

## A conceptual model of compensation/decompensation in lumbar segmental instability <sup>☆</sup>



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### ABSTRACT

Lumbar spinal instability (LSI) is a common spinal disorder and can be associated with substantial disability. The concept of defining clinically relevant classifications of disease or 'target condition' is used in diagnostic research. Applying this concept to LSI we hypothesize that a set of clinical and radiological criteria can be developed to identify patients with this target condition who are at high risk of 'irreversible' decompensated LSI for whom surgery becomes the treatment of choice. In LSI, structural deterioration of the lumbar disc initiates a degenerative cascade of segmental instability. Over time, radiographic signs become visible: traction spurs, facet joint degeneration, misalignment, stenosis,olisthesis and de novo scoliosis. Ligaments, joint capsules, local and distant musculature are the functional elements of the lumbar motion segment. Influenced by non-functional factors, these functional elements allow a compensation of degeneration of the motion segment. Compensation may happen on each step of the degenerative cascade but cannot reverse it. However, compensation of LSI may lead to an alleviation or resolution of clinical symptoms. In return, the target condition of decompensation of LSI may cause the new occurrence of symptoms and pain. Functional compensation and decompensation are subject to numerous factors that can change which makes estimation of an individual's long-term prognosis difficult. Compensation and decompensation may influence radiographic signs of degeneration, e.g. the degree of misalignment and segmental angulation caused by LSI is influenced by the tonus of the local musculature. This conceptual model of compensation/decompensation may help solve the debate on functional and psychosocial factors that influence low back pain and to establish a new definition of non-specific low back pain. Individual differences of identical structural disorders could be explained by compensated or decompensated LSI leading to changes in clinical symptoms and pain. Future spine surgery will have to carefully define and measure functional aspects of LSI, e.g. to identify a point of no return where multidisciplinary interventions do not allow a re-compensation and surgery becomes the treatment of choice.

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### Introduction

Lumbar spinal instability (LSI) is a common spinal disorder and can be associated with substantial disability. Management of LSI is

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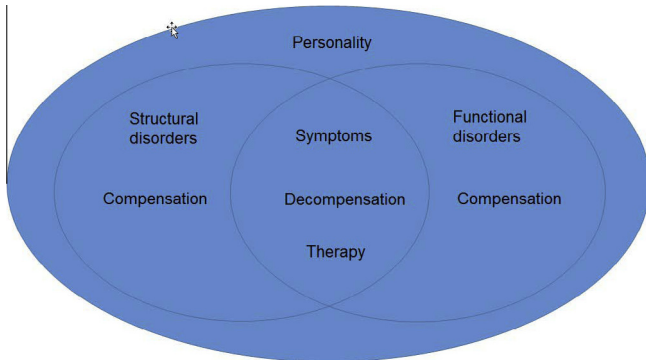
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challenging because the clinical and radiological criteria used for diagnosis are not yet clearly defined. Moreover, the existing diagnostic criteria do not perform well to distinguish between patients who are likely to benefit from conservative management and those who will not improve without surgical intervention. This has led to considerable uncertainty and wide variation in the selection of patients for surgery.

The concept of defining clinically relevant classifications of disease or 'target condition' is used in diagnostic research [1]. It is used to develop medical tests and cut-off points that can better

**Table 1**  
Definitions of lumbar segment instability.

- Reduced resistance to movement (Zhao et al., 2005)
- Ratio “neutral zone”/range of motion (Mimura et al., 1994)
- Increase “neutral zone”, or physiological limits (Panjabi, 1992)
- Condition in which a small load applied to a system causes a marked displacement (Ashton-Miller, 1991)
- Loss of the spine’s ability to maintain patterns of displacement under physiologic loads (White and Panjabi, 1990)
- Radiological retro-displacement of a vertebra during flexion (Knutsson, 1944)
- “Pseudo-spondylolisthesis” (Schmorl and Junghanns, 1932)



**Fig. 1.** Overlap of structural and functional lumbar segment instability. Adapted from Tsuij H: Comprehensive Atlas of Lumbar Spine Surgery. Mosby Year Book, St. Louis; 1990 [61].

inform clinical decisions, for example, the development of tests for staging patients with cancer to help discriminate between those at low versus high risk of disease recurrence to guide decisions for adjuvant chemotherapy. It is also used when evaluating tests, to design test accuracy studies to provide measures of test performance that are relevant to the decisions the test will be used to inform in clinical practice [1].

