

University of Connecticut DigitalCommons@UConn

Articles - Patient Care Patient Care

2-2012

Tandem Spinal Stenosis: A Case of Stenotic Cauda Equina Syndrome Following Cervical Decompression and Fusion for Spondylotic Cervical Myelopathy

Brian T. Swanson University of Connecticut School of Medicine and Dentistry

Follow this and additional works at: http://digitalcommons.uconn.edu/pcare articles



Part of the Medicine and Health Sciences Commons

Recommended Citation

Swanson, Brian T., "Tandem Spinal Stenosis: A Case of Stenotic Cauda Equina Syndrome Following Cervical Decompression and Fusion for Spondylotic Cervical Myelopathy" (2012). Articles - Patient Care. 39. http://digitalcommons.uconn.edu/pcare articles/39

Case Report

Tandem spinal stenosis: a case of stenotic cauda equina syndrome following cervical decompression and fusion for spondylotic cervical myelopathy

Brian T Swanson

University of Connecticut Health Center, Farmington, CT, USA

Tandem spinal stenosis is a clinical phenomenon which may cause a functional loss related to neurologic compression in numerous areas of the spinal cord. In this phenomenon, the second area of symptomatic neurologic insult is not revealed until the primary symptomatic area has been treated. This case describes a 71-year-old male referred to physical therapy 4 weeks following a combined anterior/posterior C3/4 decompression and fusion for treatment of cervical spondylotic myelopathy. Approximately 8 weeks post-operatively (4 weeks after initiation of physical therapy), the patient began to complain of bilateral lower extremity weakness, primarily with climbing stairs. At 12 weeks post-operatively, the patient developed bowel incontinence and saddle paresthesia. Magnetic resonance imaging revealed multiple levels of critical stenosis of the lower thoracic and upper lumbar spine, which resulted in referral for surgical intervention. Following surgical decompression there was complete recovery of lower extremity strength, saddle area sensation and bowel function. This case highlights the need for the clinician to remain vigilant for concomitant pathology despite successful surgical intervention. A thorough knowledge of the presentation of various spinal disorders, as well as a thorough neurologic examination, is required to accurately recognize both candid and subtle red flags requiring immediate referral for surgical intervention.

Keywords: Cauda equina syndrome, Concomitant conditions, Myelopathy, Spinal stenosis

Background

Tandem spinal stenosis (TSS) is an infrequent, but clinically important phenomenon, with a reported incidence of between 5 and 28% of spinal stenosis cases. 1-3 Tandem stenosis is a distinct syndrome, generally as a result of spondylotic degeneration, which results from symptomatic canal narrowing of multiple areas of the spine resulting in significant neurologic compression. 1-3 Typically, patients with tandem stenosis will present with signs of intermittent neurogenic claudication, progressive gait disturbance, and findings of mixed myelopathy and polyradiculopathy in both the upper and lower extremities. Frequently it is not until after surgical correction of the primary symptomatic area that the second area of symptomatic stenosis becomes evident. 1,3

Cervical spondylotic myelopathy (CSM) is the most common cause of spinal cord injury in older adults.⁴⁻⁶ Degenerative changes with radiographic evidence of compression are evident in up to 50% of the population

Correspondence to: B T Swanson, Department of Rehabilitation, University of Connecticut Health Center, 263 Farmington Ave, Farmington, CT 06030, USA. Email: bswanson@uchc.edu

