with normal PT should raise the suspect of non structural deformity (stenosis, hip disease or neurological disease such as Parkinson’s or dystonia).

Lumbar disc herniation

In a very similar way, patients affected by lumbar disc herniation develop sagittal and sometimes coronal malalignment as a protective mechanism against sciatic pain. Patients with LDH show anterior translation of SVA, loss of lumbar lordosis and vertical sacrum as demonstrated in a study by Endo et al. [36]. These changes, even if are of the same sagittally imbalanced patients, are not likely supported by a structural deformity, rather by contraction of lumbopelvic muscles as antalgic response (Fig. 2).

It is a common experience that conservative treatment and simple microdiscectomy (when indicated) can restore a normal alignment as soon as the pain disappear. Liang et al. have demonstrated that sagittal alignment parameters change after surgery in patients with disc herniation, with reduction of SVA and pelvic tilt and increase of lumbar lordosis, suggesting that the preoperative alignment includes

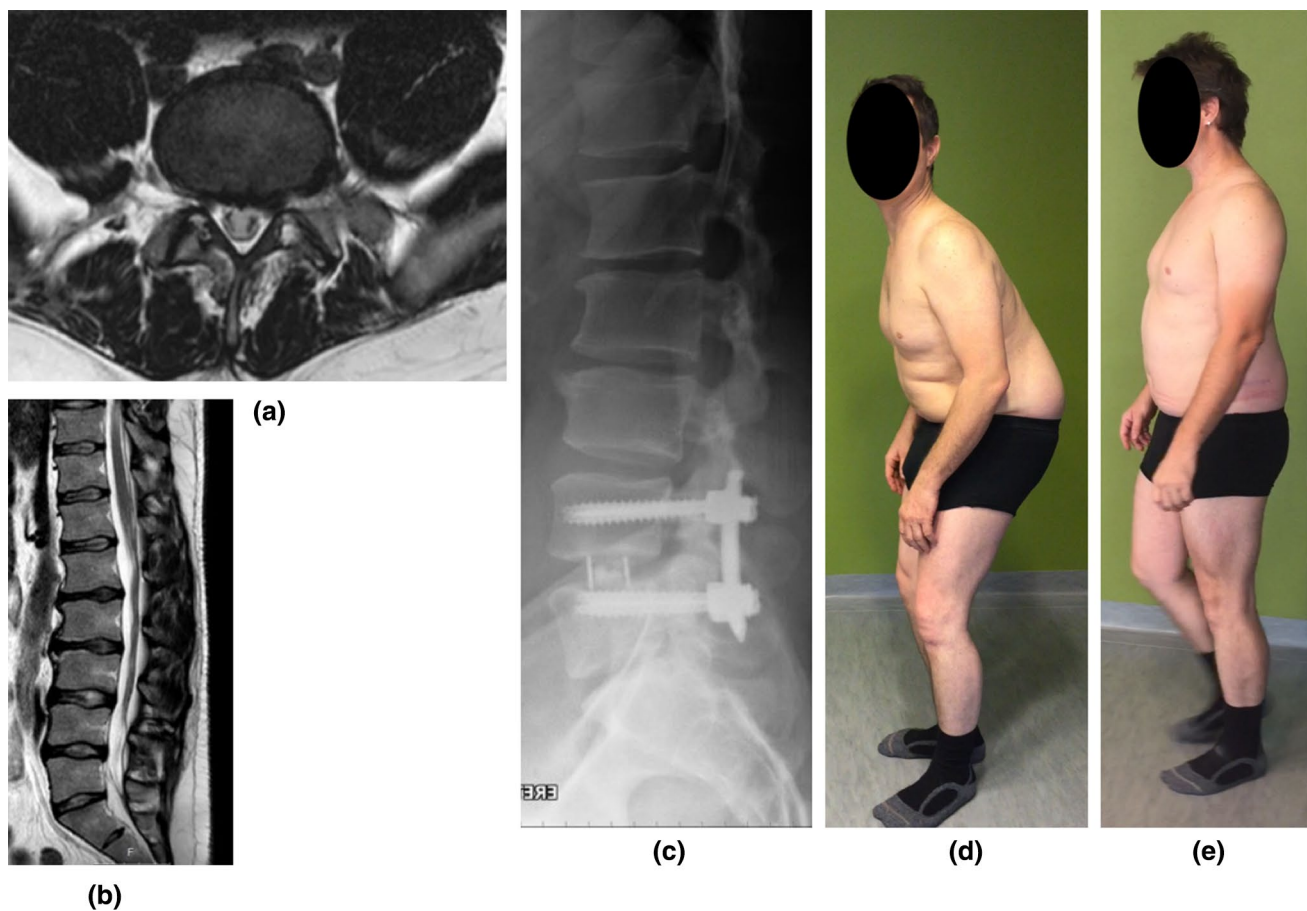


Fig. 1 A case of left foraminal stenosis (**a**, **b**) causing evident trunk flexion (**d**) and the restoration of a normal alignment after indirect decompression (**c**, **e**)

forward flexion of the trunk and retroverted pelvis to reduce the nerve root impingement [37].

Isthmic spondylolisthesis

Another condition that can affect sagittal balance is isthmic spondylolisthesis [38]. One of the key aspects in the evaluation of isthmic spondylolisthesis is lumbosacral kyphosis. When it is present, patients need to compensate for it, with increased lumbar