In this paper, we propose that the concept of defining the target condition to guide the development and evaluation of medical tests can be extended to improve clinical practice for conditions such as lumbar spinal degeneration, where more clinically meaningful definitions of disease are needed to guide treatment decisions. We discuss two of the most common degenerative disorders of the lumbar spine: structural or functional lumbar segmental instability, and lumbar spinal stenosis (LSS). For both conditions no consensus exists on the definition using clinical, radiological and pathological criteria (Table 1, Fig. 1) [2–7].

**Hypothesis**

Applying the concept of the target condition to lumbar spinal degeneration, we define the more severe spectrum of disease where symptom alleviation is unlikely to occur without surgical intervention as the target condition most relevant for clinical decisions. We hypothesize that a set of clinical and radiological criteria can be developed to identify patients with this target condition who are at high risk of ‘irreversible’ decompensated LSI for whom surgery becomes the treatment of choice. These criteria will be extremely valuable to distinguish between a ‘compensated’ condition where the patient is likely to remain in a compensated state of the disease – benefitting from conservative treatment; and a ‘decompensated’ condition where the patient has reached the point of no return into decompensation – requiring surgical treatment.

degenerative cascade (Three Joint Complex)		
Facets		Intervertebral Disc
		cellular/extracellular disorganisation
synovitis	Dysfunction	circumferential tears
capsular laxity		radial tears
subluxation	Instability	internal disruption
capsular hypertrophy		disc resorbtion
articular enlargement	„Stabilization“	osteophytes

**Fig. 2.** Degeneration of lumbar disc, i.e. degenerative cascade. Adapted from Kirkaldy-Willis WH and Bernard TN. Managing Low Back Pain. Elsevier Ltd., Oxford; 4th edition 1999 [62].

**Current concept of spinal degeneration**

The current understanding of degeneration of the spine is based on the concept of a degenerative cascade of anatomical structures within the lumbar motion segment, which comprises soft tissue (ligaments, joint capsules, local and distant musculature, intervertebral discs) and bony elements (two adjacent vertebral bodies, facet joints) [8–10] (Fig. 2).

In the lumbar motion segment model as introduced by Junghanns, the spinal segments are organized in a way that they permit individual movement [11]. Intervertebral discs, anterior and posterior longitudinal ligaments, facet joints, ligamentum flavum, parts of the vertebral body between the processus spinosus and transversus, and muscles interplay with one another for normal function. When a disorder or injury occurs, the whole motion segment is affected and the degenerative cascade begins which can lead to the symptoms and signs of LSI (and LSS).

**A new conceptual model of compensation/decompensation in lumbar spinal degeneration**

Given the poor correlation between clinical and radiographic variables for the diagnosis of LSI [12–15], a binary model of compensation/decompensation – where a period of compensation is followed by a single shift to decompensation – is likely to be an oversimplification of the natural history of LSI. Therefore, we propose a new conceptual model of compensation/decompensation that incorporates both aspects to reflect the more complex relationship between clinical signs and radiological findings that is observed in practice as outlined below.

Evidence demonstrating the association between electromyography (EMG) measured muscle activation and risk of low back pain (LBP) [16,17] suggests the functional elements of the motion segment are also influenced by non-functional factors such as psychosocial factors. The functional elements allow, within different individual limits, changes in function and position of the motion segment, and consequently, a compensation of the degeneration of the motion segment [18–20] (Table 2). They also influence the shape of the load/range of motion curve [21,22]. Compensation may happen on each step of the degenerative cascade but cannot reverse it. However, on each step, compensation

**Table 2**  
Influencing psychosocial factors on lumbar motion segment.

Mental health
Depression
Somatization
Fear-avoidance beliefs
Pain catastrophizing
Optimism/resilience
Marital/relationship status
Educational status
Social support

**Table 3**  
Definition of compensation, adaptation, and decompensation according to M. Forte, Ewen B. [Osteopathic procedures and osteopathic medicine. A manual medicine osteopathic career in chain reactions]. *Manuelle Med.* 2013;51:291–294 [60].

<i>Compensation</i>
Physiological reaction of a body part on functional or structural variation of another body part
<i>Adaptation</i>
Adaptation of tissues to maintain functional compensation
<i>Decompensation</i>
Imbalance of a body part caused by a conflict between at least 2 different mechanisms of compensation or adaptation

of LSI may lead to an alleviation or resolution of clinical symptoms. The functional elements of the motion segment are organized in a cybernetic model where the command variable may change (e.g. pain, function, and pressure relief of the nerve). It remains unclear whether a potential overload of this model might lead to degeneration per se. In return, if compensation is no longer possible, a distinct new target condition is reached: decompensation of LSI which may cause the new occurrence of clinical symptoms and pain [23–25]. The different individual limits in which compensation takes place decrease over time and are dependent upon overload of the functional elements and progression of degeneration.