older than 55, but only 10% proceed to have symptoms of nerve root or spinal cord compression.^{4,5} The pathology of spondylotic myelopathy is caused by degenerative changes of the disc, facet joints, hypertrophy of the ligamentum flavum, uncovertebral hypertrophy, and the possibility of a congenitally small central canal.^{7–10} This condition typically occurs between the ages of 50-70, with complaints of insidious onset, 3:2 male/female respectively. 7-9,11 The primary initial symptoms of patients with CSM are frequently gait disturbances due to compression or degenerative changes of the spinocerebellar and corticospinal tracts (posterior column).^{8,9,12} This is characterized by a spastic or ataxic gait^{7,12} with a wide base of support and stooping posture, 13-15 frequently described as a 'sticky footed gait'. Changes in the upper extremities typically occur later. A loss of fine motor control is the most frequent complaint, 13 typically manifesting as complaints of clumsy hands and/or difficulty writing.8,9,12 Upper extremity weakness may be present in a lower motor neuron (myotomal) distribution at the level(s) of insult. The most common presentation is upper motor neuron (UMN) weakness occurring distally and extending to the

lower extremities.^{9,13} Sensory loss is frequently present, with vibration sense being most pronounced, followed by loss of pain and temperature sensitivity.¹³ Touch sensation is frequently, but not always, unchanged.¹³ Urgency/incontinence of urine and occasionally of bowel may be present in advanced cases. Cases of primary central canal stenosis may not include radicular symptoms to the upper extremities, but will present with long tract signs only.^{9,13} The presentation during physical examination will include: generalized hyperreflexia, clonus more likely in the lower than upper extremities, a positive Hoffmann's sign, a positive L'hermitte's sign, and positive Babinski reflexes.

The typical post-operative presentation of the patient undergoing surgical correction for CSM is expected to be grossly unchanged from the preoperative state. As such, it is important to note that surgery for CSM is intended to halt the progression of the disease, without expectation of significant functional return. In a series of patients undergoing surgery for CSM, Cheung *et al.* Perorted a 37% return of upper extremity function, 23% of lower extremity function, and only 17% return of sphincter function following decompression.

Lumbar spinal stenosis (LSS) is a commonly encountered condition,¹⁷ and may also occur as a component of TSS.^{1–3} The typical LSS patient is more likely to be male, in the fifth or sixth decade of life.¹⁷ Clinically, the most frequent signs are leg pain, which is most often exacerbated by walking or extension and relieved with flexion; altered reflexes; weakness in a myotomal distribution; and decreased



Figure 1 Pre-operative cervical sagital T2 image demonstrating significant narrowing of spinal canal, with significant area of increased signal intensity in the spinal cord, representing myelomalacia.

sensation to touch and vibration.¹⁷ Gait is frequently flexed and with a wide base of support, ^{18,19} similar to that of CSM. Cauda equina syndrome (CES) is a rare, but serious, complication of LSS.^{20,21} Typical early signs and symptoms include low back and leg pain as well as abnormalities of the bulbocavernosus and ischiocavernosis reflexes.²¹ As symptoms progress, saddle area sensory disturbances and bilateral sciatica are typically present, followed by motor weakness of the lower extremities, bowel/bladder dysfunction which may progress to bowel incontinence and/or urinary retention, and reduced sexual

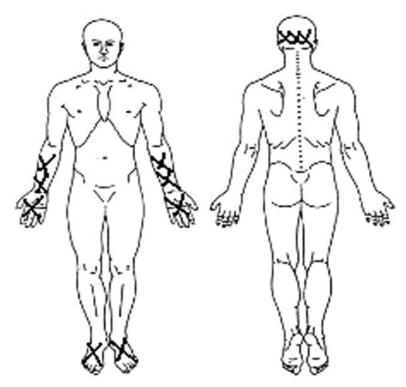


Figure 2 Body chart demonstrating patient's areas of sensory change at initial evaluation. Areas marked with "X" represent numbness or tingling.