lordosis (above L5) and/or increased pelvic tilt. These deformities can be reverted by reduction of spondylolisthesis according to Labelle and MacThiong who observed a strong correlation between percentage of slippage and high PI [39, 40]. They identified six categories of L5-S1 spondylolisthesis according to the degree of slippage and the value of PI. The most severe types are characterized by lumbosacral kyphosis that can be compensated only by pelvic retroversion. When high grade spondylolisthesis determines unbalanced pelvis and spine, surgical reduction is mandatory to achieve better outcome and restoration of a normal shape of the spine [41, 42] (Fig. 3).

Focal neoplastic and infective disease

Focal metastatic disease as well as spondylitis/spondylo-discitis may induce a malalignment in the sagittal as well as frontal plane. The focal pathology can here act as a pain source with the consequence of a protective posture, or the malalignment can be due to a damaged vertebra/segment, enhanced by the pain dependent posture. The removal of the focal pathology through decompression and/or stabilization may automatically restore the normal alignment.

Intraspinal pathology: intraspinal (extradural/intradural tumors, cysts, etc.)

Intraspinal pathologies such as tumors and cysts may induce likewise a disturbed sagittal and/or frontal malalignment. The removal of the focal pathology may automatically restore the normal alignment.

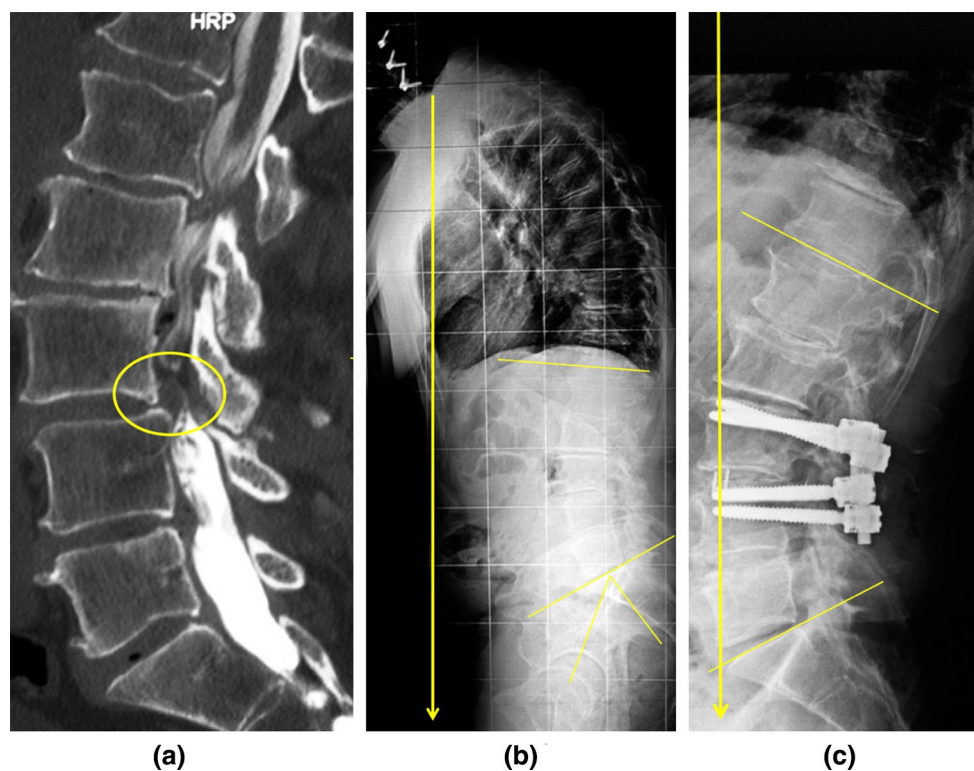


Fig. 2 This patient had a significant disc herniation determining a stooped position of the whole spine (a, b). After decompression and fusion, the patient regained a normal spinal alignment (c)

