Adopted: 8/10

Lumbar Functional Instability

(*AKA*, Lumbar: Clinical Instability, Segmental Instability, Joint Instability)

Note: In the 4-part diagnosis format used for musculoskeletal cases in the UWS clinic system, functional instability is not usually listed as a primary diagnosis. More commonly it will be accounted for as a significant contributor to a primary diagnosis linked by the phrase "complicated by lumbar functional instability." Its presence is meant to identify one type of case that may be particularly suited for stabilization exercises. Severe traumatic instability and pathological instability are not included here.

Definition

Functional instability of the lumbar spine has been proposed as a distinct subset of patients with LBP (Dellitto 1995, Demoulin 2007). Lumbar functional instability (LFI)* is a clinical diagnosis based on history and physical examination findings. It is a painful disorder hypothesized to result from a loss of the spine's ability to maintain appropriate mechanical stiffness** in neutral, midrange, or end-range movements. The most widely cited definition is a "significant decrease in the capacity of the stabilizing system of the spine to maintain the intervertebral neutral zones within the physiological limits so that there is no neurological dysfunction, no major deformity and no incapacitating pain." (Panjabi 1992)

Functional instability is not synonymous with *hypermobility* or *radiographic instability/hypermobility*. Hypermobility and radiographic instability may be asymptomatic. These terms denote circumstances where joint motion is excessive but may not be associated with qualitative (aberrant) alterations in physiologic motion. (Demoulin 2007)

* LFI is used throughout this document for functional lumbar instability, but it is not a generally recognized abbreviation and is not suitable for charting.

** In biomechanical terms, spinal stiffness refers to the spine's ability to prevent unwanted movement or buckling.

Discussion

The stabilization system of the spine can be divided into three subsystems: passive, active and neural (Demoulin 2007, Panjabi 2003). The passive system consists of the discs, bony and ligamentous structures and is primarily responsible for resistance at the end range of motion. The active system is composed of muscles and their tendinous attachments. This component plays a major role in maintaining neutral zone stability. Multiple muscles contribute to spinal stability. (Kavcic 2004, McGill 2003) The deep segmental muscles (especially multifidi and transverse abdominis) have been proposed as central players by some researchers (Hebert 2010) although whether they play a unique role is challenged by others. (Kavcic 2004, McGill 2002) The neural or motor control subsystem consists of the central and peripheral nervous system and integrates proprioceptive input from mechanoreceptors located in soft tissue structures and coordinates activation of stabilizer muscles. LFI is postulated to result from dysfunction of this stabilization system.

Neutral Zone Instability/Motor Control Insufficiency (MCI)

The neutral zone is defined as the component of physiologic intervertebral motion that can be induced with minimal internal resistance (Panjabi 2003). It is measured from the joint's starting position up to the elastic zone (Panjabi 1992). The elastic zone represents the elastic

capacity of the joint's soft tissue structures and the small amount of additional motion that is available toward the end range of joint motion. Movement into the elastic zone encounters more resistance and is dependent on forced muscular effort on the part of the patient or by additional overpressure by an examiner. When the forces applied at this point are removed, the joint springs back from its elastic limits.

Neutral zone instability is postulated to result from expansion of the neutral zone or deficiencies in motor control with or without measurable excessive end range motion. Expansion of the neutral zone is theorized to result from degeneration or attenuation in the passive stabilizers (ligaments and the disc).

Motor control insufficiency (MCI) refers to a break down in the neuromuscular system that provides the requisite stiffness needed by a functioning spine. MCI can result from poorly conditioned muscles or from errors in the neurological control and programming of the muscles. It can be seen as a cause of functional instability. This disorder is usually not associated with marked structural deformity, excessive end range radiographic hypermobility or neurologic deficits. (Panjabi 1992) Therefore dynamic radiographs will typically not reveal any significant abnormalities of quantitative movement.

MCI and neutral zone instability are associated with poor coordination of movement, lack of proper stabilization and episodes of momentary aberrant motion. These events can cause abnormal tissue loading (e.g., spinal "buckling") and local injury. Immediate triggers of painful episodes include sustained postures, repetitive low load activities, or *a single repetition of an ordinary activity of daily living*. Based on biomechanical research, The National Institute for Occupational Safety and Health (NIOSH) suggests that the spine can safely accommodate compressive loads at least up to 3,400 Newtons during lifting tasks. (Waters 1993) However, in vitro experiments have demonstrated that, *in the absence of muscles and motor control systems*, the passive elements of the spine alone cannot withstand loads of more than 90 Newtons (approximately 9.2 kg) (Crisco 1991), making even common activities potentially hazardous for the functionally unstable spine.

Excessive end range motion/ hypermobility

Excessive spinal joint end-range motion has historically been labeled as either hypermobility or instability. However, excessive end range motion is not necessarily associated with instability and *the terms should not be used interchangeably*. For the purposes of this document joint hypermobility and radiographic instability will be considered as isolated exam findings. Segmental or regional joint hypermobility is not clinically problematic unless it is associated with pain or perceived as a risk factor for injury. Joint hypermobility may be body wide or affect specific spinal regions or joints. Joint hypermobility does not cross into the realm of instability unless it is inadequately compensated for by the motor system resulting in the characteristic signs and symptoms of functional instability.

Excessive end range motion results primarily from a loss of integrity of segmental spinal ligaments, intervertebral disc or bony stabilizing structures. This excessive end range motion may result from high load traumatic events, repetitive microtrauma, a developmental byproduct of activities such as dance or sports (e.g., gymnastics) or, most commonly, from significant spinal degeneration (degenerative spondylisthesis). Spinal degeneration is thought to have a strong genetic factor (Battié 2009).