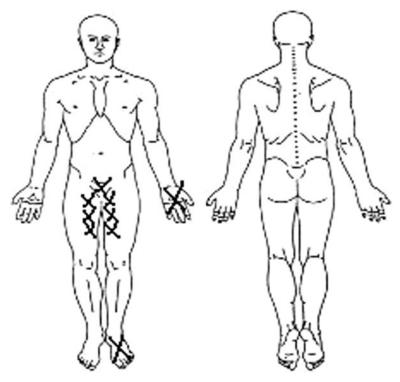


Figure 3 Body chart demonstrating patients areas of sensory change at 12 weeks post-operatively, corresponding to initial complaints of Cauda Equina syndrome. Areas marked with "X" represent numbness or tingling.

function. 21,22 Estimates of incidence range from 2% following acute disc hernations^{23,24} to as low as one per 340 cases of LSS.²⁰

This case will describe the clinical presentation of an individual with TSS. This case is unique due to the severity of the secondary lesion and the significant neurologic compromise. This case also highlights the diagnostic overlap between LSS and CSM, when cauda equina symptoms are present. The purpose of this case is to describe the identification of symptomatic LSS in the presence of CSM and emphasize the need for appropriate testing, recognition and intervention.

Case Description

A 71-year-old male initially presented to physical therapy approximately 4 weeks following a combined anterior/posterior C3/4 fusion with decompression for the treatment of CSM. Pre-operative magnetic resonance imaging (MRI) confirmed myelomalacia, with increased T2 signal of the spinal cord (Fig. 1) representing spinal cord contusion. At the initial physical therapy visit, the patient reported global improvement of his upper extremity numbness and hand function and an improved gait.

Upon physical examination, the patient walked with a slightly stooped and wide based posture, but without loss of balance or evidence of 'sticky feet'. The patient presented with grossly decreased cervical AROM, demonstrating approximately 25% rotation bilaterally, minimal side flexion, and extension to neutral. Flexion was grossly WNL. The patient continued to present with pathologic reflexes bilaterally (Hoffmann's sign,

Table 1 Summary of neurologic tests over course of treatment

Neurologic tests	Hoffmann's sign	Babinski	Clonus	Hyperreflexia	Sensation	Bowel/bladder symptoms
Pre-operative report	Positive bilaterally	Positive bilaterally	Sustained in LE bilaterally	Throughout UE/LE bilaterally	Global c/o UE/LE P&N	Negative
Post-operative initial PT visit	Positive bilaterally	Positive bilaterally	No sustained clonus evident	Throughout UE/LE bilaterally	Global c/o bilateral UE/LE decreased sensation to light touch	Negative
PT visit 11	N/T	Positive bilaterally	None noted	Bilateral LE hyperreflexia UE N/T 2° suspected cauda equina	Slight global paresthesia to light touch bilateral feet	Multiple episodes bowel incontinence
Emergency Department Assessment	N/T	N/T	N/T	Positive LE hyperreflexia Normal cremaster Normal rectal tone	Global decrease bilateral feet	Multiple episodes of bowel incontinence, c/o difficulty with urinary initiation.

Note: LE, lower extremity; PT, physical therapy; UE, upper extremity.

52

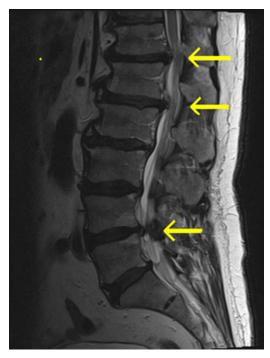


Figure 4 T2 sagital image indicating areas of significant stenosis with increased signal intensity within the distal conus medullaris. Note the change in angulation of the neural structures at the T12-L1 level (uppermost arrow). Arrows indicate involved levels of T12-L1, L1-2, and L4-5.

Babinski, generalized hyper-reflexia), which is typical following surgery for myelopathy. ^{16,25} Significant sensory changes were also present (Fig. 2). Romberg's test was positive, with ability to balance for 5 seconds with the eyes closed. Upper extremity strength was intact and grossly WNL for the C5-T1 myotomes bilaterally. Sensation was decreased to light touch over multiple dermatomes in the hands and lower extremities bilaterally.