Hip disease

There is certain evidence that sagittal alignment can be altered also by diseases that occur outside the spine. Hip osteoarthritis (HOA) is a very common disease and in the last 50 years arthroplasty literally revolutionized the surgical management of this pathology so that total hip arthroplasty has been defined the operation of the twentieth century [43]. The association of HOA and low back pain (LBP) is very frequent and was defined as hip–spine syndrome (HSS) by Offierski and MacNab that observed improvement of LBP after the treatment of HOA [44]. The connection between hip and spine has been extensively investigated over the years but the exact mechanism of HSS is still not completely clear. The hips play a key role in maintaining a postural balance so that the term “hip strategy” was introduced by Nashner and McCollum to explain the complex mechanisms that involve the pelvis in adapting its position to postural changes [45]. A stiff hip in a fixed flexed deformity causes an obligated antevergence of the pelvis that is unable to retrovert to compensate a spinal deformity. On the other hand, in people with healthy spine, a fixed anteverted pelvis determines a compensatory increase of lumbar lordosis that leads to facet joint overloading and arthritis. Weng et al. observed that people affected by severe HOA are characterized by more

anteverted pelvis, forward tilted spine and flexed hips than healthy controls [46]. However, conflicting observations have been reported regarding sagittal spino-pelvic alignment in patients with HOA. Although total hip arthroplasty should allow the restoration of a full range of motion, and thus the pelvic compensation, this hypothesis is not supported in the literature and pelvic tilt shows little changes after hip replacement (Fig. 4).

Another paper published in 2016 showed that LBP improved after total hip replacement in patients affected by HSS but no significant change in spino-pelvic alignment was observed [47]. This suggests that there should be some other mechanism underlying the improvement of back pain after hip arthroplasty, and thus the exact explanation of HSS remains unclear.

However, another study regarding changes in alignment in patients undergoing surgery for hip osteoarthritis showed that 1 year postoperatively these patients had a subtle but consistent and statistically significant increase in hip extension and a posterior translation of T1 in the standing position. Patients with preoperative low back pain had an improvement on low back pain and lumbar spine function 1 year after hip replacement [48].

How far the iliosacral joints play a key role in the pain etiology is not clear. With limited motion in the osteoarthritic