In osteopathic and manual medicine the term compensation is used in a similar way. In contrast, decompensation is understood as conflict of opposing mechanisms of functional compensation or structural adaptation (Table 3).

**Table 4**  
Influencing genetic/physiological factors of degenerative cascade.

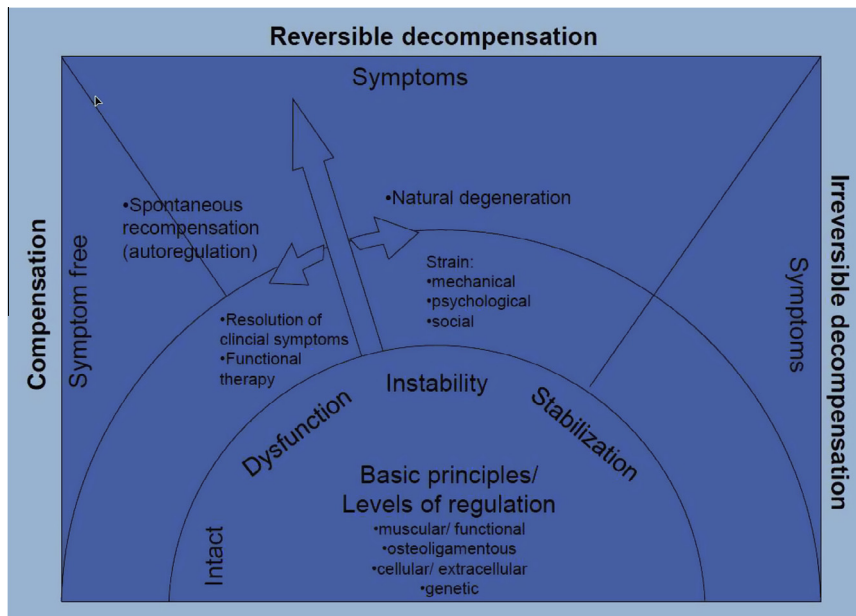
Segmental spinal mobility
Gait
Unequal leg length
Body mass index
Serum lipid levels
Electric activity of skeletal muscles
Muscle strength
Cross-sectional area of muscles
Pelvic incidence
Pelvic tilt and sacral slope

**Table 5**  
Radiographic signs of lumbar segment instability.

Abnormal angulation/translation in flexion/extension
Abnormal angulation/translation lateral bending
Vacuum phenomenon in facet joints or disc with or w/o movement
Traction spurs
Black disc
Modic signs
Pathological discography
Facet joint degeneration
Misalignment in sagittal, frontal and transverse plane
Central stenosis
Degenerative olisthesis
De-novo scoliosis

Structural deterioration of the lumbar disc – influenced by genetic [26–28] and physiological factors – leads to lumbar misalignment in coronal and sagittal balance and consequently, to the loss of lumbar lordosis [29–31] (Table 4). This initiates a degenerative cascade of LSI also leading to LSS [32,33]. Over time radiographic signs become visible such as traction spurs, facet joint degeneration, misalignment, stenosis, olisthesis and de novo scoliosis [34–36] (Table 5).

Diagnostic and management decisions in LSI are often made on the grounds of expert opinions focusing on symptoms and imaging. However, as clinical and radiographic information poorly correlates, there is an apparent missed opportunity between the daily clinical practice of diagnosing patients with back disorders and



**Fig. 3.** Model of compensation/decompensation.

the scientific evidence available from systematically gathering and interpreting data from groups of similar patients. Furthermore, a thorough holistic approach when assessing patients with LSI including physiological and psychological factors is rarely taken into consideration.

There is a need for an extended pathophysiological/psychosocial concept of compensation/decompensation in LSI (Fig. 3) [37–40]. Functional compensation and decompensation are subject to numerous factors that can vary between individuals and change over time which results in a poor correlation between radiographic degenerative signs and clinical symptoms. This makes estimation of an individual's prognosis of the benefit of surgical or non-surgical treatment difficult [41]. In the following a broader conceptual framework of compensation/decompensation and examples will be presented that may help solve the debate on the prognostic value of functional and psychosocial factors of influence.

Compensation and decompensation may influence some non-structural radiographic signs of degeneration, e.g. the degree of misalignment and segmental angulation caused by LSI is influenced by the tonus of the local musculature [42,43]. Factors which might influence whether a patient with LSI, LSS or lumbar misalignment will be symptomatic, i.e. be in the state of decompensation, are different electric activities of skeletal muscles, muscle strength [44–47], cross-sectional area of muscles and pelvic parameters [30,31,48].

