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PICTORIAL REVIEW

Upright positional MRI of the lumbar spine

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Supine magnetic resonance imaging (MRI) is routinely used in the assessment of low back pain and radiculopathy. However, imaging findings often correlate poorly with clinical findings. This is partly related to the positional dependence of spinal stenosis, which reflects dynamic changes in soft-tissue structures (ligaments, disc, dural sac, epidural fat, and nerve roots). Upright MRI in the flexed, extended, rotated, standing, and bending positions, allows patients to reproduce the positions that bring about their symptoms and may uncover MRI findings that were not visible with routine supine imaging. Assessment of the degree of spinal stability in the degenerate and postoperative lumbar spine is also possible. The aim of this review was to present the current literature concerning both the normal and symptomatic spine as imaged using upright MRI and to illustrate the above findings using clinical examples.

Introduction

Low back pain (LBP) is a common and costly problem in the UK.[1] Clinical presentation is non-specific with signs and symptoms being related to a variety of overlapping aetiological factors. Supine magnetic resonance imaging (MRI) has an established role in the assessment of spinal disorders[2]; however, its findings, such as stenosis and disc herniation, have both high false-positive and false-negative rates.[3–5]

Part of the reason relates to the supine patient position with hips and knees flexed, resulting in relative spinal flexion. This maximizes the dimensions of the canal and exit foramina, thus reducing the magnitude of any stenotic effect. Clinical symptoms can develop with sitting, standing, or dynamic manoeuvres (including flexion and extension) and may not be adequately assessed by supine MRI. Development of these symptoms reflects the morphological changes in normal or degenerate disco-ligamentous structures due to the effects of gravity, changes in size of the intervertebral foramen, and relative motion between adjacent vertebrae on assumption of the upright posture and with dynamic manoeuvres.

Therefore, upright and dynamic imaging is important, and a number of techniques are already available for such assessment. Plain radiographs use ionizing radiation and fail to visualize the soft tissues. There are also measurement errors associated with magnification, rotation and alignment artefacts related to imaging a three-dimensional structure in two-dimensions.[6] Myelography suffers from similar drawbacks, but is also invasive with risks of infection, contrast medium reaction, and headache.[7] Furthermore, myelography does not visualize the thecal sac and cauda equina/nerve roots in all cases due to dilution of contrast medium, and cannot assess the exit foramen.[8] Axially loaded supine MRI has been performed to simulate the upright position,[9] but may not truly reflect postural spinal changes related to muscle tone, loads on the lumbar spine that increase in a caudal direction rather than being uniform at each spinal level, and the effects of core muscle activation on the spine.[10]

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Improvements in available open MRI systems from increased field strength (mid strength = 0.5–0.6 T), field homogeneity, gradient generation, coil technology (quadrature coils), and faster image acquisition (dynamic equilibrium and magnetization transfer) have resulted in better signal-to-noise ratios, contrast and spatial resolution, and therefore, image quality. Open MRI systems with a vertical gap allow imaging under the influence of gravity in the upright position (seated or standing), with varying kinetic manoeuvres (flexion, extension, lateral bending, rotation etc,), as well as with the patient supine. Imaging in the physiologically representative upright position and with kinetic manoeuvres, allows accurate assessment and measurement of changes in the relationship between the components of the functional spinal unit and the potential to correlate radiological signs with positional symptoms.

This review illustrates how upright MRI can demonstrate changes from supine to the erect imaging and between-erect flexed and erect-extended positions in the normal, degenerate, and postoperative spine. The changes will be divided into the following sections: the intervertebral disc, central canal, exit foramen, spinal alignment, and spinal stability/instability. In addition, we review the current literature in this area,[11–23] which is a combination of both subjectively and objectively measured observations.

Imaging technique

The MRI system (Upright MRI, Fonar Corporation, Melville, NY, USA) has a 0.6 T horizontal field generated between two resistive magnets. A tilting table placed at right angles between these coils can be positioned at any angle from −20 to 90° (vertical), allowing supine and standing imaging. An MRI compatible seat can be added in the upright position. Extension is achieved by positioning of a small cylindrical cushion just above the lumbrosarcal junction. Flexion is achieved by leaning forward over a wedge-shaped cushion and supporting the hands on a horizontal bar. A dedicated lumbar spine quadrature coil is used for signal acquisition.

The imaging protocol for upright MRI includes standard sagittal and axial T1-weighted (W) fast spin-echo (FSE) and T2W FSE sequences through the lumbar spine in the neutral sitting position. Flexion sagittal T2W FSE and extension sagittal and axial T2W FSE images are performed when positional MRI is requested. This may be supplemented with sagittal and axial T2W FSE sequences in the standing position 5° off vertical (to minimize motion). Dedicated coronal and axial scouts are obtained for each of these positions (to minimize errors from rotation or lateral bending). All images are obtained with 4.5 mm section thickness and 0.5 mm overlap, acquisition time being approximately 3.5 min. Imaging parameters are described in Table 1. The lower field strength minimizes artefact from metal and chemical shift. Claustrophobic patients can also be safely imaged, without the need for prior sedation.[24] In addition, the upright position allows the study of patients with significant cardiac and respiratory disease who cannot tolerate supine imaging.

Normal postural alignment changes

On supine imaging, spinal alignment does not reflect the true postural effect of body weight, which can increase by 80%,[25] and the action of paraspinal and abdominal musculature.[11,12] Normally the upper lumbar spine undergoes increased extension in the upright sitting[11,13] and kneeling positions, the degree of extension decreasing in a caudal direction (Table 2).[10] The lumbrosacral junction, representing the angle between the posterior margins of the L5 and S1 vertebral bodies becomes less flexed,[15] and also more vertical to support the increased load in the upright position.[26] This flexed position becomes more prominent in the seated compared with the standing position.

Table 1 Imaging parameters

Sequence	Echo time (TE)	Time to repetition (TR)	Number of excitations (NEX)	Echo train length (ETL)	Field of view (FOV)/cm	Matrix	Echo spacing (ESP)
Sagittal T2W FSE	140	1734	2	13	32	220 × 220	–
Sagittal T1W FSE	17	671	–	3	32	220 × 220	13
Axial T2W FSE	135	2080	3	13	25	256 × 224	–
Axial T1W FSE	17	671	3	3	25	256 × 224	–

T1W FSE, T1-weighted fast spin-echo.

Table 2 Changes in anterior and posterior disc height (mean ± S.D. in mm) from the supine to upright kneeling position in normal subjects, from Lee et al.[10]

Level	Anterior disc height	Posterior disc height	Difference in means: anterior−posterior
L2/L3	2 ± 1.4	−1.4 ± 1.2	3.4
L3/L4	1.2 ± 1.4	−1.6 ± 1.1	2.8
L4/L5	0.9 ± 1	−1 ± 0.9	1.9
L5/S1	1.2 ± 0.9	−0.5 ± 0.8	1.7

On movement from flexion to extension, there is normally an increase in lumbar lordosis of 1.1−2.9° per vertebral level.[15] From a neutral upright seated to flexed position, paradoxical extension at L5/S1 or L4/L5 and L5/S1, can be observed in normal subjects,[16,17] whereas a change in anterior−posterior translation of between 1−1.5 mm. may also be evident.[15]

Intervertebral disc—morphological and signal intensity changes

Posterior disc contour

Normal Discs. From the supine to upright position, subjective observation has shown no change in the degree of posterior disc bulge[13,18]; however, objective measures show conflicting results. Schmid et al.[19] found no significant change on sitting, whereas Lee et al.[10] found a small increase (0.5 ± 0.4 mm at L2/3, 0.5 ± 0.6 mm at L3/L4, 1.1 ± 0.7 mm at L4/L5 and 0.6 ± 0.8 mm at L5/S1), which was most prominent at the L4/L5 level in normal/asymptomatic subjects in the kneeling position. These latter differences are related to the increased extension that occurs in the kneeling compared with the sitting positions. The majority of normal intervertebral discs probably show a small[10,19] but insignificant increase in posterior disc bulge[18,19] or a small decrease (<0.7 mm in the lower two discs)[20] from upright flexion to extension.

