

Lumbar spinal stenosis: a brief review of the nonsurgical management

La sténose du canal lombaire: une courte synthèse de la prise en charge non chirurgicale

De Q. H. Tran, MD · Silvia Duong, BScPhm ·
Roderick J. Finlayson, MD

Received: 15 January 2010 / Accepted: 12 April 2010 / Published online: 29 April 2010
© Canadian Anesthesiologists' Society 2010

Abstract

Purpose *The purpose of this brief narrative review is to summarize the evidence derived from randomized controlled trials pertaining to the nonsurgical treatment of lumbar spinal stenosis (LSS).*

Source *The MEDLINE (January 1950 to the fourth week of January 2010) and EMBASE (January 1980 to 2009, week 53) databases, the MESH term “spinal stenosis”, and the key words, “vertebral canal stenosis” and “neurogenic claudication”, were searched. Results were limited to randomized controlled trials (RCTs) conducted on human subjects, written in English, and published in peer-reviewed journals. Only RCTs pertaining to nonsurgical treatment were considered. Studies comparing conservative and surgical management or different surgical techniques were not included in the review.*

Principal findings *The search criteria yielded 13 RCTs. The average enrolment was 54 subjects per study. Blinded assessment and sample size justification were provided in 85% and 39% of RCTs, respectively.*

The available evidence suggests that parenteral calcitonin, but not intranasal calcitonin, can transiently decrease pain in patients with LSS. In the setting of epidural blocks, local anesthetics can improve pain and function, but the benefits seem short-lived. The available evidence does not support the addition of steroids to local anesthetic agents. Based on

the limited evidence, passive physical therapy seems to provide minimal benefits in LSS. The optimal regimen for active physiotherapy remains unknown. Although benefits have been reported with gabapentin, limaprost, methylcobalamin, and epidural adhesiolysis, further trials are required to validate these findings.

Conclusions *Because of their variable quality, published RCTs can provide only limited evidence to formulate recommendations pertaining to the nonsurgical treatment of LSS. In this narrative review, no study was excluded based on factors such as sample size justification, statistical power, blinding, definition of intervention allocation, or clinical outcomes. This aspect may represent a limitation as it may serve to overemphasize evidence derived from “weaker” trials. Further well-designed RCTs are warranted.*

Résumé

Objectif *L'objectif de cette courte synthèse narrative consiste à résumer les données probantes provenant des essais comparatifs randomisés se rapportant au traitement non chirurgical de la sténose du canal lombaire (SCL).*

Source *Les bases de données MEDLINE (de janvier 1950 à la quatrième semaine de janvier 2010) et EMBASE (de janvier 1980 à 2009, semaine 53), le terme MESH « spinal stenosis » et les mots clés « vertebral canal stenosis » et « neurogenic claudication » ont été utilisés. Les résultats ont été limités à des essais comparatifs randomisés (ECR) menés chez des sujets humains, rédigés en anglais et publiés dans des publications évaluées par les pairs. Seuls les ECR se rapportant au traitement non chirurgical ont été considérés. Les études comparant les approches conservatrice et chirurgicale ou les études comparant différentes techniques chirurgicales ont été exclues de cette synthèse.*

D. Q. H. Tran, MD (✉) · R. J. Finlayson, MD
Department of Anesthesia, Montreal General Hospital, McGill
University, 1650 Ave Cedar D10-144, Montreal, QC H3G 1A4,
Canada
e-mail: de_tran@hotmail.com

S. Duong, BScPhm
Faculty of Pharmacy, University of Toronto, Toronto,
ON, Canada

Résultats principaux *Les critères de recherche ont permis d'identifier 13 ECR. Le taux de participation moyen était de 54 sujets par étude. L'évaluation en aveugle et la justification de la taille des échantillons étaient fournies dans 85 % et 39 % des ECR, respectivement.*

Les données probantes disponibles suggèrent que la calcitonine administrée par voie parentérale, et non par voie intranasale, peut réduire de manière transitoire la douleur chez les patients atteints de SCL. Dans le cadre d'une anesthésie épidurale, les anesthésiques locaux peuvent réduire la douleur et améliorer la capacité fonctionnelle, mais leurs bienfaits semblent être de courte durée. Les données probantes disponibles n'appuient pas l'ajout de stéroïdes aux anesthésiques locaux. Il existe des données limitées en faveur d'avantages minimes de la physiothérapie passive en cas de SCL. Le programme optimal de physiothérapie active demeure inconnu. Bien que certains avantages aient été notés lors de l'utilisation de la gabapentine, du limaprost, de la méthylcobalamine et de la lyse d'adhérences épidurales, d'autres essais sont nécessaires afin de valider les résultats.