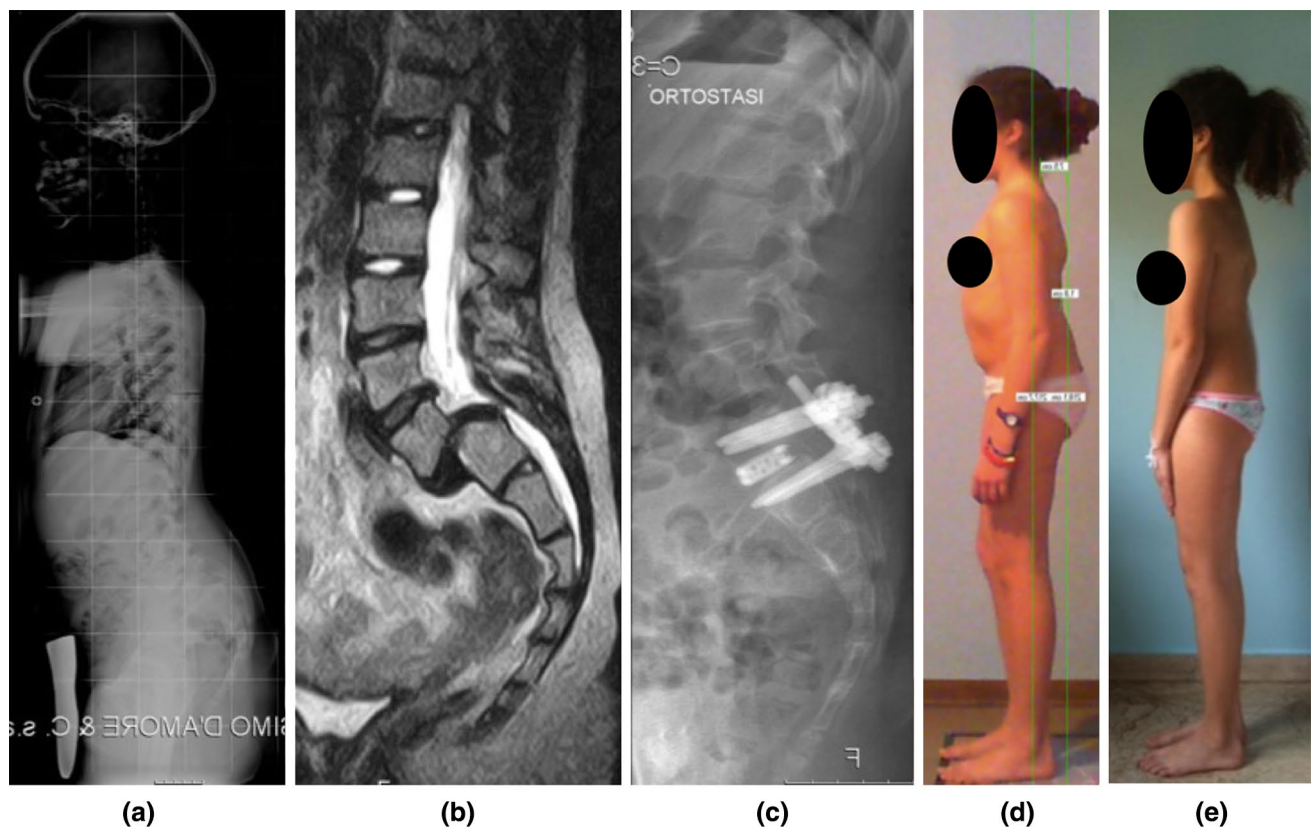


Fig. 3 This patient with grade 4 spondylolisthesis (Labelle and MacThiong's type 5) had a preoperative deformity with L5-S1 kyphosis determining compensatory hyperlordosis, reduction of tho-

racic kyphosis, increased pelvic tilt and knee flexion. After correction of the focal deformity (reduction of both L5 kyphosis and olisthesis), LL and PT have been normalized

hip joints, the iliosacral joints may become “loose” and allow a bigger extent of motion, what may also contribute to the unchanged pelvic tilt due to compensatory mechanisms in the iliosacral joints.

Leg length discrepancy

Leg length discrepancy determines pelvic obliquity, and hence, a scoliosis. The main feature of such a deformity is not a relevant rotation but a coronal deviation of the spine which helps to maintain an efficient position of the gravity line. Leg length equalization, when applied before growth completion, results in elimination of compensatory scoliosis [49].

Neurological conditions causing sagittal misalignment

Furthermore, Ferrero et al. investigated the sagittal alignment of a particular group of individuals with low pelvic tilt despite forward flexion of the spine [50]. This feature seems to be related to the inability to recruit the extensor muscles of the hips due to alterations in the neuromuscular control of posture. This unusual category was already described by

Lamartina and Berjano, who suggested a correlation with Parkinson disease [17]. Parkinson disease (PD) is known to be strongly correlated with spinal deformity that can be defined as flexible since it worsens in standing position and may reduce when laying down. The deformity in PD can involve either the sagittal or the coronal plane (or both), and camptocormia and Pisa syndrome are the two terms that indicate forward and lateral flexion of the spine, respectively [51], when it is caused just by the neurological condition (Fig. 5).

Although the exact pathophysiology of spinal deformity in PD is not clear, some papers demonstrate a correlation between the grade of deformity and the severity of neurological disease [52, 53]. This association may depend on muscular rigidity, dystonia, impaired proprioception, myopathy and can also explain the outcomes of the surgical correction of this kind of deformity that are generally poorer than in patients without PD [54].