Degenerate discs. In a subjective study, Zamani et al.[18] reported that degenerate discs show no increased posterior disc bulge on sitting. However, our own observations and other reports suggest that posterior disc bulge does increase on upright imaging.[11,12] Similarly, a subjective study by Weishaupt et al.[13] showed a decrease in the degree of posterior disc bulge on upright flexion compared with supine in 7.5% (4/53) of discs and an increased degree of posterior disc bulge in 11.3% (6/53) of discs on upright extension compared to the supine position, whereas 1.9% (1/53) discs showed reduced disc bulge.

When comparing upright extension with flexion, Zamani et al. found that 57% (25/44) of degenerate discs showed no change, 41% (18/44) demonstrated an increase, and 2.2% (1/44) a decrease in posterior disc bulge.[18] Flexion was shown to decrease posterior disc bulge in some patients.[18] These findings are consistent with our observations and those reported by others[11,12] (Fig. 1).

Disc degeneration results in a posterior annular tear. From the supine to the upright position, there is increasing compressive force on the disc as a result of gravity and muscle activation. Telescoping of the spine results in circumferential bulging of degenerate discs,[11,12] these changes increasing in a caudal direction.[10] During extension, tension increases in the anterior longitudinal ligament (ALL), displacing the semi-liquid nucleus pulpous posteriorly where little resistance from the torn annulus allows posterior herniation.[13,18,27] As well as gravity, increased spinal extension in the upright position has an additional effect on posterior disc herniation.

Posterior disc bulges/herniations may also develop or increase in degenerate discs during flexion[13] (Fig. 2). We speculate this may result from weakness/dysfunction of the posterior longitudinal ligament (PLL) that may be involved in more extensive annular tears. Paradoxical extension during flexion in the lower lumbar spine may offer an alternative explanation in the lower lumber region.[16,17]

Whether these changes cause neural compression not only depends on the degree of posterior bulging (Figs. 3−5), but also on other factors, such as the size of the central canal, which may be congenitally small or narrowed due to facet hypertrophy. Even without neural comprise a change from disc protrusion to extrusion (Fig. 4) may be clinically significant.[13] Studies by Weishaupt et al.[13] and Zamani et al.[18] suggest that these are not infrequent occurrences. Weishaupt et al.,[13] in a study of 152 nerve roots reported a change in diagnosis (disc herniation, nerve root compromise, or foraminal stenosis) in 26.3% (40/152) between supine and flexion, in 22.4% (34/152) between supine and extension, and in 28.2% (43/152) between flexion and extension. Weishaupt et al.[13] found an increase in nerve root contact (34 to 45 nerves; 7.2%) and nerve root contact and displacement (10 to 13 nerves; 2%) on upright extension from the supine position.[13] However, changes more strongly associated with symptoms,[4,5] such as protrusion to extrusion (Figs. 3−5) and nerve root contact/displacement to nerve root compression were rare. Weishaupt et al.[13] also found a correlation between increase in visual analogue pain scores

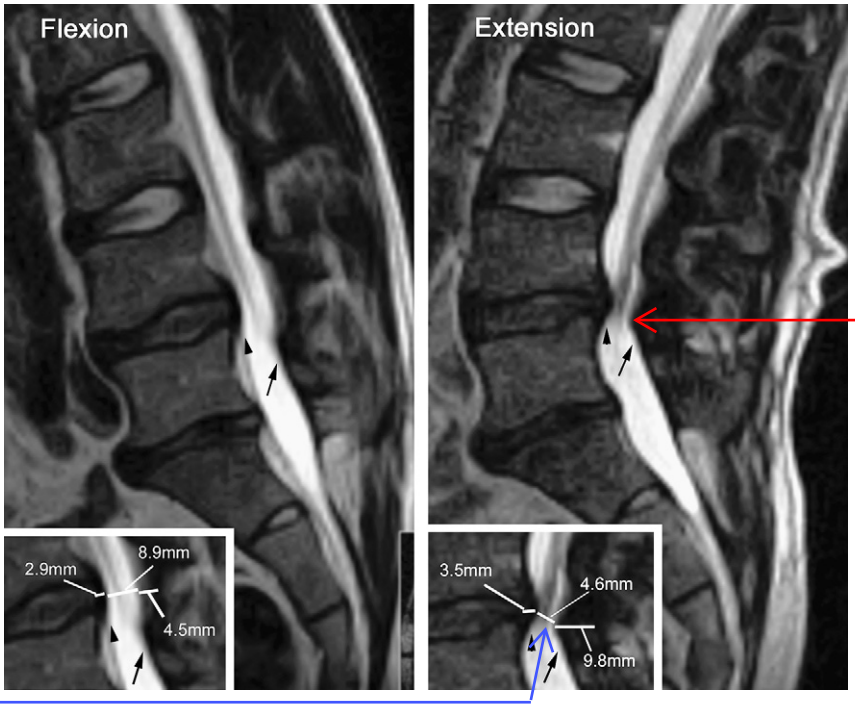

Figure 1 A 30-year-old man with non-specific LBP demonstrating increased posterior disc bulge on extension. Sagittal T2W images in seated flexion and extension demonstrate posterior L4/L5 disc bulge that increases on extension (arrowheads). There is also inward bulging of the ligamentum flavum on extension (arrows), contributing to narrowing of the central canal. Insets demonstrate the changes in the disc herniation and ligamentum flavum, which have resulted in a reduction in anteroposterior canal diameter of 48.3%.

and increased degree of disc herniation (increased by 22% points) or nerve root compromise (increased by 19% points) as observed in extension compared with flexion.[13]

Development of high intensity zones (HIZs)

The HIZ represents a radial annular tear, but conventional T2W MRI has a relatively poor sensitivity (26.7%) for identifying annular tears when compared to discography,[28] although the specificity in predicting LBP is high (79%) in patients selected to undergo discography.[29] We have found the HIZ to develop on extension (Fig. 6) and on assumption of the upright posture (Fig. 7) compared with the neutral position, and have also identified morphological changes in the HIZ from a globular and radial (neutral, flexion) to a more linear and vertical (extension) appearance. A possible explanation is that in the extended/upright position, raised intradiscal pressure and annular stresses force fluid out of the semi-liquid nucleus into the posterior annular tear resulting in increased T2W signal, which consequently renders it visible on MRI. This occurs particularly in the posterolateral disc and in the extended position.[30] Similar findings have been reported on supine axially loaded MRI[31] and help to explain the relatively poor sensitivity of conventional supine MRI for the detection of annular tears, but the clinical relevance of such findings is yet to be determined.