Although there may be clues from a patient's history and physical examination (i.e., palpation) suggesting hypermobility, it can only be definitively demonstrated on dynamic radiographs by excessive listhesis at end range. A variety of radiographic measures and ranges has been proposed (see diagnostic imaging). There is, however, little agreement on a clinically useful cut point dividing excessive from normal range of motion (Demoulin 2007). In the absence of established standards, increased end-range segmental motion must be associated with clinical findings to be considered significant. Dynamic radiographic evaluation is neither routine nor recommended as a screening tool for the identification of possible joint hypermobility.

Excessive end range motion may be also be associated with generalized joint laxity. In such cases it is associated with an increase in gross ROM throughout the body and can be identified by an instrument like the Beighton Ligamentous Laxity Scale. (See Appendix I.)

Finally, end range joint motion may be clearly excessive and if there is significant disability and/or significant neurologic deficits, orthopedic consultation and potential surgical fusion may be appropriate.

Diagnosis

The diagnosis of functional instability is made solely on clinical grounds. At this time there is no clearly established consensus on the criteria for diagnosing LFI (Cook 2006, Demoulin 2007). There are, however, certain patterns of presentation that suggest that the patient's spine may have a higher probability of being functionally unstable or would benefit from a spinal stabilization exercise program (Hicks 2005).

Preliminary research has identified 4 prognostic factors and a clinical prediction rule which identifies LBP patients who have better outcomes with stabilization exercises. These factors are

- age under 40
- SLR mobility above 91 degrees (average of both legs)
- aberrant movement with lumbar flexion (i.e., painful arc, instability catch, or reverse lumbopelvic rhythm)
- a positive prone instability test. (Hebert 2010, Hicks 2005)

The presence of these prognostic indicators has also been positively correlated with ultrasound evidence of poorer multifidus activity compared to controls (but not related to transverse abdominal muscles). (Hebert 2010)

Although improvement with stabilization exercise does not confirm the presence of LFI, lumbar stabilization exercises would be the therapy of choice, reinforcing the value of utilizing the clinical prediction rule. Patients fulfilling these criteria were also more likely to be associated with aberrant middle range motion on fluoroscopic video in one study (Teyhen 2007).

Most of the other clinical signs and symptoms frequently cited are based on expert opinion and extrapolation from biomechanical research. In 2006, using a Delphi methodology, a group of experts identified 15 subjective and 14 objective identifiers for instability reflecting current practice profiles (Cook 2006). The clinical clues most cited appear to derive from 1) the onset and behavior of the symptoms, 2) assessment of the quality of regional and segmental motion, and 3) indicators of poor motor control.

Coding note

Functional instability does not have its own ICD code and there does not appear to be a commonly used code used in the profession. However, including functional instability in a diagnosis and coding for it may help communicate that the patient has a more complex mechanical injury. Several codes can be considered. The 2009 ChiroCode Desk Book recommends 724.6, which the ICD code book lists as ankylosis or instability of the sacroiliac or lumbosacral joint. There is a 718.80 code that is variously defined as acquired joint hypermobility and elsewhere as instability and again as post-traumatic instability. There is a 728.5 code for hypermobility syndrome, which might be used, but would appear to be more compatible with generalized joint hypermobility (see Appendix 1). Because the problem of functional instability is thought to relate to poor muscle control and because exercises suggested to promote greater spinal stability often target the muscles, the 729.1 myofascial code has also been used.

History

The following is an attempt to organize a variety of observations from the literature into suitable profiles for pattern recognition. (Cook 2006, Delitto 1995, Demoulin 2007, Grieve 1982, Kirkaldy-Willis 1982, MacDonald 2009, Maigne 2003, Meadows 1999)

Summary of clues from history

- Episodic nature (triggered by trivial events)
- Reports of catching, locking, giving way
- Immediate pain with sitting
- Temporary response to manipulation
- Decreased response to manipulation over time

1. Episodic Nature

- Multiple unpredictable episodes: onset triggered by sudden or trivial movements or sustained postures is one of the most frequently cited patterns suggestive of instability (Cook 2006, Demoulin 2007).
- Pain free intervals: Between episodes the patient may be relatively asymptomatic with full range of motion.
- Progressive course: Episodes appear more often or become more persistent.

Note: Chronic LBP, whether progressive or not, may also be associated with instability, demonstrating similar physical examination characteristics.

Rationale/level of evidence: Recurrent episodes are thought to be the result of sudden spinal buckling sustained under normal loads. The level of evidence is based on expert opinion. In Cook's 2006 Delphi review, recurrent episodes ranked among the 3 most cited characteristics from the history.

2. Subjective sense of instability

- Reports of catching or locking associated feeling of giving way which may be followed by a minor aching for a few days. The patient may report consistent clicking or clunking noises.*

Patients classified by O'Sullivan et al (2000) as having instability used similar descriptors: describing pain as catching (45%), a "feeling of instability" (35%), locking (20%), or giving way (20%).

Rationale/level of evidence: Painful catches are thought to be associated with at least temporary loss of motor control and buckling at a particular segment(s) during a particular range of motion or during a specific activity. The level of evidence is based on expert opinion. Cook's 2006 Delphi survey it ranked among the 3 most cited characteristics from the history of patients suspected of having LFI.