Sagittal misalignment and idiopathic scoliosis

People affected by idiopathic scoliosis are a population that escapes from the rules of sagittal balance. Since the

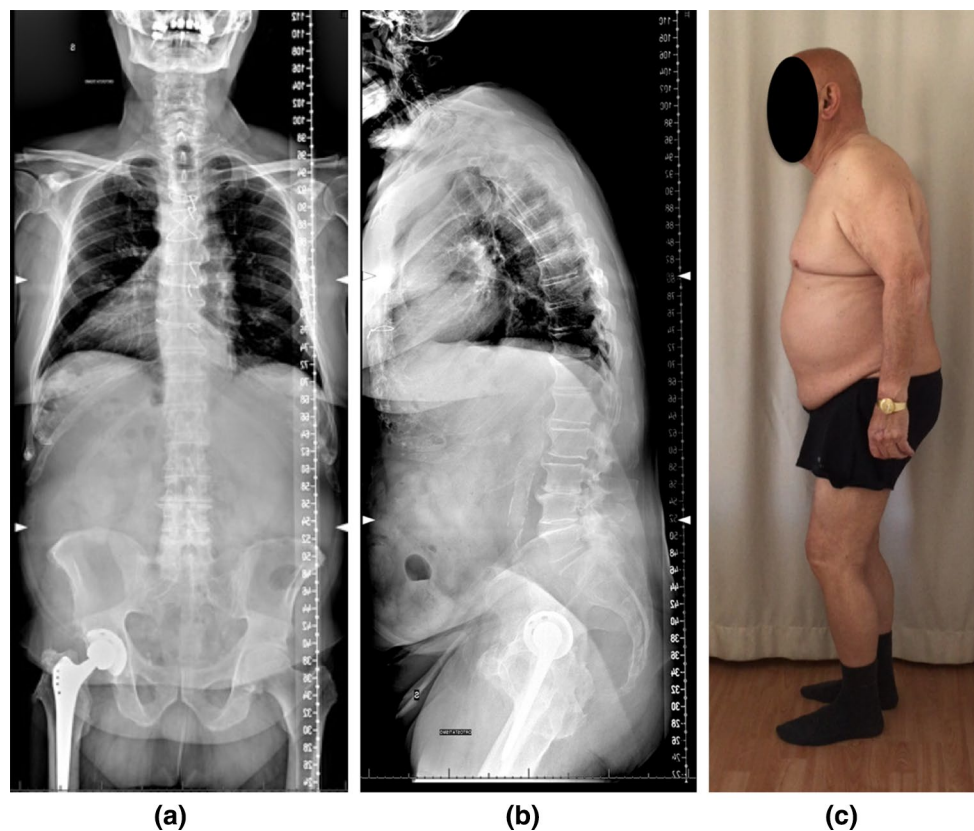


Fig. 4 This patient has bilateral hip disease (left hip replacement and right hip osteoarthritis). Bilaterally, the hips had rigid flexion contracture. The replaced hip has substantial acetabular anteversion that prevents the hip from fully extending. Consequently, the patient has developed knee flexion that has become rigid and forward flexion of

the trunk not compensated by pelvic retroversion. This corresponds to a Lamartina and Berjano's pelvic kyphosis with the characteristic findings of high SVA with low pelvic tilt, indicating a non-spinal cause of forward trunk inclination

childhood, a period in which big transformations of posture occur [55, 56], they develop an altered sagittal and coronal imbalance without any symptom. La Maida et al. observed that adolescents with idiopathic scoliosis have a slightly posterior imbalance without the activation of any compensatory mechanism despite the lumbar lordosis is less than expected [57]. The same authors noticed that scoliotic patients remain hypolordotic after surgery even if the value of lordosis increases compared to preoperative condition. Moreover, thoracic kyphosis in scoliosis population is also reduced and the correlation between spino-pelvic parameters is weaker than in adults, suggesting a more “immature” control of posture in adolescents [58]. These data suggest that scoliotic patients spend most of their lives with an altered imbalance without any kind of disability (Fig. 6).

A similar situation is the one of subjects with lumbosacral transitional vertebra, a common congenital anomaly that occurs in up to 20% of general asymptomatic population. This condition has been proven to be correlated to positive sagittal balance despite complete absence of symptoms [59].

As previously mentioned, compensatory mechanisms constitute the distinctive trait of both structural and adaptive deformity. In the first case, they seem to be the attempt to maintain a vertical standing position in a spine altered by the deformity; in the second case, a compensatory mechanism may be considered as an involuntary response to pain evoked by a focal pathology that alters the form of spinal alignment.