Conclusions *En raison de leur qualité variable, les ECR publiées ne peuvent offrir qu'une quantité limitée de données probantes en vue de formuler des recommandations se rapportant au traitement non chirurgical d'une SCL. Dans le cadre de cette synthèse narrative, aucune étude n'a été exclue en raison de facteurs tels que la justification de la taille des échantillons, la puissance statistique, l'insu, la définition de l'attribution d'intervention ou les résultats cliniques. Cet aspect peut constituer une limite puisqu'il peut être utilisé pour amplifier l'importance des données provenant d'essais « plus faibles ». D'autres ECR bien conçus sont nécessaires.*

First described more than one hundred years ago,¹ lumbar spinal stenosis (LSS) is characterized by narrowing of the spinal canal with encroachment on neural structures by the surrounding soft tissues and bones.² Although LSS can be congenital, it is more often the result of degenerative phenomena, such as spondylolisthesis and age-related changes (loss of intervertebral disc height, disc bulging, infolding of ligamentum flavum, facet joint osteoarthritis/hypertrophy/osteophyte/cystic formation).³ Lumbar spinal stenosis is the most common indication for back surgery in geriatric patients.^{4,6} In 1994, it was estimated that one billion dollars was spent annually in the United States alone to provide surgical decompression for LSS.⁷ However, in elderly patients, surgery is not devoid of complications.⁴ Thus conservative management is often tried first, and includes physical therapy, pharmacotherapy,

as well as ancillary measures, such as manipulation, bracing, traction, and electrical stimulation.⁸ Epidural injection of steroids can also be used for pain control.⁹

The last five years have seen the publication of ten review articles of variable quality pertaining to LSS.¹⁰⁻¹⁸ In all cases, recommendations stemmed from the combined results of non-randomized as well as randomized controlled trials (RCTs), and scrutiny of the reference lists reveals the omission of RCTs (Appendix). Furthermore, 30% of the available trials were published in the last two years (2008-2009) and, to date, they have not been incorporated in a review article.¹⁹⁻²² Accordingly, using a comprehensive literature search for level 1 evidence (RCTs), we decided to produce a brief and up-to-date narrative review focusing exclusively on the nonsurgical management of LSS.

Search strategy and article selection criteria

The literature search for this review was conducted during the fourth week of January 2010, using the MEDLINE (January 1950 to the fourth week of January 2010) and EMBASE (January 1980 to 2009, week 53) databases.

The MESH term, “spinal stenosis”, as well as the key words, “vertebral canal stenosis” and “neurogenic claudication”, were searched. Results were limited to RCTs pertaining to nonsurgical treatment conducted on human subjects, written in English, and published in peer-reviewed journals. We excluded trials that investigated the impact of interventions on parameters measured *in vitro* (nerve root blood flow) without assessing the clinical response of patients. Randomized controlled trials published in the form of abstracts or correspondence were also discarded. Furthermore, the RCTs needed to deal exclusively with LSS. We excluded trials that enrolled patients suffering from LSS and other spine pathologies (such as radiculopathy due to disc herniation) and that indiscriminately pooled results from all subjects. However, studies providing data specific to LSS were considered in this review. For trials that included both a randomized cohort and a concurrent observational cohort of patients who declined to undergo randomization, only results pertaining to the former were kept. Studies comparing conservative and surgical management or different surgical techniques were not included in the review.

After selecting the initial articles, we examined the reference lists as well as our personal files for additional material. No RCTs were excluded based on factors such as definition of intervention allocation or primary and secondary (clinical) outcomes. However, non-randomized studies, observational case reports, and cohort studies were

excluded to avoid potential biases introduced by institutional practices.

Findings

Our initial search criteria yielded 14 RCTs. One RCT was excluded because the authors did not conduct statistical tests to compare the results.²³ Six of the remaining 13 RCTs studied pharmacological treatment (Table 1), and five investigated neuraxial blocks or epiduroscopic adhesiolysis (Table 2). Physical therapy was addressed by three

studies (Table 3). As per this classification, the sum of these RCTs (14) exceeds the total found (13), because one trial compared both epidural blocks and physiotherapy to control treatment.²²

Overall, the quality of the RCTs was variable. The average enrolment was 54 subjects per study. Blinded assessment and sample size justification were provided in 85% and 39% of RCTs, respectively. The duration of symptomatic LSS prior to enrolment was provided in 77% of studies and varied from 12.0 weeks to 11.4 years. Pain was the most commonly studied endpoint (69% of trials); patient follow-up varied from 4.0 weeks to 2.5 years.