Early treatment consisted of cervical active range of motion exercises to address impaired mobility, and endurance focused stabilization training. Stabilization was addressed in this manner due to the post-operative nature of the patient's condition, as endurance deficits have been found to be related to continued pain and disability following cervical fusion surgery. Neck

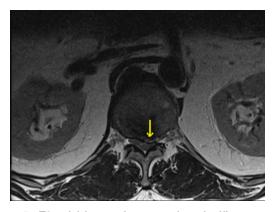


Figure 5 T2 axial image demonstrating significant central canal stenosis at L1-2, with central disc bulge.

muscle endurance deficits have been found to be directly correlated to higher levels of disability.²⁶

Approximately 8 weeks post-operatively (4 weeks/6 visits after initiation of physical therapy) the patient began to complain of weakness and fatigue of the bilateral lower extremity (quadriceps), primarily with stair climbing. As the patient reported that he had just recently resumed this activity, and reported no change in his paresthesias or pain, it was felt that this was a normal residual deficit due to the nature of his initial cervical pathology and surgery. ^{4,7,25} To address this finding, stationary biking was added to the training regime. The patient reported continued, but not progressive, lower extremity weakness over the following 2 weeks of treatment.

The patient returned to therapy again at approximately 12 weeks post-operatively (11 visits) following a period away to care for a sick relative. At this visit, the patient reported daily episodes of lower extremity weakness associated with stair climbing, as well as three episodes of bowel incontinence. Consequently, further questioning was performed that led to the patient revealing a generalized decreased sensation bilaterally in the saddle area (Fig. 3).

Clinical Impression

Due to the clinical presentation of possible CES, the patient's surgeon was notified, as it was the physical therapists impression that the patient required immediate medical attention. The surgeon agreed with the therapist's impression, and it was decided that the patient should be referred to the emergency department to facilitate a same day MRI, which would help to determine the need for surgical decompression. A summary of neurologic testing is presented in Table 1.

Magnetic resonance imaging of the lumbar spine revealed a T12-L1 paracentral disc protrusion causing moderate to severe canal stenosis without foraminal narrowing. Increased signal was seen in the T2 sequence over the distal conus medullaris, representing demyelination (Fig. 4). Additional findings

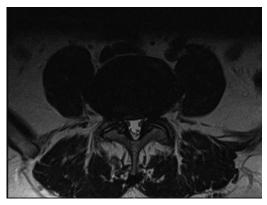


Figure 6 Axial T2 image at the L4-5 level demonstrating central and foraminal stenosis.

NO. 1

included moderate to severe L1-2 canal stenosis (Fig. 5) with T2 hyperintensity of the distal conus medullaris (Fig. 4), and critical stenosis of L4-5 (Fig. 6). The patient underwent lumbar decompressive laminectomy, medial fasciectomy and foraminotomy bilaterally at T12-L1, L1-2, and L4-5 approximately 2 weeks later.

Outcomes

At the first follow-up visit with his surgeon 9 days postoperatively, satisfactory improvement was reported, including resolving groin paresthesias and improved leg strength. At the four month physician follow-up visit, the patient reported complete resolution of lower extremity weakness, normalized lower extremity sensation, and no further episodes of bowel incontinence. There was no further physical therapy intervention during this period.

Discussion

Tandem stenosis is an infrequent and poorly recognized diagnosis with prevalence of 5–25% on imaging, and observed much less frequently, clinically. 1–3,27 This case followed the typical pattern, whereby one area is treated surgically, leading to the recognition of the second symptomatic area. This delayed recognition is due, at least in part, to the overlapping symptoms of lumbar stenosis and/or CES with those of CSM. Early CSM regularly presents with lower extremity weakness, sensory changes, 15 and a flexed gait that may resemble that of lumbar stenosis. 13–15 Lumbar cord compression can also demonstrate comparable symptoms to cervical myelopathy, including motor and sensory changes in the lower extremities in a UMN pattern. 28