Unsolved problems of sagittal balance

During the last 20 years, there has been the attempt to switch from a qualitative to a quantitative point of view in the study of sagittal balance. The identification of spino-pelvic parameters able to correlate with quality of life and the finding of ideal goal of surgery allowed to demonstrate the efficacy of surgical correction of spinal deformity and to measure its outcome. However, some points remain unsolved such as the correct alignment of cervical spine, the standard position for standing X-rays and again the difference between alignment and balance. The cervical portion of the spine is the most complex and

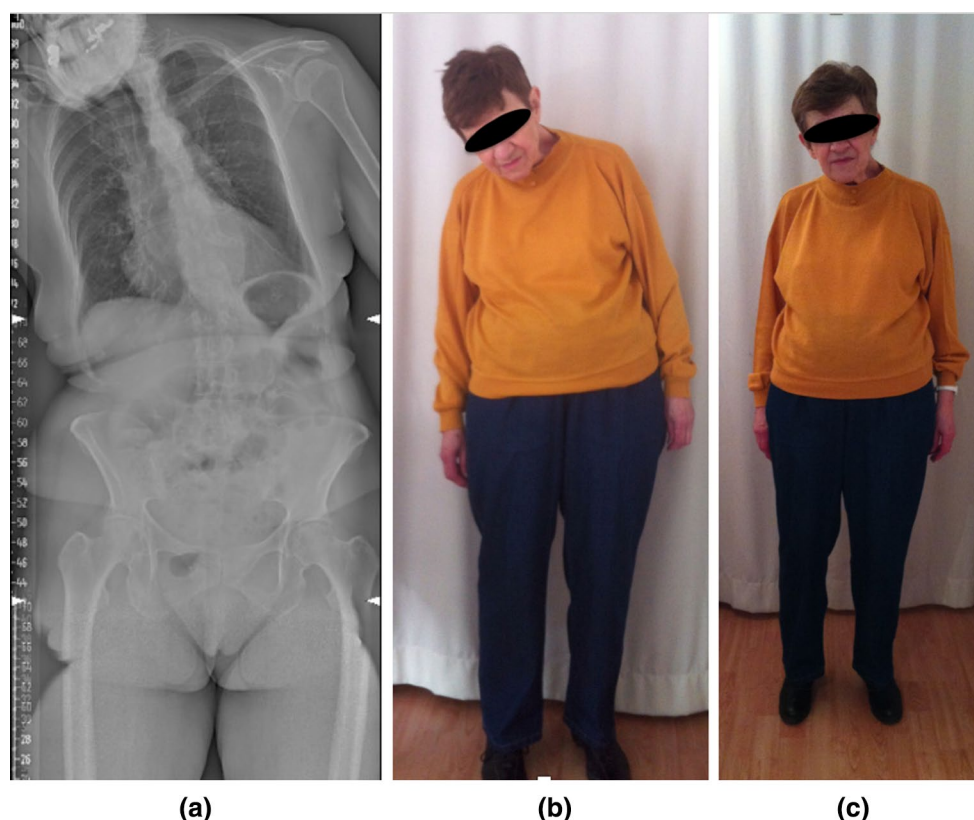


Fig. 5 This patient presented a lateral trunk and head deviation developed in few months (**a**, **b**). The patient had also extra-pyramidal type rigidity of the upper limb. Radiograph showed scoliotic attitude with

a long, unique coronal curve with little apical rotation (**a**). Therapy with antiparkinsonian drug determined significant improvement of coronal deformity (**c**)

the less known part of the spine: it guarantees the widest range of motion so that there are many “normal” cervical alignments that depend simultaneously on the mass of the head and on the position of the other parts of the spine. The increase of cervical lordosis in response to kyphotic degeneration of thoracic and lumbar spine is a well-known mechanism and the influence of cervico-thoracic junction on cervical alignment was demonstrated by Lee et al. [60]. However, the head and its content certainly affect the sagittal shape of cervical spine. Indeed, at these levels the load distribution relies more on facet joints than intervertebral discs, differently to what happens in lower segments of the spine. Moreover, opposed to the lumbar spine in which the majority of the lordosis is in the caudal part [61], in the cervical spine approximately 75–80% of lordosis lies between C1 and C2 [62]. These two findings can be explained by the presence of the head and its gravity center that is almost above the center of C1 and C2 vertebral bodies [63]. As the close connection between pelvis and spine allowed Jean Dubousset to identify the pelvis as the “pelvic vertebra”, in the same way we could refer to the head as “skull vertebra”, though a normative value such as pelvic incidence has not yet been found [64].