Central canal and lateral recess

Normal subjects

In normal subjects, on assumption of the erect from the supine position observational studies have found a reduction in the cross-sectional area (CSA) of the central canal and lateral recesses.[11,12] Schmid et al.[19] reported a 5.2% reduction (235.8 to 224.1 mm^2, $p > 0.05$) in central canal CSA between the supine extended and upright extended positions, with a 16.4% reduction (268 to 224.1 mm^2, $p < 0.0001$) from the upright flexed to extended position.[19] However, asymptomatic subjects do not have a reduction in CSA to that which was borderline (100–130 mm^2) or unequivocally (<100 mm^2) stenotic.[32]

The reduction in central canal and lateral recess CSA is due to a combination of factors. There is

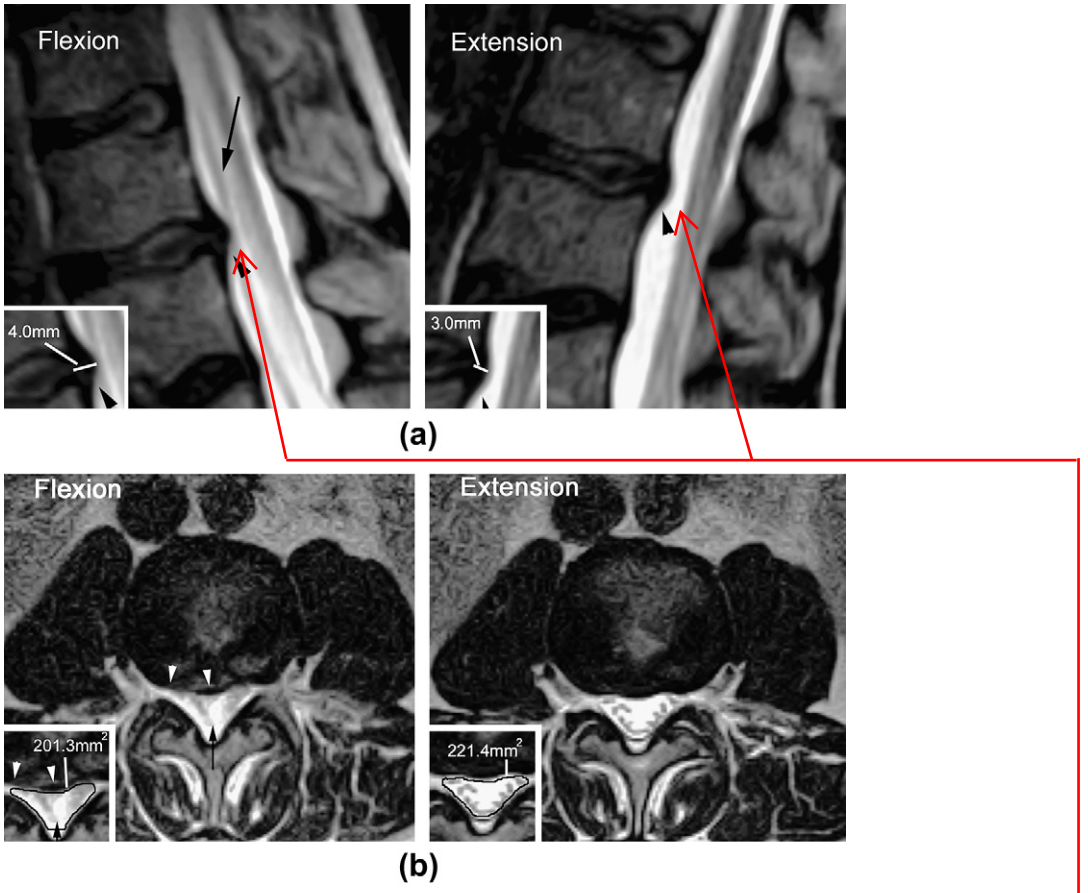

Figure 2 A 56-year-old man with left leg pain demonstrating increased bulging of the L1/L2 disc on flexion. Sagittal T2W images in sitting flexion and extension (a) and axial T2W images in flexion and extension (b). There is increase in disc bulge on flexion compared with extension (arrowheads), which has resulted in some narrowing of the right lateral recess. There is also a reduction in the volume of cerebrospinal fluid around the nerve roots on flexion. Insets demonstrate a reduction in CSA of the central canal of 9.1% in flexion.

a reduction in disc height due to body-weight and muscle activation.[11,12,19] This has been demonstrated as a small increase in the anterior disc height and a reduction in the posterior disc height in the upright position, with a greater change at the L2/3 and L3/4 levels.[10] This results in redundancy of the cauda equina, meninges and ligaments (ligamentum flavum),[11,12,19] the latter

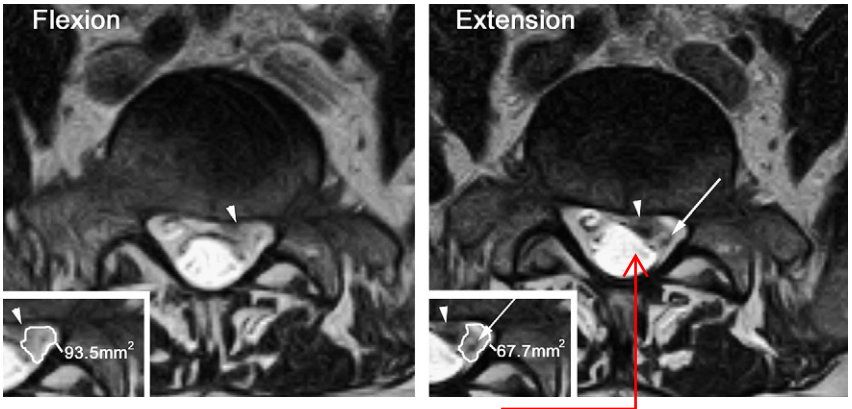

Figure 3 A 58-year-old man with left leg pain demonstrating neural compression on extension due to increasing L5/S1 disc protrusion narrowing the lateral recess. Axial T2W images in seated flexion and extension demonstrate a left L5/S1 paracentral disc protrusion, which increases on extension (arrowheads), displacing the thecal sac and compressing the left S1 nerve root. Insets demonstrate a reduction in CSA of the lateral recess of 27.6%.