- Immediate pain with sitting relieved with standing:
  A rare presentation is pain initiated immediately with sitting down and relieved with standing. This pattern has been associated with radiographic signs of hypermobility in 31% of the cases (i.e., > 4.5 translation on flexion-extension films) or with loss of anterior disc space in 55% of the cases. (Maigne 2003)

3. Temporary response to treatment decreased over time

- Medical Treatment: traditional medical or physical therapy treatments have failed or have afforded only temporary relief.
- Manual therapy: temporary, symptomatic improvement.
- Relief with bracing: temporary relief with a back brace or corset.

* Another possible cause of a patient describing a click, a shift, a clunk or their back going out is the **snapping hip syndrome**. This syndrome is sometimes organized by causes that are external (outside the pelvis, e.g. the iliotibial band snapping over the greater trochanter), internal (the iliopsoas tendon snapping over the iliopectineal eminence) or intrarticular. The common feature is pain and audible snapping with hip movement. The internal snapping hip may be reported as snapping or popping or shifting in the lower back, presumably because the snapping sensation is transmitted to the spine at the attachment of the psoas.

Snapping hip syndrome has been reported in ballet dancers. (Blankenbaker 2008) A 2007 cross sectional study of 87 elite dancers found ninety-one percent of dancers reported snapping hip. Blinded clinical examiners were able to identify the site of snapping with palpation in 46 of 50 snapping hips. Ultrasound testing was used to demonstrate a snapping iliopsoas tendon in 59% of cases. 4% of the snapping hips had iliotibial band snapping that was demonstrated with ultrasound. The epidemiology of snapping hip syndrome is not very well defined. Most of the cases reported are in young, athletic people although elderly people with osteoarthritis of the hip may also be at risk. (Winston 2007)

Physical Exam

The physical exam findings can be generally grouped as follows: 1) altered quality of active motion, 2) specific segmental findings, and 3) evidence of poor motor control.

Summary of clues from the physical exam

1. Altered quality of movement
   - Painful arc
   - Aberrant motion (i.e., Minor's sign, instability catch, reversed lumbosacral rhythm)
2. Specific segmental findings
   - Positive prone instability test
   - Decreased resistance with prone joint play
   - Increased motion with motion palpation
3. Evidence of poor motor control
   - Segmental abnormal movement (i.e., segmental hinging during AROM)
   - Painful arc abolished with abdominal bracing
   - Poor motor control during trunk forward lean
   - Poor motor control of pelvic clocking and abdominal hollowing
   - Poor motion control during hip extension test
   - Poor motor control during the single leg stand

1. Altered quality of active motion

The patient is observed going through normal active ranges of motion, looking for signs of altered *quality* of movement. The abnormalities are most frequently observed in the sagittal plane, but may also be detected in lateral flexion. *Observation is best done with the back exposed during range of motion testing.*

- Painful arc**:** During flexion and extension a patient may display a range of movement which reproduces his/her complaint and may be accompanied by a painful catch. This arc of motion is not at the end range and may be experienced on return from flexion.

Rationale/level of evidence: The ability to detect a painful arc in the sagittal plane has been reported as having good inter-examiner reliability (*k* .61 to .69) (Hicks 2003). This finding has also been incorporated into a clinical prediction rule for stabilization exercises which has shown some promise (Hicks 2005).

- Aberrant motion**:** One group of findings has been loosely referred to as *aberrant* motion and consists of one or all of the following: Minor's (Gower's) sign, an instability catch, and reversal of lumbopelvic rhythm. (Hicks 2003)
  - Minor's/Gower's sign: The patient supports hands on lower extremity, "walking up one's thighs," when returning from a flexed position.
  - "Instability catch" (Demoulin 2007, Hicks 2003): A sudden acceleration or deceleration of motion or abnormal quality of motion (e.g., movement into planes outside the primary plane of motion such as rotation or lateral bending while the patient is attempting to move through the sagittal plane).
  - Reversal of lumbopelvic rhythm (Delitto 1995, Demoulin 2007, Hicks 2003): When returning from forward flexion, the patient bends the knees and initiates the motion in the lumbar spine instead of at the pelvis, extending the lumbars first and then extending at the hips.

Rationale/level of evidence: Various signs of poor movement quality are thought to signal individual segments painfully buckling or displaying aberrant motion perhaps secondary to poor motor control. As individual signs, Minor's, instability catch, and reversal of lumbopelvic rhythm have demonstrated poor to only fair inter-examiner reliability. As a group, however, they have a reported k value of .60 (moderate to good) (Hicks 2003). This finding has also been incorporated into a clinical prediction rule, which has shown some promise for identifying patients who benefit from a stabilization rehabilitation program. (Hicks 2005)

2. Specific segmental findings

Prone instability test: The patient is placed in a prone position with the lower extremity off the end of the table and feet supported on the floor. During the first part of the test, the practitioner applies springing P-A pressure with his/her hypothenar to the lumbar spinous processes testing for pain. If pain is elicited the patient is instructed to raise his/her feet off the floor and pressure is reapplied to the spine to assess whether the pain has been abolished. Abolished pain is a positive test, suggesting

functional instability and a potentially good response to a program of stabilization exercises. (Demoulin 2007, Gill 2002)

Rationale/level of evidence: This test is based on the premise that muscle activity associated with raising the legs stabilizes a clinically unstable joint or region against a shear load, eliminating the provoked pain (Demoulin 2007). It has demonstrated moderate to good inter-examiner reliability. Hicks (2003) reported a *k* value of .87 with a narrow confidence interval. Schneider (2008) reported *k* .54 for step 1 and .46 for step 2. When combined with a presentation of patients under 40, bilateral SLR flexibility over 91 degrees and aberrant lumbar flexion, this procedure successfully predicted patients more likely to respond to stabilization exercises. (Fritz 2005, Hebert 2010, Hicks 2005)

- Prone joint play (JP)/ passive accessory intervertebral motion tests (PAIVMS): The patient is placed in a prone neutral position. The doctor applies springing P-A pressure with his/her hypothenar to the lumbar spinous processes evaluating for pain and mobility (Peterson 2003). Functional instability is suspected if normal resistance and recoil is absent or if excessive P-A glide is encountered. Crepitus or "clunking" may also be noted.