The differentiation of sensory changes presents a significant challenge in cases of tandem stenosis. The loss of vibration sense, which has been reported to be the most prominent sensory finding in cervical myelopathy, 13 is also a frequent finding of lumbar stenosis. 19 Sensation to light touch is frequently intact with CSM, and in LSS a deficit is common in a dermatomal distribution. However, the sparing of touch sensation in CSM is not a universal finding, and should be interpreted with caution. A positive Rhomberg test, indicating proprioceptive loss, has been shown to be a frequent finding in patients with LSS and suggested to be diagnostic of the disease. 18,19 This, however, is also a common finding in cervical myelopathy. 13 It has been suggested that in patients with suspected LSS and gait abnormalities, a positive Romberg test should raise the level of suspicion of either CSM or intracranial pathology.²⁹ Adequate screening of the proximal structures, through a comprehensive neurologic assessment, can help clarify this picture.

The absence of low back pain does not rule out the possibility of LSS. In a study of asymptomatic individuals, MRI results revealed that in subjects 60 years old or older, 21% had spinal stenosis.⁶ While

lack of walking tolerance is considered to be the classic complaint in LSS, ¹⁷ neurogenic claudication was present in only 62% of surgical cases. ³⁰ Additionally, objective neurologic findings, such as positive straight leg raise tests and neurologic findings are present in only 50% of cases. ^{17,30} In this case, typical diagnostic aides to CES, such as altered reflexes and lower extremity weakness were not useful. These symptoms, particularly in the absence of significant low back pain, could be attributed to previous cervical myelopathy with longstanding neurologic compromise.

In their review of LSS, Fritz et al. 17 determined that the most predictive variables for the presence of lumbar stenosis were those based on postural elements of symptom reproduction. Relevant to this case, absence of symptoms when sitting (+likelihood ratio 3.1), 17,19 and standing/walking being the worst postures (-likelihood ratio 0.33)^{17,19} could have been considered in the differential diagnosis. In this case, the patient did not complain of pain or difficulty walking, but did complain of difficulty with stair climbing, which is a standing/weight bearing activity. Additionally, rapid relief with sitting was reported; however, the differentiation of this symptom relating to lumbar stenosis did not fit the classic pattern of neurogenic claudication and could be attributed to residual weakness following myelopathy.

In retrospect, the patient's ability to cycle without complaints of difficulty may have served as a subtle indicator of a lumbar lesion. The 'bicycle test' has been suggested as a means of identification of lumbar versus vascular claudication. ^{18,31,32} While not performed in the classic fashion (cycle both flexed and extended comparing time to symptoms), ³¹ the fact that the patient was relatively asymptomatic during flexed, seated cycling rather than during a standing activity (stair climbing) could be considered to be clinically meaningful. It would be expected that LSS would demonstrate a position dependant mechanical behavior, while the neurologic deficits following CSM would show no symptomatic change, despite the mechanical change of position.

The presence of a previous stenosis may serve as an aide in the diagnosis of additional stenosis in other areas of the spine. In a cadaveric study, Lee *et al.*²⁷ found that stenosis in one part of the spine was predictive of stenosis in other areas of the spine 15–32% of the time. Additionally, Houten and Noce³³ reported on a prospective review of the prevalence of cervical myelopathy in patients presenting with isolated low back complaints. They found a positive Hoffmann's sign in 12% of patients presenting with lumbar spine complaints, with bilateral positive findings being highly sensitive for occult cervical cord compression.³³ The prevalence of tandem

stenosis in up to 32% of cadaver specimens,²⁷ up to 25% of individuals on imaging,¹ and signs of cervical cord compression in 12% of patients with isolated lumbar spine complaints³³ should be a cautionary note for the manipulative practitioner.