Besides, the whole sagittal balance theory is linked to the radiological acquisition of the spinal shape although there is still controversy about the optimal position for radiographic acquisition. The different positions of the arms can influence the sagittal alignment of the spine as suggested by different authors and these findings can constitute a bias that rarely is taken into account [65–67]. Nevertheless, independently from the position of the subject, X-rays are only able to describe static shape of the spine, and as showed by Dubousset, the alignment is only a complementary feature of balance for human standing position [68]. Balance is “stability within movement” and is a wider concept than simple alignment and involves vestibular, ocular, neurological and muscular properties that are all together linked to determine the reciprocal position of bones.

Compliance with ethical standards

Conflict of interest None of the authors has any potential conflict of interest.

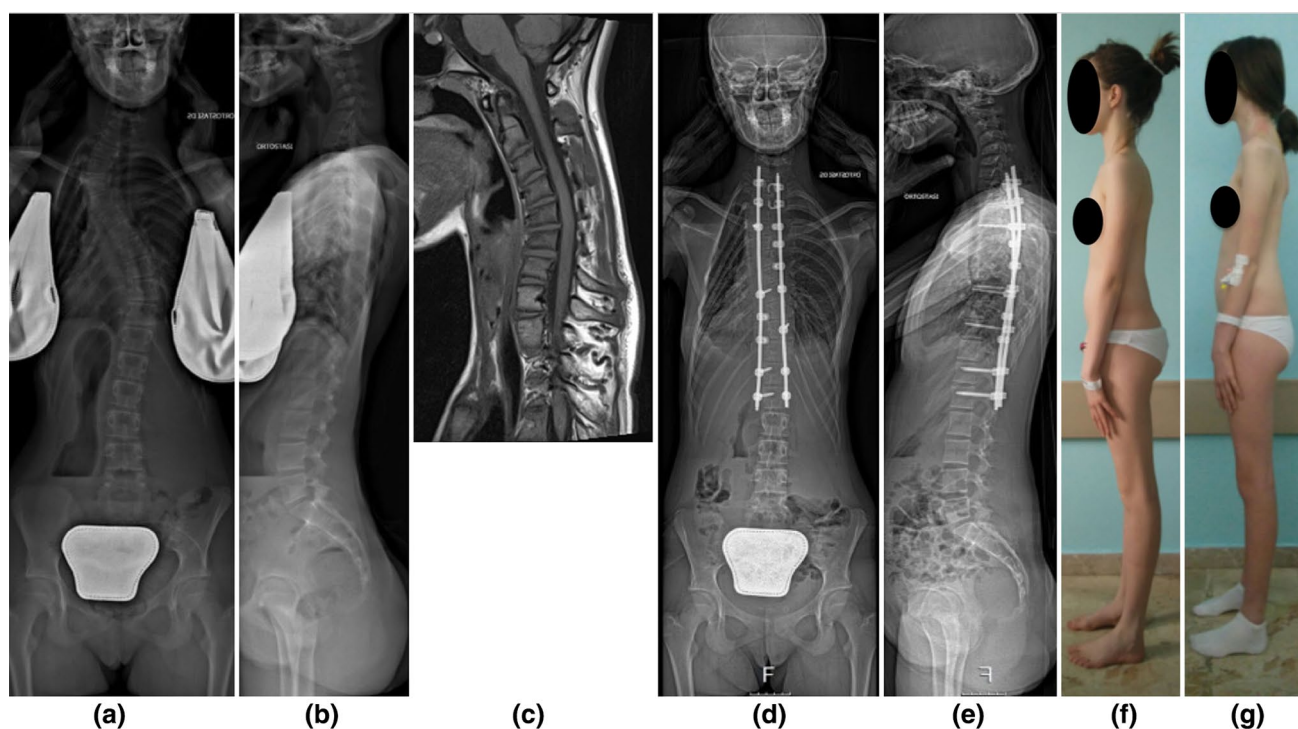


Fig. 6 A case of a Type 2A-adolescent idiopathic scoliosis (**a**, **b**) with a flat thoracic spine and cervical kyphosis (**c**). The correction of the deformity on the coronal plane was almost complete but little to none improvement in the sagittal plane was observed (**d**, **e**). Clinical

pictures showed a normal sagittal alignment both before and after surgery without compensatory mechanism despite the significant alteration of physiological curves (**f**, **g**)

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