Conflicting results are presented in the literature regarding the influence of muscle activity on patients with LSI, LSS or lumbar misalignment. This could be due to the fact that different individual spine curves require different individual stabilizing musculature [49–54].

This conceptual model of compensation/decompensation can be extended to include LSS as one example of its application beyond LSI. Consider an 88-years-old patient with symptoms for 8 weeks and dysfunction with claudication and walking distance of 40 m due to mechanical strain and natural degeneration, with images illustrating degenerative stenosis and instability due to segmental kyphosis, leading to the condition of spontaneous recompensation and resolution of clinical symptoms without functional therapy (Fig. 3). Patients with early compensated LSS usually present with LBP as an initial symptom. At a later stage, leg fatigue, pain, numbness and weakness of the lower extremities commence. These symptoms begin or worsen with the onset of walking or by standing upright and are promptly relieved when leaning forward, sitting or lying down.

As another example, consider a female patient aged 69 with symptoms for 6 months and dysfunction with claudication and no pain free walking distance due to mechanical and psychosocial strain, with images demonstrating degenerative stenosis and instability due to a facet cyst and secondary lumbar kyphosis, resulting in irreversible decompensation and no resolution of clinical symptoms despite functional therapy. Once symptoms of neurogenic intermittent claudication that occurs during walking and forces patients to rest are present the condition of decompensated state has usually been reached. In radiological terms decompensation could be expressed by a positive Nerve Root Sedimentation Sign where nerve roots are tethered at the level of the stenosis [55,56]. It is assumed that similar to the degenerative cascade in LSI there is a 'narrowing' cascade in LSS whose determining factors are still poorly understood, as illustrated by common over and underdiagnosis of LSS [57,58].

## Discussion

In this paper we propose that the diagnostic research concept of defining clinically meaningful target conditions can be extended to

**Table 6**

Current multidisciplinary interventions.

Multidisciplinary biopsychosocial rehabilitation
Rehabilitation programs
Functional restoration programs
Back schools
Work hardening
Conditioning exercises

address the challenging problem of selecting treatments for patients with lumbar spinal degeneration. We hypothesize that additional clinical and radiological criteria can be developed to define a target condition of decompensated spinal degenerative disease to identify patients who have reached the point where there is only a very low probability of returning to a compensated state. These criteria will be enormously valuable to identify patients at high risk of 'irreversible' decompensated LSI/LSS for whom surgery becomes the treatment of choice.

To test this hypothesis, we suggest further research is needed to identify the criteria that can be used to define the decompensated target condition. Future studies need to identify predictors that can help discriminate between patients at low and high probability of remaining in an irreversibly decompensated state. This will require prognostic studies, i.e. data collected from clinical studies of patients managed with conservative care, whereby the potential radiological, physiological and psychological predictors are compared with the clinical outcome (symptom improvement). The two types of studies that can provide data to identify the optimal clinical and test criteria or cut-points to define the presence of the target condition for treatment decisions are: (i) cohort studies that can be used to examine the relationship between clinical factors, test results and subsequent clinical outcomes (prognosis) to develop risk groups that can inform decisions about whether treatment is indicated; and (ii) treatment trials that can be used to identify patient subgroups (defined by clinical factors and test results) where the benefits of treatment outweigh the harms.

Finally, this conceptual model of compensation/decompensation may be extended to spinal disorders in general and beyond. It could help solve the debate on the proportion of influence of functional and psychosocial factors on LBP and to establish a new definition of non-specific LBP. Individual differences could be explained by compensated or decompensated LSI leading to changes in clinical symptoms and pain. As long as identically structural characteristics, such as translation in ante and retroflexion for LSI or the area of the dural sac in MRI for LSS, are employed it is not surprising that these characteristics are of only limited predictive value for the presence of clinical symptoms.

## Conclusions

This model of compensation/decompensation aims to solve contradictions in existing concepts in the spine field by explaining why identical structures may lead to varying symptoms. Future spine surgery will have to carefully define and measure functional aspects of LSI. This will have to include the identification of specific signs for a point of no return where multidisciplinary interventions do not allow a re-compensation and surgery becomes the treatment of choice [59] (Table 6). Knowledge of signs for a point of no return, i.e. a status of decompensation is crucial for decision-making in today's health care systems that are scarce of resources.

## Conflict of interest statement

None to declare.

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