The existing data suggest that tandem stenosis may occur more frequently than is recognized clinically, and may occur as a congenital narrowing in up to 9% of stenosis cases. 17,34 Experimental studies have shown that in extension, the canal dimension of a normal spine is reduced approximately 9%, while this reduction in the stenotic spine can be up to 67%. 17,35,36 The dramatic reduction in canal diameter may reduce the margin of error for the practitioner. In a study of individuals with spinal cord injury following trauma, those individuals with the largest canals had fewer injuries, and smaller canal diameters resulted in more significant neurologic injury.³⁷ The conclusion was that a larger canal has a protective effect on the spinal cord.³⁷ Therefore, it has been suggested that high velocity manipulative forces may be contraindicated in cases of spinal canal stenosis. 17,38,39 There does, however, exist case evidence of successful manipulative management of patients with LSS utilizing traction and flexion levers (flexion-distraction techniques). 40-42 It is the author's opinion that when providing treatment for a patient with confirmed or suspected stenosis, a complete neurologic evaluation is essential prior to the application of manipulative forces. Extension biased positioning and forces should be avoided in the treatment of patients with suspected or confirmed stenosis.

A significant diagnostic aide in this case was the progression of symptoms beyond baseline despite previous surgical management of CSM. Considering that the normal course following surgery for CSM is a halt of progression, the onset of new bowel symptoms was concerning. It was not until the patient was questioned directly regarding saddle area symptoms that the clinical picture of CES became clear: progressive LE weakness, sensory loss in the saddle area, and bowel incontinence. In any case where bowel and bladder disturbances are present, urgent referral must be considered.²⁸

The decision to facilitate same day imaging was due in large part to the time dependent nature of recovery of neurologic function in the presence of CES. 21,43 Shapiro, 44 in a case series, reported on return of bowel and bladder function following decompression for CES due to disc herniation. All patients undergoing surgery within 48 hours reported return of sphincter function, while all cases reporting continued deficits underwent surgery greater than 48 hours after onset of symptoms. 44 Advanced imaging became the diagnostic modality of choice to confirm the nature of the lesion, due to the diagnostic difficulty involving multiple areas of

neurologic injury. Magnetic resonance imaging was selected due to the high levels of both sensitivity and specificity in the detection of spinal cord compression, ^{15,45} and has been suggested to be the optimal method for non-invasive evaluation of possible CES. ⁴⁶ While surgery was not performed emergently, the performance of surgery promptly following the onset of symptoms of spondylotic CES most likely resulted in resolution of symptoms and a prevention of further neurologic decline or compromise.

Conclusion

The clinical presentation of TSS may be initially subtle, and occur in both surgically and conservatively managed patients. The neurologic findings of tandem stenosis are often confusing, due to the similar findings that occur in the lower extremities early in both CSM and lumbar stenosis. Recognition of CSM, CES, and the possibility of tandem stenosis are crucial skills for the practicing orthopedic physical therapist, who may be the provider of initial contact, particularly for secondary lesions. This recognition of tandem stenosis is of particular importance to the manipulative practitioner, and should be considered in older individuals with symptoms of lumbar stenosis or gait deviations prior to the application of manipulative forces. Early recognition and appropriate referral are the key elements to the management of patients with significant neurologic compromise. This can be achieved through a comprehensive neurologic examination, including screening for UMN signs for all patients, as well as frequent reassessment during the course of treatment.

Acknowledgements

This paper is based on a case study completed in partial fulfillment of the requirements for the DScPT program at Andrews University, Berrien Springs, MI, USA. The author would like to thank Mr Sean Riley, DPT, and Mr Thomas Durant, PT for their assistance in the preparation of this manuscript.

References

- 1 LaBan MM, Green M. Concurrent (tandem) cervical and lumbar spinal stenosis. Am J Phys Med Rehabil 2004;83(3):187–90.
- 2 Dagi TF, Tarkington MA, Leech JJ. Tandem lumbar and cervical spinal stenosis: natural history, prognostic indices, and results after spinal decompression. J Neurosurg 1987;66:842–9.
- 3 Kikuike K, Miyamoto K, Hosoe H, Shimizu K. One-staged combined cervical and lumbar decompression for patients with tandem spinal stenosis on cervical and lumbar spine. J Spinal Disord Tech 2009;22(8):593–601.
- 4 Klineberg E. Cervical spondylotic myelopathy: a review of the evidence. Orthop Clin North Am 2010;41(2):193–202.
- 5 Gore DR, Sepic SB, Gardner GM. Roentgenographic findings of the cervical spine in asymptomatic people. Spine 1986:11:521-4.
- 6 Boden SD, Davis DO, Dina TS, Patronas NS, Wiesel SW Abnormal magnetic resonance scans of the cervical spine in asymptomatic subjects. A prospective investigation. J Bone Joint Surg Am 1990;72:1178–84.
- 7 Tracy JA, Bertelson JD. Cervical Spondylotic Myelopathy. Neurologist 2010;16(3):176–87.