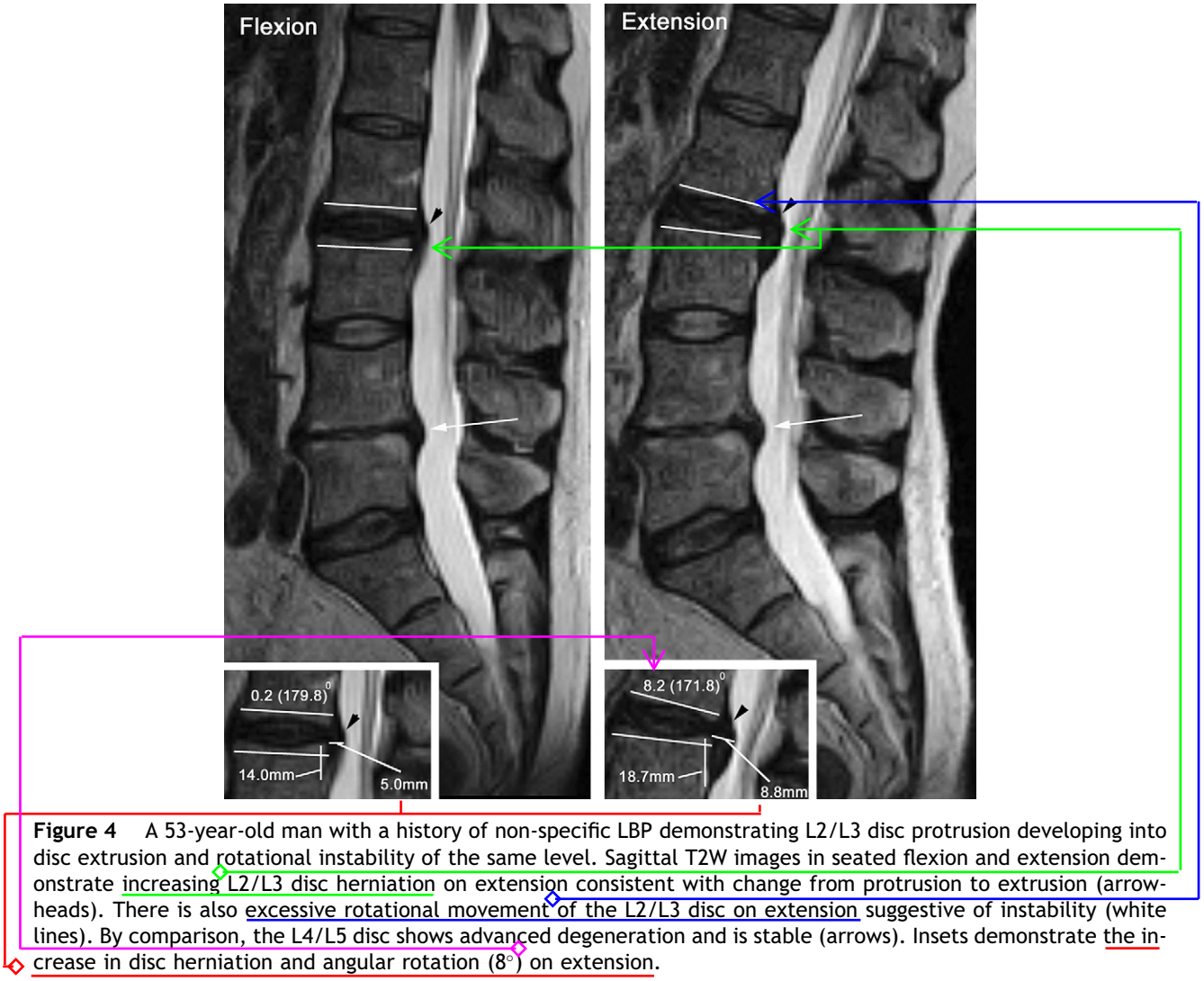

Figure 4 A 53-year-old man with a history of non-specific LBP demonstrating L2/L3 disc protrusion developing into disc extrusion and rotational instability of the same level. Sagittal T2W images in seated flexion and extension demonstrate increasing L2/L3 disc herniation on extension consistent with change from protrusion to extrusion (arrowheads). There is also excessive rotational movement of the L2/L3 disc on extension suggestive of instability (white lines). By comparison, the L4/L5 disc shows advanced degeneration and is stable (arrows). Insets demonstrate the increase in disc herniation and angular rotation (8°) on extension.

being the main contributor to reduction in canal size in the normal subject, increasing in thickness from 3.3 to 4.3 mm ($p < 0.0004$) from supine extension to upright extension.[19] At the same time, there may be a smaller effect due to increased posterior disc bulge from the supine to upright positions, which increases in the caudal direction, as previously discussed.[10] From flexion to extension, there are similar changes in ligamentum flavum thickness (from 1.8 to 4.3 mm, $p < 0.0001$) with a smaller contribution from increasing posterior disc bulge.[10,19] These changes, when occurring from the supine to the erect position, are described as the telescoping effect.[11,12]

Symptomatic subjects

In the symptomatic subject, there is a comparable reduction in central canal CSA from the supine to the upright extended position (Table 3; Fig. 7) and from flexion to extension (Table 3; Figs. 1, 4, 5, and 8).[13] The smallest CSA is found in upright extension compared with the supine position.[11-13] Degeneration of the ligamentum flavum and disc further contribute to this narrowing,[11,12] which results in increased nerve root and thecal sac laxity (Figs. 5, 7 and 8), loss of disc height and posterior disc herniation[13] (Figs. 1, 5, 7, and 8). There is a greater and increasing contribution from the disc, compared to the normal spine. With increasing degrees of disc herniation, there is an associated increased reduction of CSA (Table 3).[13] We have found that the standing position can reveal occult narrowing compared with seated extension (Fig. 7). The greater the degree of disc degeneration, the greater the disc height loss compared to normal.[11,12,14] Complete loss of CSF around nerve roots on T2W images is a good sign of central canal stenosis[33] and may be seen with dynamic extension (Figs. 5 and 8) and erect imaging (Fig. 7).