Rationale/level of evidence: Good intra-examiner and poor interexaminer reliability have been reported for mobility assessment (Demoulin 2007, Schneider 2008). Schneider found the interexaminer reliability for restricted motion to be poor (*k* range from -.20 to .17). However, in an attempt to correlate physical exam findings with radiographic hypermobility, Fritz et al found the *lack* of hypomobility (i.e., either a "normal" feel or hypermobility) to have a positive LR of 4.3 (with a relatively wide 95% CI range of 1.8-10.6) and a negative LR of 0.22 (95% CI 0.10-0.50). Hicks (2005) found palpatory hypermobility to be part of a successful prediction rule identifying patients best suited for a rehabilitation exercise program. In this study the NNT to prevent one misassignment into an unsuccessful treatment regime (i.e., physical rehabilitation plus manipulation vs. manipulation only) was 1.6 (95% CI 1.2-10.2).

- End Play (EP): excessive end range motion or elasticity is suspected by a lack of resistance at the end range of motion detected during seated or lying motion palpation. This has been described as an empty end feel or "boggy" end feel. Crepitus or "clunking" may also be noted.

3. Evidence of Poor Motor Control

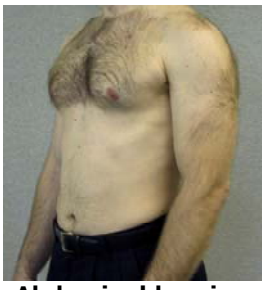
A variety of exam findings have been reported that suggest poor motor control, which in turn, may further suggest the presence of functional instability. It is important to note that what initially appears to be poor motor control can be simply the result of guarding or splinting in response to acute pain.

- **Segmental abnormal movement.** Segmental hinging or pivoting with active movement observed during active ROM. (Cook 2006) Other signs of poor segmental control include "wiggling" or non-smooth spinal motions in any plane. Spinal "hinging" or sharp angulation of the spine may be present during lateral flexion.

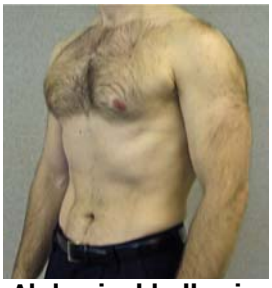
- **Painful arc abolished.** Improvement or abolishment of painful arcs and movements if performed while maintaining an

abdominal brace (Cook 2006, Demoulin 2007) or deep abdominal activation (i.e. abdominal hollowing). (O'Sullivan 2000)

- **Trunk forward lean.** This procedure attempts to detect poor lumbopelvic coordination during forward flexion or while getting out of a chair. The seated patient is instructed to lean forward from the hips while their ability to maintain spinal curves is monitored. Inability to bend forward more than 15 degrees without either flexing the lumbar spine or overcompensating with hyperlordosis is a positive finding This has been reported in low back patients (Hamilton 1995) and is speculated to reflect poor motor control issue, suggesting functional instability, or at least indicating the need to establish core stability.
- **Pelvic clocking and abdominal hollowing.** Difficulty in learning how to control pelvic motion (e.g., inability to find and hold a neutral pelvis) or inability to perform controlled abdominal hollowing (which may be measured with a spinal stabilizer or directly observed). See CSPE protocol, *Low Back Rehabilitation*.



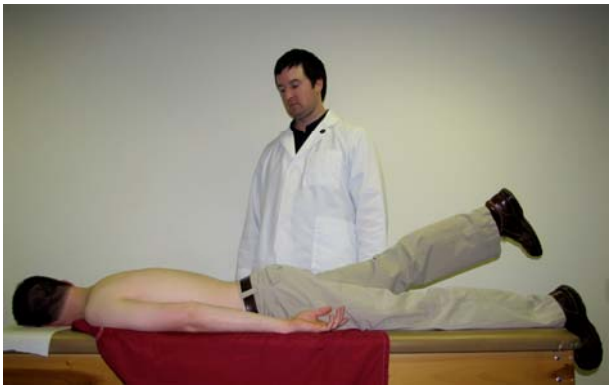
Abdominal bracing



Abdominal hollowing

Pelvic control monitored with spinal stabilizer

- **Hip Extension test.** Difficulty in maintaining proper form or mechanics while performing prone hip extension (Murphy 2006). The patient, lying prone, is instructed to lift their lower extremity off of the table. Lateral shift toward the side of hip extension, excessive lordosis, or rotation of the spine (spinous processes rotating toward the side of the hip extension) denotes test failure. It has been proposed that this failure may reflect poor motor control and functional instability. It has also been proposed as part of a decision making rule in identifying patients suitable for stabilization exercises. (Murphy 2009)



Hip extension test (movement pattern)

- **Single leg stand.** Poor motor control during a 20 second single leg stand or sitting on an exercise ball (Tidstrand 2009). These procedures are performed with the patient's eyes open and the observer's eye positioned at the level of the pelvis and directly behind. Poor motor control is identified by any of the following:
    - spine deviates from the vertical position
    - shift in pelvic crest height
    - compensatory movements of the arms or opposite leg
    - two or more brief corrective movements from the starting position
    - one prolonged corrective movement.