NO. 1

- 8 Bernhardt M, Hynes RA, Blume HW, White AA, 3rd. Cervical spondylotic myelopathy. J Bone Joint Surg Am 1993;75:119–28.
- 9 Ferguson RJL, Caplan LR. Cervical spondylitic myelopathy. Neurol Clin 1985;3:373–82.
- 10 Ogino H, Tada K, Okada K, Yonenobu K, Yamamoto T, Ono K, et al. Canal diameter, anteroposterior compression ratio, and spondylotic myelopathy of the cervical spine. Spine 1983;8:1–15.
- 11 Bartleson JD, Deen HG, editor. Spine disorders: medical and surgical management. New York: Cambridge University Press; 2009.
- 12 Geck MJ, Eismont FJ. Surgical options for treatment of cervical spondylotic myelopathy. Orthop Clin North Am 2002;33(2):329–48.
- 13 Clark CR. Cervical spondylotic myelopathy: history and physical findings. Spine 1988;13:847–9.
- 14 Cook C, Roman M, Stewart K, Leithe LG, Isaacs R. Reliability and diagnostic accuracy of clinical special tests for myelopathy in patients seen for cervical dysfunction. J Orthop Sports Phys Ther 2009;39(3):172–8.
- 15 Cook C, Brown C, Isaacs R, Roman M, Davis S, Richardson W. Clustered clinical findings for diagnosis of cervical spine myelopathy. J Man Manip Ther 2010;18(4):175–80.
- 16 Cheung WY, Arvinte D, Wong YW, Cheung KM. Neurological recovery after surgical decompression in patients with cervical spondylotic myelopathy. Int Orthop 2008;32(2):273–8.
- 17 Fritz JM, Delitto A, Welch WC, Erhard RE. Lumbar spinal stenosis: a review of current concepts in evaluation, management and outcome measures. Arch Phys Med Rehabil 1998;79:700–8.
- 18 Nowakowski P, Delitto A, Erhard R. Lumbar spinal stenosis. Phys Ther 1996;76:187–90.
- 19 Katz JN, Dalgas M, Stucki G, Katz NP, Bayley J, Fossel AH, et al. Degenerative lumbar spinal stenosis: diagnostic value of history and physical examination. Arthritis Rheum 1995;9:1236–41.
- 20 Johnsson KE, Sass M. Cauda equina syndrome in lumbar stenosis. J Spinal Disord Tech 2004;17(4):334–5.
- 21 Fraser S, Roberts L, Murphy E. Cauda equina syndrome: a literature review of its definition and clinical presentation. Arch Phys Med Rehabil 2009;90(11):1964–8.
- 22 Ahn UM, Ahn NU, Buchowski JM, Garrett ES, Sieber AN, Kostuik JP. Cauda equina syndrome secondary to lumbar disc herniation: a meta analysis of surgical outcomes. Spine 2000;25:1515–22.
- 23 Kostuik JP, Harrington I, Alexander D, Rand W, Evans D. Cauda equina and lumbar disc herniation. J Bone Joint Surg Am 1986;68:386–91.
- 24 Shi J, Jia L, Yuan W, Shi G, Ma B, Wang B, *et al.* Clinical classification of cauda equina syndrome for proper treatment. Acta Orthop 2010;81(3):391–5.
- 25 Zhang Y, Shen Y, Wang L, Ding W, Xu J, He J. Magnetic resonance T2 image signal intensity ratio and clinical manifestation predict prognosis after surgical intervention for cervical spondylotic myelopathy. Spine 2010;35(10):E396–9.
- 26 Peolsson A, Kjellman G. Neck muscle endurance in nonspecific patients with neck pain and in patients after anterior cervical