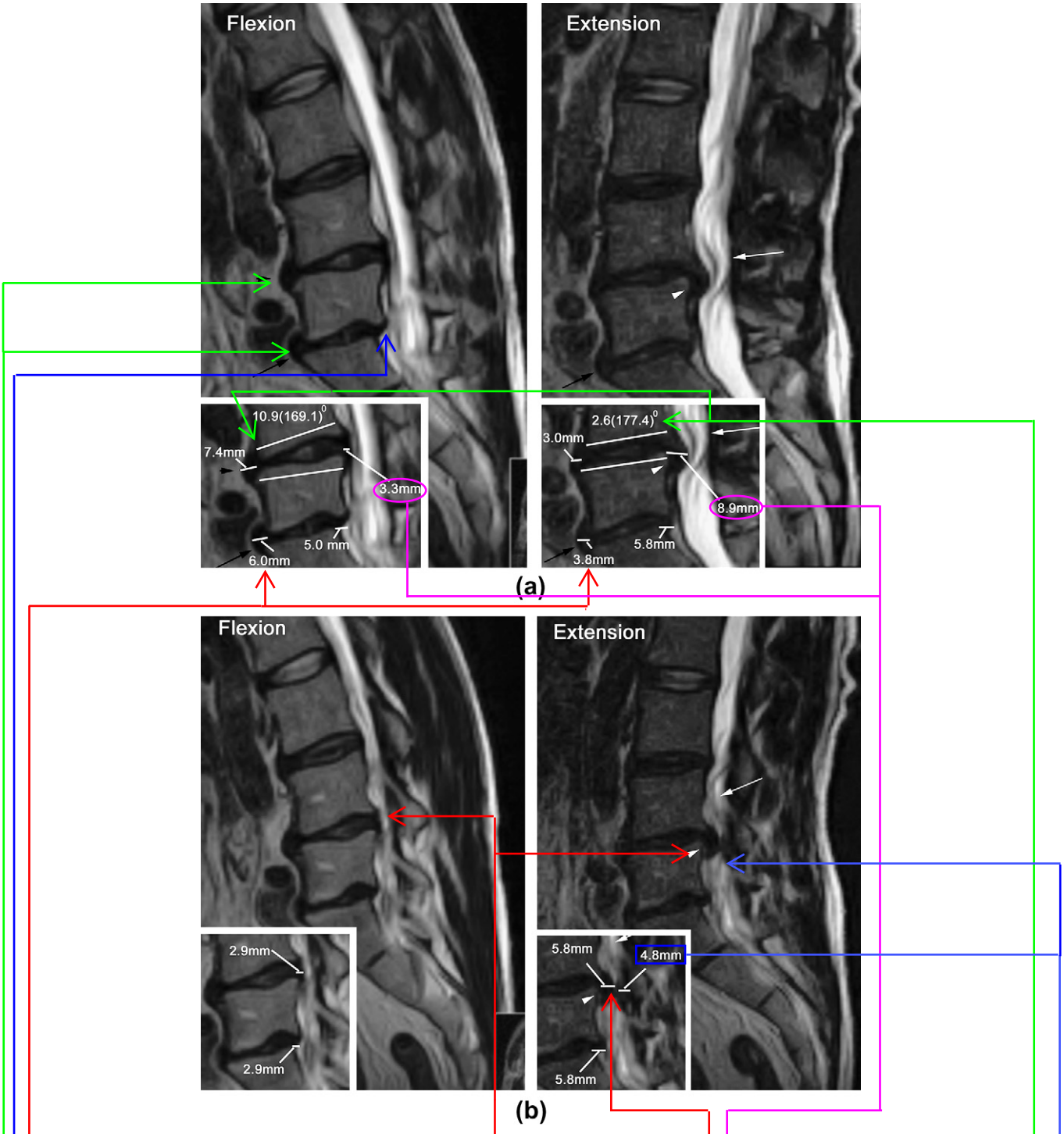

Figure 5 A 37-year-old woman with previous discectomy at the L4/5 and L5/S1 levels presenting with recurrent right calf pain demonstrating central canal and lateral recess stenosis from a disc extrusion developing on extension. Sagittal T2W images through the midline in flexion and extension (a) and through the right parasagittal level in flexion and extension (b) demonstrate development of a disc herniation at the L4/L5 level on extension (white arrowheads), which compresses the lateral recess on the right. Increased redundancy of the nerve roots on flexion (white arrows) contributes to this narrowing. Note the increase in anterior disc bulge in flexion at the L4/5 and L5/S1 level (black arrowheads, black arrow). There is also minor L5/S1 retrolisthesis, which appears stable (black arrow). Midline insets (a) demonstrate the increase in anterior disc herniation (59.5%) on flexion, posterior disc herniation on extension (36.7%), and reduced anteroposterior central canal diameter on extension (49.1%). The degree of angular rotation (8.3°) at the L4/L5 level is also indicated. Parasagittal images (b) show the increase in disc herniations (50% L4/L5, and 50% L5/S1) and the increase in anterior bugling of the ligamentum flavum (68.8%) on extension.

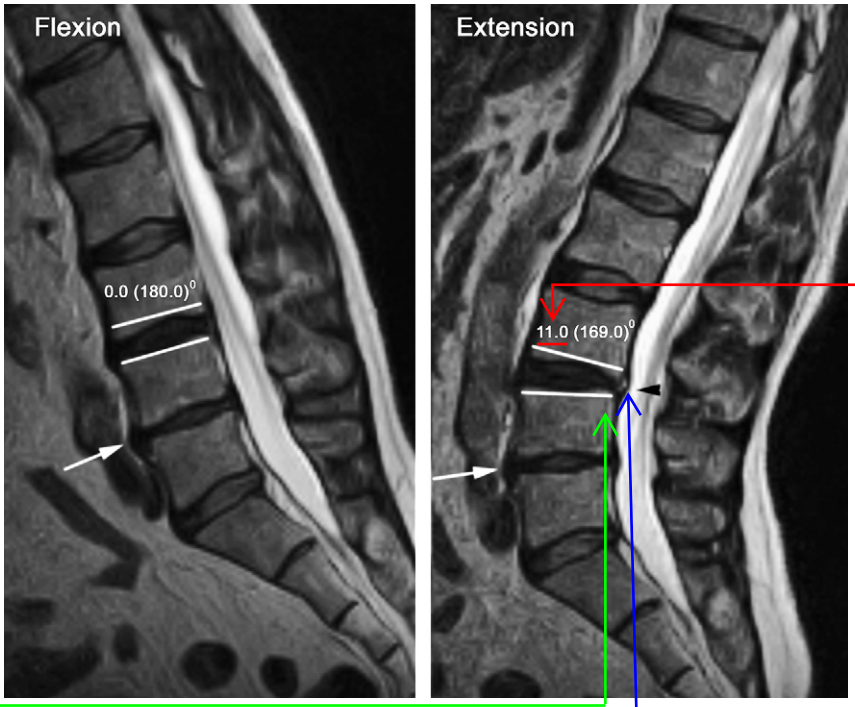

Figure 6 A 56-year-old man with non-specific LBP demonstrating development of a HIZ on extension and rotational instability. Sagittal T2W images in seated flexion and extension demonstrate a HIZ that develops posteriorly in the moderately degenerate L3/L4 disc on extension (arrowhead). There is hypermobility of the same level (white lines), with apposition of the end-plates posteriorly in extension suggestive of instability. There is 11° of angular rotation at this level. This level responds differently to other levels, particularly L4/L5, which shows more advanced disc degeneration (arrow).

It should be noted that with flexion, compared with the supine and neutral upright positions, there is normally anterior movement of the nerve roots, which will contact but not be compressed by the disc. [4,11] This is reported in 11.1% of nerves (17/152 nerves from extension to flexion)[13] (Figs. 2a, 5a and 7a).

Exit foramen

Both subjective[8,11–13,18] and objective[19,20] studies have demonstrated a reduction in exit foramen height from supine to upright and from flexed to extended positions. Normal subjects demonstrate an average reduction in foraminal CSA of between 33.9% (at L4/5, L5/S1)[20] to 35.6% (reduction of 167.4 to 107.9 mm^2 from L1/2 to L5/S1)[19] from flexion to extension in the upright position.

In symptomatic patients, subjective assessment of foraminal stenosis has been made using a four-grade scale described by Wildermuth et al.,[8] with increasing grades reflecting reduced epidural fat surrounding the exiting nerve root (Table 4).[13] Wildermuth et al.[8] demonstrated that a slightly greater number of subjects were classified with higher grades of foraminal stenosis as the position changed from supine to upright (Table 4). However, this was only found in the upright extension position in the study by Weishaupt et al.,[8] with upright flexion resulting in reduced grading of foraminal stenosis.[13] The change in grade on flexion may, in part, reflect the development of spondylolisthesis ($n = 5$, Wildermuth[8]), whereas increased grades in extension reflect foraminal narrowing from approximation of the pedicles, increased disc bulging, and redundancy of the ligamentum flavum.[11,12]

Wildermuth et al.[8] and Weishaupt et al.[13] identified only one and two subjects, respectively, who developed foraminal nerve root compression on changing from upright flexion to extension (Fig. 9). Weishaupt et al.[8] also found that worsening foraminal narrowing was associated with a significant increase in pain score (21% points $p = 0.046$).[13] Two cadaveric studies have revealed that reduction in disc height of greater than 4 mm will result in nerve root compression.[34,35]

Spinal instability

Imaging in the supine position and with non-dynamic methods can only identify indirect radiological signs of instability (i.e., degenerative