  Other causes of poor balance (e.g., cerebellar, posterior column and vestibular lesions) also need to be ruled out.

  Rationale/level of evidence: Indicators of poor segmental control are consistent with the Punjabi model of breakdown in the motor or motor control systems. A trend of

biomechanical experimental evidence based on differentiating low back pain patients from healthy controls supports this theory. Motor control deficits identified include poorer postural control associated with increased body sway, decreased ability to reproduce isometric contractions of the trunk, delayed motor response, poor reaction time to sudden loads, and failure of the flexion-relaxation phenomenon to occur at end range flexion. But whether these findings are more likely to be found in a subset of patients designated as having functional instability is unknown. Simpler methods for assessing motor control and endurance in a routine clinical setting are cited above but none of them have been directly validated. Their current use is based on expert opinion, biomechanical plausibility, and pragmatic inclusion in a number of outcome studies looking at stabilization exercises. Poor lumbopelvic control was the most cited criterion in Cook's Delphi survey. Interexaminer reliability has been reported to be good to very good for the hip extension test (*k* 0.72-.76) (Murphy 2006), the single leg stand (*k* 0.88-1.0) (Tidstrand 2009), and sitting on an exercise ball (*k* 0.88) (Tidstrand 2009).

Other potential physical findings:

- Similar to postural syndromes, patients may report that sustained sitting, standing or sustained flexion are aggravating (O'Sullivan 2000). The patient may report or demonstrate difficulty with unsupported sitting and may exhibit postural "restlessness."
- Functional instability may be associated with spondylolisthesis in some cases. In these cases the patient may have a spinal "ledging" (step deformity) at the level of the spondylolisthesis and may be sensitive to posterior to anterior pressure on the spinous. Functional radiographs may demonstrate excessive movement.
- Instability is not usually associated with referred or radicular leg pain (Sullivan 2000), but leg signs and symptoms can accompany the condition is some cases. In cases where it is radicular, tension tests may be positive and there may be at least mild neurological deficits (Boden 1997). In one cohort of 111 patients with symptomatic spondylolisthesis severe enough to warrant surgery, 62% had sciatica (Moller 2000). The presentation may rarely include a positive SLR (sensitivity of 12% compared to 80-100% in disc herniations). (Moller 2000) Likewise, nerve root deficits are not common (12% in one study). The L5 nerve root is the most commonly involved, followed by the L4 nerve root in more severe cases. (Moller 2000)

Diagnostic Imaging

The role of diagnostic imaging in the evaluation of patients with suspected lumbar instability is the topic of ongoing debates and numerous studies and is ultimately difficult to define. Common practices among physicians and radiologists are not well supported by evidence. The terminology used can be confusing and misleading. When excess motion is identified by imaging, it may be referred to as radiographic instability or radiographic hypermobility. Neither of these terms is clearly defined nor are the parameters for normal motion widely accepted. These findings are often discussed as evidence of "structural instability" to separate them from the clinical concept of functional instability. The following discussion attempts to evaluate and apply the available evidence regarding radiographic hypermobility and its correlation with the clinical entity of instability and the role of imaging in the evaluation of patients with suspected instability.

Imaging modalities have been used in the evaluation of patients with suspected lumbar instability. In certain cases, radiography, computed tomography (with or without myelographic contrast), MRI and stress studies (with radiographs or MRI) may provide patient management information. Imaging may be incorporated in patient evaluation to rule out other causes of symptoms, to determine the degree and nature of any associated neurologic compromise, and to support the diagnosis of instability. Still, instability remains a clinical diagnosis and imaging findings cannot be used to confidently rule this diagnosis in or out and do not provide significant information to direct the course of patient care. Normal segmental spinal movement is not a well defined entity and there is no consensus about the definition

of abnormal movement or how to measure it (Abbot 2006, Mulholland 2008). Clinical and radiographic findings of instability are often discordant. (Boden 1996,1990, Leone 2007) Some authors have suggested the association of findings such as traction osteophytes (small, horizontal osteophytes arising a few millimeters from the endplate margin) and vacuum phenomena (gas density in the disc space) with instability. These are common findings with more advanced degenerative disease and may reflect excess motion, but they have not been clearly associated with current clinical symptoms of instability. (Leone 2007) Imaging is most appropriate when results may affect the treatment plan. Imaging studies are not generally indicated for low back pain and should only be used to confirm or rule out a diagnosis which would affect patient management. Basic imaging guidelines for low back pain should be applied.

Basic imaging guidelines for low back pain

Imaging is not indicated in the first six weeks of low back pain if the following criteria are met:

- No neurologic symptoms
- No constitutional symptoms
- No history of trauma
- No symptoms of malignancy
- Patient is 18 to 50 years old

Note that the presence of any of the above criteria, except for symptoms of malignancy, does not mandate imaging, but rather raises it as a clinical consideration.

If clinically significant improvement is not seen in six weeks and potential diagnoses or complicating factors could be identified with imaging evaluation should begin with AP and lateral lumbar films. (Boden 1996, Kalichman 2008, Pitkanen 1997, 2002, ACR 2008). Radiographs may be acquired sooner or may be delayed for eight to twelve weeks based on the level of clinical suspicion and clinical indicators of significant pathology.