- decompression and fusion. J Manipulative Physiol Ther 2007 Jun;30(5):343-50.
- 27 Lee MJ, Garcia R, Cassinelli EH, Furey C, Riew KD. Tandem stenosis: a cadaveric study in osseous morphology. Spine J 2008;8:1003–6.
- 28 Cook C, Hegedus E, Pietroban R, Goode A. A pragmatic neurologic screen for patients with suspected cord compressive myelopathy. Phys Ther 2007;87(9):1–10.
- 29 Hilibrand AS. Degenerative lumbar stenosis: diagnosis and management. J Am Acad Orthop Surg 1999;7:239–49.
- 30 Turner JA, Ersek M, Herron L, Deyo R. Surgery for lumbar spinal stenosis. Attempted meta-analysis of the literature. Spine 1992;17:1–8.
- 31 Dong GX, Porter RW. Walking and cycling test in neurogenic and intermittent claudication. Spine 1989;14:965–9.
- 32 Dyck P, Doyle JB, Jr. 'Bicycle test' of van Gelderen in diagnosis of intermittent cauda equina compression syndrome. Case report. J Neurosurg 1977;46:667–70.
- 33 Houten JK, Noce LA. Clinical correlations of cervical myelppathy and the Hoffmann sign. J Neurosurg Spine 2008;9:237–42.
- 34 CJM Getty. Lumbar spine stenosis: the clinical significance and results of operation. JBJS Br 1980;62:481–5.
- 35 Sortland O, Magnaes B, Hauge T. Functional myelography with mitrazamide in the diagnosis of lumbar spinal stenosis. Acta Radiol 1977;355(suppl):42S-54S.
- 36 Inufusa A, An H, Lim T, Hasegawa T, Haughton V, Nowicki B. Anatomic changes of the spinal canal and intervertebral foramen associated with flexion-extension movement. Spine 1996;21:2412–20.
- 37 Lintner DM, Knight RQ, Cullen JP. The neurologic sequelae of cervical spine facet injuries: the role of canal diameter. Spine 1993;18:725–9.
- 38 Grieve GP, editor. Moblization of the spine. 4th ed. New York: Curchill Livingstone; 1984.
- 39 Shekelle PG, Coulter I. Cervical spine manipulation: summary report of a systematic review of the literature and a multidisciplinary expert panel. J Spinal Disord 1997;10:223–8.
- 40 Snow GJ. Chiropractic management of a patient with lumbar spinal stenosis. J Manipulative Physiol Ther 2001;24(4):300–4.
- 41 Gay RE, Bronfort G, Evans RL. Distraction manipulation of the lumbar spine: a review of the literature. J Manipulative Physiol Ther 2005;28:266–73.
- 42 DuPriest CM. Nonoperative management of lumbar spinal stenosis. J Manipulative Physiol Ther 1993;16(6):411–4.
- 43 Gleave JR, Macfarlane R. Cauda equina syndrome: what is the relationship between timing of surgery and outcome? Br J Neurosurg 2002;16:325–8.
- 44 Shapiro S. Cauda equina syndrome secondary to lumbar disc herniation. Neurosurgery 1993;32(5):743–37.
- 45 Yin B, Tang Y, Ye J, Wu Y, Wang P. Sensitivity and specificity of in vivo diffusion-weighted MRI in acute spinal cord injury. J Clin Neurosci 2010;17:1173–9.
- 46 Coscia M, Leipzig T, Cooper D. Acute cauda equina syndrome: diagnostic advantage of MRI. Spine 1994;19(4):475–8.

56