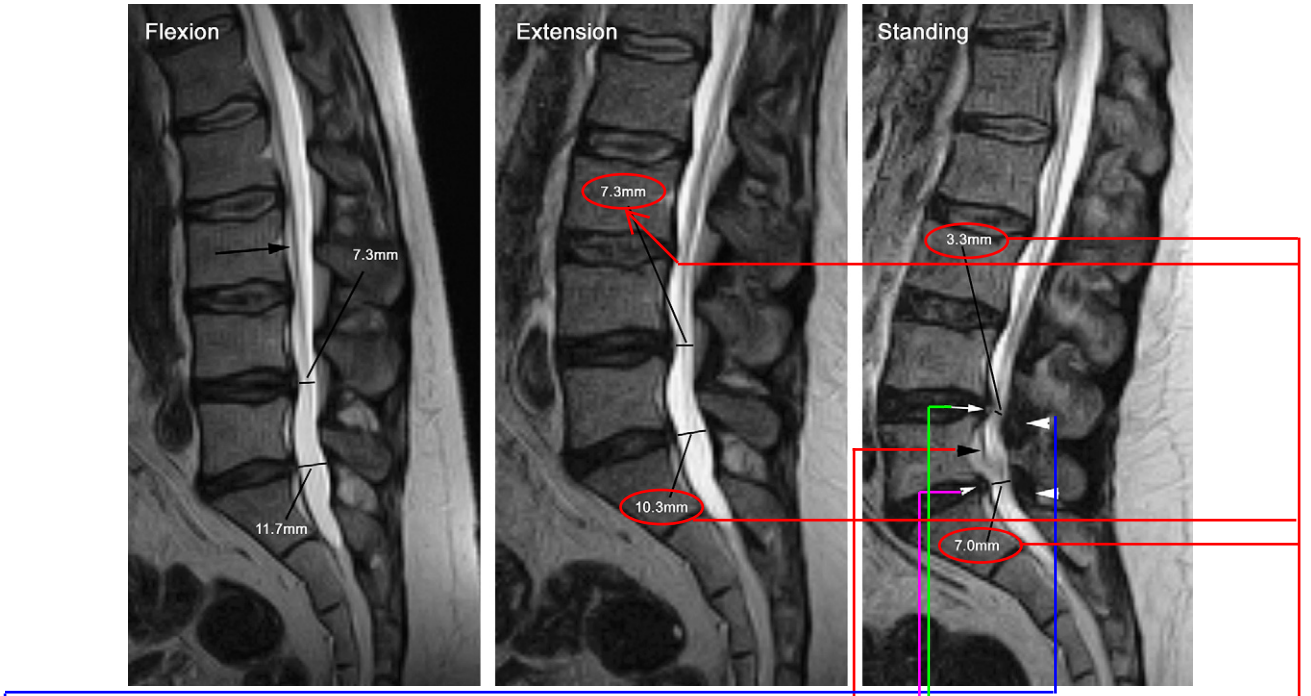

Figure 7 A 37-year-old postman with a history of LBP and sciatica on carrying his postbag, demonstrating development of a HIZ and central canal stenosis on standing. Sagittal T2W images in seated flexion, seated extension, and standing. There is congenital canal stenosis and mild disc degeneration, with minor posterior disc bulges at the lower two discs. On standing, there is development of a HIZ at the L4/L5 level (white arrow) and more prominent disc bulges at L4/L5 (white arrow) and L5/S1 (small white arrowhead), with marked inward bulging of the ligamentum flavum (large white arrowheads). Redundancy of the nerve roots (black arrowhead) contributes to development of severe stenosis at L4/5 and mild-moderate central canal stenosis at L5/S1. Overall there is a reduction of anteroposterior diameter at the L4/L5 and L5/S1 of 0 and 12%, respectively, between flexion and extension, and 54.8 and 40.2% between flexion and standing.

changes of the disc, ligaments, and facet joints) and some direct signs (malalignment of the vertebral bodies).[36] Upright and positional MRI can demonstrate changes in intersegmental motion that may correlate with clinical symptoms of LBP and neurogenic claudication. As yet, however, no published studies have dealt with this topic.

Instability can be considered as part of the normal degenerative process of the lumbar spine, which has three phases. Initially, there is abnormal motion of the spinal segment (disc, adjacent vertebrae, ligaments, facet joints) and pathological signs of degeneration are minimal; this stage being termed ''spinal dysfunction''. The signs of relative spinal motion (e.g., translation and sagittal rotation of the vertebral bodies with respect to each other)[36] can be uncovered with upright/positional MRI (Fig. 10).

During the second or ''instability phase'', signs of degeneration are more prominent and there is increased and abnormal intersegmental movement. Instability can be demonstrated as relative hypermobility at the spinal motion segment compared with adjacent motion segments on positional MRI[11]

Table 3 The thecal sac CSA in the supine, flexed seated, and extended seated positions divided into groups with different disc morphology in symptomatic subjects

Disc morphology	No.	Cross-sectional area (CSA)			Significant *p*-value (% reduction in CSA)		
		Supine	Flexion	Extension	Supine versus flexion	Supine versus extension	Extension versus flexion
All	76	175.3 ± 57.4	174.8 ± 58.7	158.4 ± 55.7	–	<0.001 (9.6)	<0.001 (9.4)
Normal	23	178.6 ± 50.2	181.2 ± 52.4	165.5 ± 40.5	–	– (7.3)	– (8.6)
Bulging	14	172.3 ± 50.0	175.9 ± 52.2	158.2 ± 53.3	–	0.02 (8.1)	0.07 (10.9)
Protrusion	22	181.0 ± 70.4	177.4 ± 70.5	161.2 ± 67.9	–	<0.001 (10.9)	<0.001 (9.1)
Extrusion	17	165.9 ± 57.5	161.9 ± 58.1	144.6 ± 60.1	–	0.001 (12.8)	0.01 (10.6)

CSA is measured in mm ± S.D. and *p*-values are stated if significant. Based on Weishaupt et al.[13]

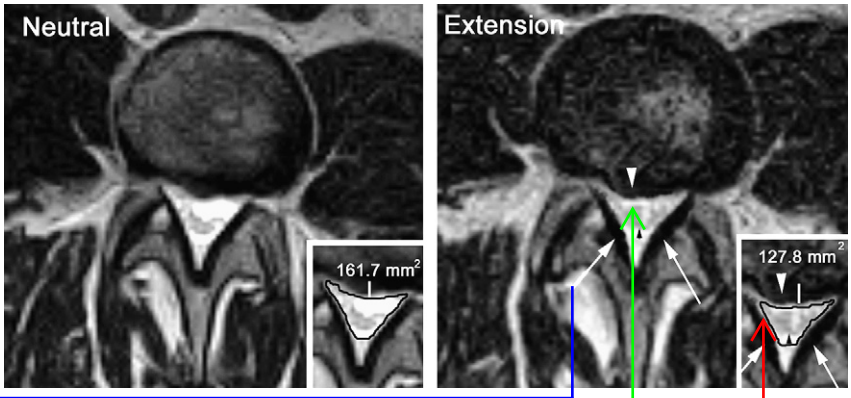

Figure 8 A 50-year-old woman with LBP and decreased sensation in the right leg, worse on extension demonstrating development of central canal and lateral recess stenosis. Axial T2W images at L4/L5 in seated neutral and extension demonstrate an increase in posterior central disc protrusion (white arrowhead), which together with thickening of the ligamentum flavum (white arrows) causes central canal and right lateral recess stenosis. Insets demonstrate a reduction in CSA of the central canal of 20.1%.