Specific recommendations for suspected instability

In general, imaging is not necessary in cases of patients who present with clinical signs and symptoms of instability where there is no reason to suspect major trauma, neurological compromise, or an underlying disease process. When imaging is ordered, plain film radiographs are typically the first imaging used. This modality is usually adequate for identifying the changes of degenerative disease of the intervertebral disc or facets. Most radiographic hypermobility appears to be secondary to degenerative changes. (Prathria 2005, Kalichman 2008) It may also identify pars defects found in spondylolytic spondylolisthesis which is a condition less frequently responsible for radiographic hypermobility. Studies should include a minimum of an AP and a lateral view of the lumbar spine. Oblique radiographs or an axial lumbosacral view may be required to visualize the pars interarticularis and the facets.

Stress films

Stress films can be ordered to document radiographic hypermobility. The significance of this finding is debated and even when present doesn't usually alter conservative care. Even the finding of listhesis in a neutral lateral radiograph does not necessarily indicate the need for stress radiographs. Stress radiographs are usually lateral views with flexion and extension or with traction and compression. The comparison of upright to recumbent views may also provide this information. Each of these methods may produce both false negative findings and many positive findings which cannot be correlated with symptoms or clinical findings. In one study, 42% of asymptomatic patients met the radiologic criteria for instability at one lumbar level. (Biden1990)

Flexion-extension films have been determined to be better than traction-compression for identifying excessive motion though at least one study has shown advantages for traction-compression. (Kalebo 1989, Pitkanen 1997, 2002) The value of upright versus recumbent flexion-extension films has not been determined though muscle guarding may be lessened in a recumbent or seated position. (Boden 1990, 1996, Maigne 2003) The best choice of technique for stress studies is often patient and facility dependent. Flexion-extension is typically easier to perform than traction-compression. Patient and technician abilities may affect the choice of upright versus

recumbent films. Strong evidence is not available to support a given technique.
The currently accepted standard for evidence of radiographic hypermobility is greater than 3mm translational or greater than 10 degrees angular (sagittal rotation) motion. These findings have been associated with greater limitations in activities of daily living (ADLs) due to pain. It has been suggested that translational motion (>3mm)* is associated with symptoms more frequently than changes in angular motion. Clinical findings compared included low back pain, leg pain and/or numbness, walking ability, straight leg raise, sensory function and motor function. (Iguchi2004) An association has been noted between anterior translational instability and degenerative disc disease. (Fujiwara 2000) Some studies consider larger measurements (up to 5mm translation and 20 degrees sagittal rotation) for the threshold of instability and some provide thresholds by level. (Dupuis 1985, Dvorak 1991, Hayes 1989, Knutsson 1944, Nachemson 1985, Shaffer 1990, White 1990) Marked loss of anterior disc space on flexion (greater than 5 degrees of anterior wedging) was correlated with the clinical finding of pain made worse by sitting. (Maigne 2003) Aberrant associated motion or malposition including lateral flexion and rotation may make landmarks for measurement difficult to identify. The abnormal motion that may occur in functional instability may not be visible on radiographs. For diagrams demonstrating these various measurements, see Appendix II.

Videofluoroscopy

Fluoroscopy has the seeming advantage of showing segments throughout the range of motion compared to plain film which depicts end-range motion. (Teyhen 2005) Demoulin et al note that functional instability may be primarily an issue of problems in the neutral zone. (Demoulin 2007) Still, fluoroscopy has the potential for the same errors as plain film stress studies and no definitive relationship has been identified between segmental motion and clinical symptoms. (Teyhen 2005, Ahmadi 2009)

Advanced imaging

In cases where there are additional signs of radicular involvement or intermittent claudication, MRI or CT may be indicated.

MRI

MRI is the imaging of choice in patients with significant neurologic symptoms. The impact on the cauda equina and nerve roots is well delineated. MRI may provide the best detail of degenerative changes and pars interarticularis abnormalities as well. Increased collection of fluid in the facet capsule has an 82% positive predictive value for L4-5 radiographic instability. (Rihn 2007) Modic changes in the marrow adjacent to the endplate are associated with degenerative disc disease. Type 1 changes (increased signal intensity on T2 weighted images, decreased signal intensity on T1 weighted images) have been associated with symptomatic degenerative disease, but an association with instability has not been established. (Rahme 2008) Fatty degenerative changes may be seen in paraspinal musculature which may reflect atrophy from inhibition.

Stress studies may be performed with MRI using upright scanners or axial compression devices. These studies can identify the abnormal motion as well as its impact on neural tissues. (Perez 2007, Jinkins 2003, Kong 2009) Changes may be identified in disc height, sagittal translation, disc angle, lordosis and dural cross-sectional area. A 2009 study of 59 patients with chronic back pain and degenerative spondylolisthesis demonstrated a correlation between changes in disc angle and Oswestry Disability Index and physical function scores. Additional findings of disc herniation, synovial cysts, facet subluxation and lateral recess narrowing may add to the clinical picture. (Huang 2009) Significant decreases in dural cross-sectional diameter after axial loading has been identified more frequently in symptomatic than asymptomatic patients. (Danielson 2001) Additional information from axial loading has been identified primarily in patients with neurologic symptoms, particularly neurogenic claudication. (Willen 2001)

Computed tomography

Computed tomography (CT) may best delineate the osseous changes associated with degenerative disease and radiographic hypermobility. Radiographic hypermobility may result in dynamic stenosis. Neural canal stenosis can be quantified and the causative factors of the stenosis defined. (Kalichman 2008) Myelographic contrast may be used to

better evaluate the effect on neurologic structures. CT may also identify other osseous abnormalities such as radiographically occult pars defects. CT is usually the best alternative for a patient unable to undergo an MRI scan.