(Figs. 4, 6 and 11). Excess extension can in turn increase the degree of foraminal, central and lateral recess stenosis,[11–13] which may correlate with increased levels of pain.[13] Instability of a degenerative or isthmic spondylolisthesis can increase central or exit foraminal stenosis. The disc below a degenerate spinal level can be susceptible to degeneration and can be identified by increased degree of motion.[14] In the postoperative spine, damage to the paraspinal musculature and ligaments, and reduced motion at levels of fusion together with increased motion at the adjacent levels contribute to instability.[11,12,36]

As degeneration progresses, fibrosis and osteophytosis result in re-stabilization and consequential reduction in movement (third phase).[37] This ''re-stabilization phase'' can be difficult to distinguish from the instability phase without positional imaging (Figs. 4 and 5),[37] an important consideration if surgery is being contemplated. How often and when this distinction needs to made remains to be determined.

In the case of degenerative and isthmic spondylolisthesis, most appear stable, with no significant positional change in either angular rotation or horizontal translation.[21]

As the degree of degenerative disc disease increases, the amount of angular rotation from the supine to the erect position significantly increases, with maximal rotation occurring at a normal level below the slip.[14]

Assessment of orthopaedic hardware

In vivo studies have been performed to determine the effect of novel orthopaedic devices. One such device, the X-STOP (St. Francis Medical Technologies, Concord, CA, USA), is placed between adjacent spinous process distracting them and putting the spine into a flexed position. The consequent tension on the adjacent soft tissues and bones reduces encroachment on the central canal and exit foramen, potentially reducing symptomatic spinal stenosis and cauda equine/nerve root compression. Studies on the X-STOP device have shown it to increase the CSA of the canal and exit foramen (Table 5) without significantly changing spinal posture[22] or range of motion.[23]

Limitations of the technique

Although the lower field strength of the system results in a reduced signal:noise ratio, and thus,

Table 4 Position dependent changes in foraminal stenosis in symptomatic subjects ($n = 30$) as identified in studies by Wildermuth et al.[8] and Weishaupt et al.[13]

	Percent grade increase	
	Wildermuth	Weishaupt
Supine versus upright flexion	3.1–3.9 −1.5–1.6	3.9 −11.8
Supine versus upright extension	5.6–6.6 −0.8–1.1	10.5 −3.9
Upright flexion versus upright extension	1.2–2.3 −2–2.3	15.2 −10.5

The foraminal grades are defined as follows[8,13]: grade 0 = normal foramen, with a normal posterolateral disc margin and normal morphology of the foraminal epidural fat (oval or inverted pear shape). Grade 1 = slight foraminal stenosis and deformity of the epidural fat, with the remaining fat still completely surrounding the exiting nerve root. Grade 2 = marked foraminal stenosis, with epidural fat only partially surrounding the nerve root. Grade 3 = advanced stenosis with obliteration of the epidural fat.

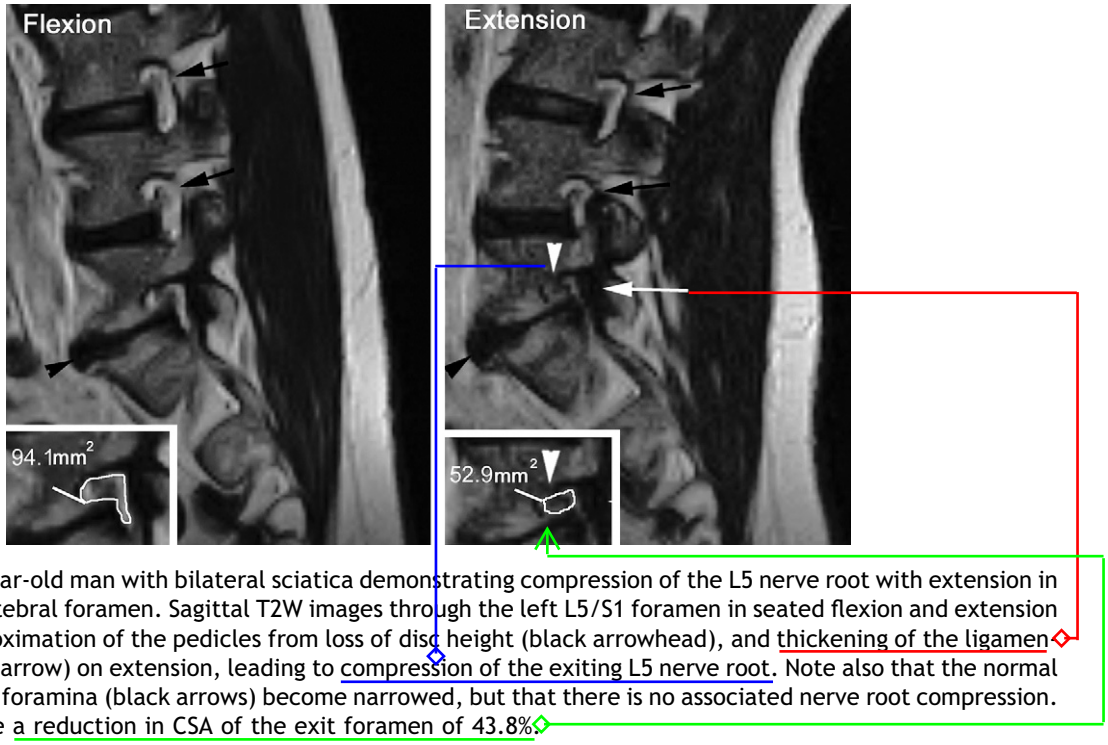

Figure 9 A 20-year-old man with bilateral sciatica demonstrating compression of the L5 nerve root with extension in the L5/S1 intervertebral foramen. Sagittal T2W images through the left L5/S1 foramen in seated flexion and extension demonstrate approximation of the pedicles from loss of disc height (black arrowhead), and thickening of the ligamentum flavum (white arrow) on extension, leading to compression of the exiting L5 nerve root. Note also that the normal L3/4 and L4/5 exit foramina (black arrows) become narrowed, but that there is no associated nerve root compression. Insets demonstrate a reduction in CSA of the exit foramen of 43.8%.

overall reduced image quality compared with high-field magnets, we have found that image quality is certainly adequate for the demonstration of lumbar canal stenosis and nerve root compression. This fact outweighs the limitations of closed high-field systems, which to our knowledge, do not allow for any form of erect lumbar spinal imaging.

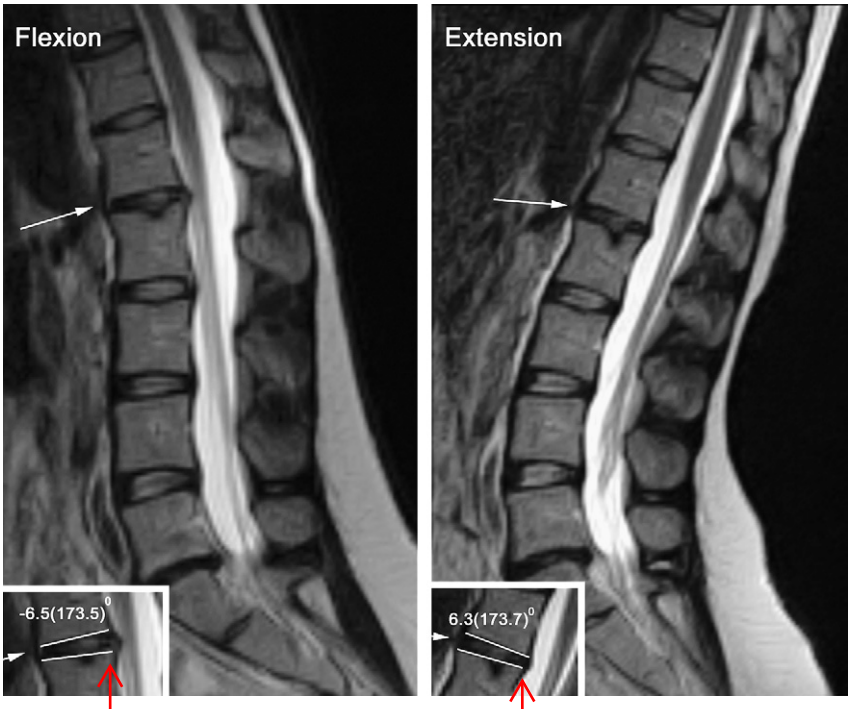

Figure 10 A 26-year-old man with a history of previous L5/ S1 discectomy and sciatica presenting with non-specific back pain, demonstrating spinal dysfunction. Sagittal T2W images in seated flexion and extension demonstrate early L1/L2 disc degeneration (arrows) with greater sagittal rotational motion (white lines) than accepted at this level. There is moderate L5/S1 disc degeneration, which shows little segmental instability. Insets demonstrate the change in angular rotation of 12.8° from flexion to extension.

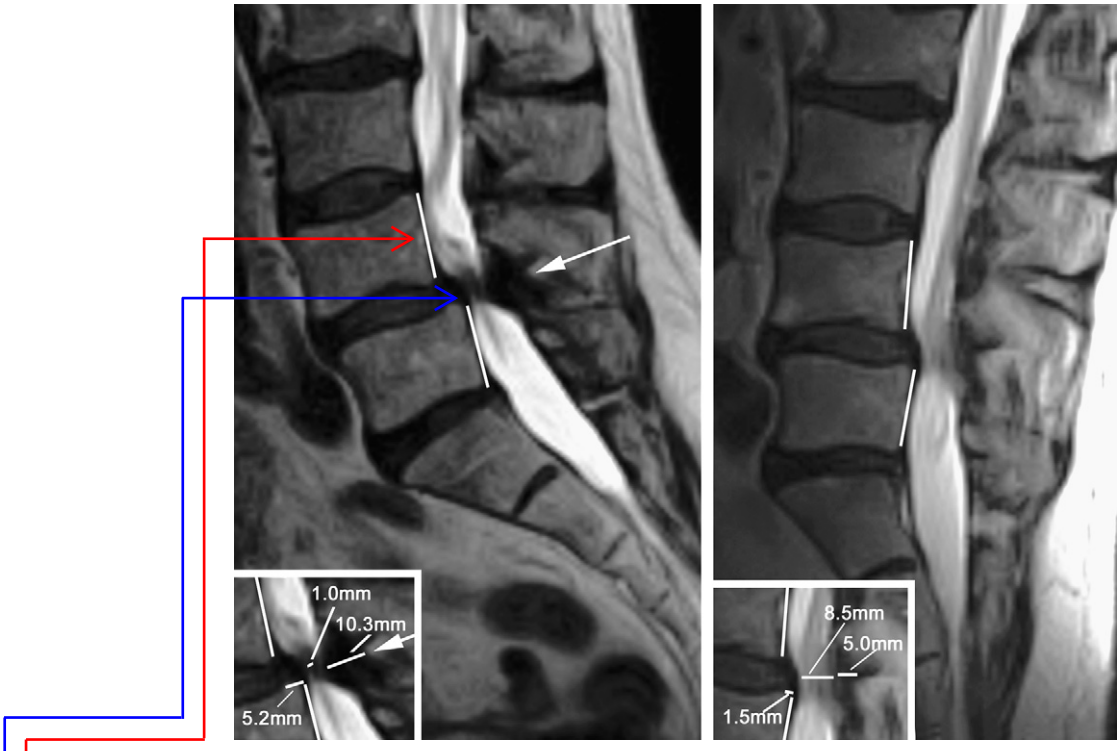

Figure 11 A 63-year-old man with a history of bilateral sciatica, and position-dependent lower back pain. MRI demonstrates grade 1 degenerative spondylolisthesis and translational instability with central canal stenosis. Sagittal T2W images in seated flexion and neutral positions demonstrate slightly increased forward slip with flexion (lines), associated with a greater degree of central canal stenosis (white arrow). Insets demonstrate the increase in forward slip (71.2%), increase in ligamentum flavum bugling (51.5%) and resultant reduction central canal anteroposterior diameter (88.2%) from neutral to flexed positions.

The extent of flexion and extension that can be performed is limited by the physical parameters of the chair[15] and the position that the patient can most comfortably hold without movement. This will improve with even faster image acquisition and real-time imaging. Additionally, the seated position may also underestimate the true extent of disease due to relative flexion of the lumbrosacral junction compared with the standing position. Imaging in the position of symptoms may be worthwhile, but raises the issue of motion degrading images.

Table 5 The change in CSA of the central canal and exit foramen with flexion, extension, standing and sitting before and after insertion of the X-STOP device

Position			Preoperative CSA	Postoperative CSA	Change	*p*-Value
Sitting	Central canal		76.8	93.2	16.4	0.011
	Exit foramen	Left	–	–	–	–
		Right	–	–	–	–
Flexion	Central canal		97.7	99.8	2.1	0.82
	Exit foramen	Left	100	118.7	18.7	0.023
		Right	95.6	111.4	15.8	0.233
Extension	Central canal		84.4	92.4	8	0.363
	Exit foramen	Left	82.3	98.4	16.1	0.027
		Right	82.3	93.1	10.8	0.09
Standing	Central canal		74.4	91.7	17.3	0.003
	Exit foramen	Left	–	–	–	–
		Right	–	–	–	–

Based on a study of 26 subjects by Siddiqui et al.[23] CSA is measured in mm^2.

Many studies have commented on difficulty in visualizing the structures of the exit foramen due to section thickness and degree of patient rotation and lateral flexion.[13] This has made it difficult to identify the exit foramen in some cases and to make an objective assessment of changes in size.[8,13,18] Consequently, subtle differences in canal and lateral recess dimensions can be difficult to appreciate if sections are not matched exactly between different imaging positions.

Conclusions

Conventional high-field MRI with the patient in the supine position is now widely available and remains the technique of choice for the investigation of degenerative lumbar spine disorders associated with lower limb symptoms. However, there is no doubt that clinically relevant spinal canal stenosis can be uncovered by imaging in the erect position. In cases where conventional MRI shows no evidence of cauda equina or lumbar nerve root compression in the setting of convincing clinical symptoms that warrant surgical intervention, re-imaging in the upright position, with the addition of flexion and extension, is recommended.

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