Discography

Discography is not supported as a useful imaging modality in evaluating instability. (McCormick 1997)

Instability Table

(LeFebvre, 7/26/10)

Clinical clue for instability	Compared to other types of mech LBP (e.g., sprains, disc derangement)	Speculated reason	Based on…
A pattern of multiple episodes set off by minor triggers*	40-60% of typical LBP patients experience recurrence or low level chronic symptoms—but a pattern of sudden flare ups caused by minimal loading events may linked in the instability phenomena.	Poor stability increases the risk of a "spinal buckling" event that triggers episodes; or accumulative buckling prevents the tissue from ever healing.	Expert opinion, practice consensus, biomechanical plausibility
Response to joint CMT may only be temporary or poor	CMT more likely to be useful overall.	CMT does not address underlying cause, but may temporarily close the pain gate early on.	Expert opinion.
Report of painful catch (by patient report or witnessed during the PE)	Less likely although this has been suggested as linking with a meniscoid entrapment.	Painful catch is associated with unstable moment while the joint moves through the neutral zone.	Expert opinion, practice consensus
Active ROM demonstrates painful arc	Pain more likely to increase the further into active end range motion as load increases on painful tissue (e.g., sprains, disc derangement, postural syndromes)	Poor motor control or excessive motion at a segment alters the quality of movement during certain gross motions—but end range loading may be well tolerated, except perhaps at its terminus or if sustained.	One unvalidated study of a clinical decision making rule; expert opinion; practice consensus
Poor quality of movement (e.g., Minor's sign, instability catch, altered lumbopelvic rhythm)	May also be found less frequently in typical low back cases especially when acute (e.g., Minor's sign)	Poor motor control.	One unvalidated study of a clinical decision making rule; expert opinion; practice consensus
Associated with segmental hypermobility or at least with "normal" resistance	Less likely.	Segmental hypermobility may contribute to instability.	One unvalidated study of a clinical decision making rule; expert opinion.
Positive lumbar prone instability test	Less likely. Or if positive, may still indicate a need for stabilization exercises.	Due to poor muscular or structural stability, the joint may be sensitive to sheer loads.	One unvalidated study of a clinical decision making rule; expert opinion.
When symptomatic, symptoms may be exacerbated by relaxing splinting muscles (e.g., by heat or massage)	Thought to be less typical of mechanical low back pain except in some instances of very acute, severe pain.	Muscle splinting is a compensation mechanism to stabilize unstable joint.	Expert opinion, biomechanical plausibility
A variety of tests reflecting poor motor control (including hip extension movement pattern, inability to control pelvis, etc.)	Thought to be present in a variety of causes of recurrent and chronic LBP, but contribute to the suspicion of instability or at least as indicators for the need for a stabilization exercise program.	Poor motor control is one of the central concepts behind functional instability	Expert opinion, practice consensus, biomechanical research.

* Instability can also be associated with chronic back pain.

APPENDIX I: Generalized Joint Hypermobility

Generalized hypermobility is thought to be a different phenomenon from functional instability at a segmental level. It is often asymptomatic, but may be associated with joint pain. This condition is sometimes associated with connective tissue disorders (e.g., Ehlers-Danlos syndrome).

Making the Diagnosis

The patient is asked to demonstrate a discrete series of movements. Most people score less than 2 on this scale. Only three or four in a hundred healthy people score 4 or more points.

THE CRITERIA

The 1998 Beighton Criteria	
Major Criteria	**Minor Criteria**
Beighton score of 4 or more (see scoring system below)	Beighton score of 1—3 (or even 0 if aged over 50) (see scoring system below)
Joint pain in 4 or more joints for longer than 3 months	Pain in 1—3 joints, or back pain, for longer than 3 months
	Joint dislocation
	3 or more instances of damage to the soft tissues (lesions)
	Exceptionally tall, slim build with unusually long, slender fingers ('Marfanoid habitus')
	Thin or unusually stretchy skin, stretch marks or scarring from minor cuts
	Drooping eyelids, short-sightedness or slanting eyes
	Varicose veins, hernia or prolapse of the womb (uterus) or rectum

SCORING SYSTEM (9 possible points).

Award one point each of the patient can do the following:

	Left	*Right*
Hyperextend elbows (bend elbow backwards)….....	1	1
Hyperextend the knees.....	1	1
Bend the thumb back on to the front of the forearm....	1	1
Bend the little finger up at 90° (right angles) to the back of the hand…	1	1
Place hands flat on the floor with knees straight.....	1	

PUTTING IT ALL TOGETHER

Generalized hypermobility is determined by the presence of

- 2 major criteria **or**
- 1 major + 2 minor criteria **or**
- 4 minor criteria **or**
- 2 minor criteria + a first-degree relative (parent, child, brother or sister) with confirmed hypermobility.

APPENDIX II: Radiographic Hypermobility

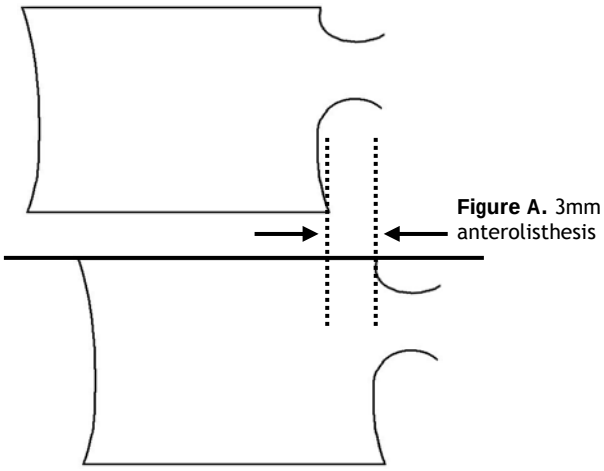


Figure A. 3mm anterolisthesis

Flexion view

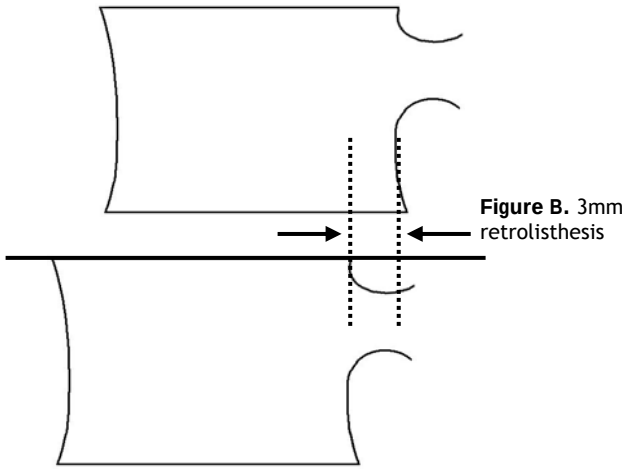


Figure B. 3mm retrolisthesis

Extension view

1. The combined findings of the 2 views above (which are stress views) must represent a <u>total sagittal translation</u> of > 4mm to meet the standard for radiographic hypermobility.
2. A or B alone does not indicate radiographic hypermobility.
3. The criterion is met in the diagram above by adding the listhesis in the flexion and extension views yielding a total sagittal translation of 6mm.
4. This criterion could be met in other ways. For example, a 1mm anterolisthesis on neutral could become a 6mm anterolisthesis on flexion for a total of 5mm of sagittal translation. (*Not shown*)

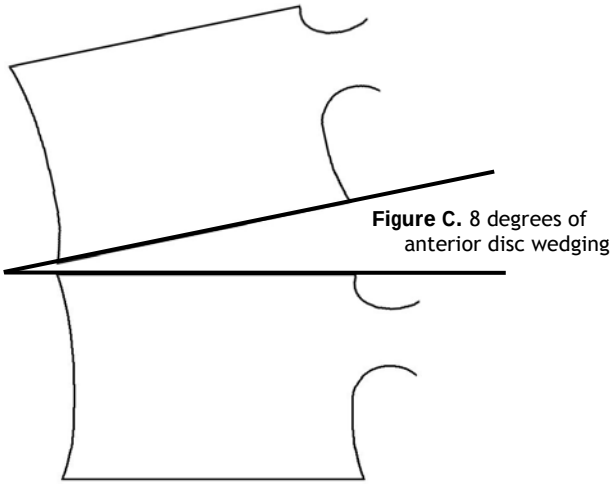


Figure C. 8 degrees of anterior disc wedging

Flexion view

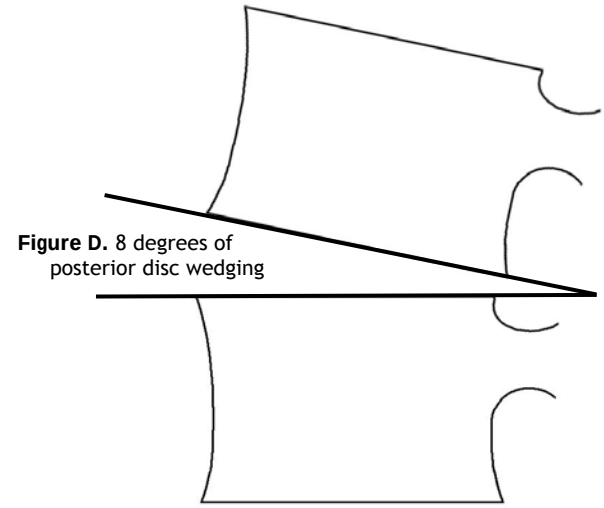


Figure D. 8 degrees of posterior disc wedging

Extension view

1. The combined flexion and extension findings (which would require stress views) must represent a total sagittal rotation of >10 degrees change from the neutral view to meet the standard for radiographic hypermobility
2. C or D alone does not indicate radiographic hypermobility
3. The criterion is met in the diagram above by a total sagittal rotation of 16 degrees.
4. This criterion could be met in other ways. For example, a 0 degree angle on extension could become 12 degrees of anterior wedging on flexion. (*Not shown*)

Authors: Dave Peterson, DC; Ronald LeFebvre, DC

Imaging section: Lisa Hoffman, DC, DACBR

Reviewed and Adopted by CSPE Committee

- Daniel DeLapp, DC, DABCO, LAc, ND
- Jaysun Frisch, DC
- Lorraine Ginter, DC
- Stover Harger, DC
- Sean Herrin, DC, CCSP
- Owen T. Lynch, DC
- Ryan Ondick, DC
- Anita Roberts, DC
- Bonnie Wickwire, DC, ND
- Laurel Yancey, DC

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