Table 1 Pharmacological treatment trials related to lumbar spinal stenosis

Authors (year)	Blinded Assessment/ Sample Size justification	Description	Number of Patients/ Groups	Primary Outcomes and Duration of Follow-Up	Main Findings
Eskola <i>et al.</i> ²⁵ (1992)	Y/N	SC calcitonin (100 units) vs placebo every other day for 4 wks	39/2	Pain (VAS), walking distance, jumping time until 12 wks after start of treatment	Compared with baseline, greater decreases in static and dynamic pain scores in calcitonin for duration of 3 months No differences in walking distance and jumping times
Podichetty <i>et al.</i> ²⁶ (2004)	Y/N	Crossover after 2-month washout period Daily nasal calcitonin (400 units) vs placebo for 6 wks	47/2	Pain (VAS), walking distance, walking time, physical and emotional functional assessment (SF-36) until 6 wks after start of treatment	No differences
Tafazal <i>et al.</i> ²⁷ (2007)	Y/N	Daily nasal calcitonin (200 units) vs placebo for 3 wks	37/2	Pain (VAS), walking distance, ODI, LBOS until 4 wks after start of treatment	No differences
Yaksi <i>et al.</i> ³¹ (2007)	N/N	4-month course of gabapentin (starting daily dose of 900 mg; weekly increments of 300 mg until maximum of 2,400 mg) combined with conservative management (physical therapy, corset, NSAIDs) vs conservative management alone	55/2	Pain (VAS), walking distance, sensory deficits, motor deficits until 4 months after start of treatment	Gabapentin: greater walking distance (2 nd , 3 rd , and 4 th months), lower pain scores (3 rd and 4 th months), and lower incidence of sensory deficits (4 th month)
Matsudaira <i>et al.</i> ²¹ (2009)	N/N	Limaprost (15 µg <i>po tid</i>) vs etodolac (400 mg <i>po bid</i>) for 8 wks	66/2	SF-36 at 8 wks after start of treatment	Limaprost: greater improvements in SF-36 subscales of physical function, role physical, bodily pain, vitality, and mental health Limaprost: greater walking distance, subjective improvement, satisfaction, and improvement in leg numbness
Waikakul <i>et al.</i> ³⁵ (2000)	Y/N	6-month course of methylcobalamin (0.5 mg <i>po tid</i>) vs control	152/2	Pain, limitations of spinal motion, walking distance, neurological exam (sensorimotor functions, DTR, SLR) until 2 yrs after start of treatment	Methylcobalamin: greater walking distance at 6, 12, and 18 months No other intergroup differences

bid = *bis in die* (twice daily); DTR = deep tendon reflexes; LBOS = Low Back Outcome Score; N = no; NSAIDs = non-steroidal anti-inflammatory drugs; ODI = Oswestry Disability Index; *po* = per os; SC = subcutaneous; SF-36 = Medical Outcomes Study Short Form-36; SLR = straight leg raise test; *tid* = *ter in die* (three times a day); VAS = visual analogue scale; wk = week; Y = yes

Table 2 Trials pertaining to epidural block and adhesiolysis for lumbar spinal stenosis

Authors (year)	Blinded Assessment/ Sample Size justification	Description	Number of Patients/ Groups	Primary Outcomes/ Duration of Follow-Up	Main Findings
Fukusaki <i>et al.</i> ³⁷ (1998)	Y/N	Series of 2 non fluoroscopy-guided epidural injections: NS vs 1% M 8 mL vs 1% M 8 mL and methylprednisolone 40 mg	53/3	Walking distance until 3 months after treatment	Both treatment groups: greater walking distance at 1 wk No intergroup differences at 1 and 3 months No differences between 2 treatment groups
Koc <i>et al.</i> ²² (2009)	Y/N	Control vs physiotherapy (2-wk course) vs fluoroscopy-guided interlaminar epidural (0.5% B 15 mg and triamcinolone 60 mg)	29/3	Pain (VAS), walk test, sit-to-stand test, weight-carrying test, RMDI, NHP until 6 months after treatment	Epidural: greater improvement in VAS, RMDI, and NHP than control at 2 wks No differences between 2 treatment groups
Cuckler <i>et al.</i> ⁴⁵ (1985)	Y/Y	Interlaminar epidural with 1 % P 5 mL and NS vs methylprednisolone 80 mg	37/2	Success (75 % improvement in pain compared with baseline) until 13-30 months after treatment	No difference in short term (24 h) and long term (13-30 months) success
Manchikanti <i>et al.</i> ¹⁹ (2008)	Y/Y	Fluoroscopy-guided caudal injection: 0.5% L 10 mL vs 0.5% L 9 mL and betamethasone 6 mg (injections could be repeated based on clinical response)	40/2 (Preliminary results of ongoing trial aiming to recruit total of 120 patients)	Pain (NRS) until 1 yr after treatment	No intergroup differences in NRS, ODI, employment status, opioid intake at 3, 6, and 12 months
Manchikanti <i>et al.</i> ²⁰ (2009)	Y/Y	Fluoroscopy-guided caudal (catheter threaded to S3 and injected with 5 mL 2%, 6 mL NS and 6 mg betamethasone) vs adhesiolysis (catheter threaded to level of defect, adhesiolysis performed and catheter injected with 2% 5 mL, 10% 6 mL sodium chloride solution and betamethasone 6 mg) (treatments could be repeated based on clinical response)	50/2 (Preliminary results of ongoing trial aiming to recruit total of 120 patients)	Pain (NRS) until 1 yr after treatment	Adhesiolysis: lower NRS and ODI at 3, 6, and 12 months No differences in employment status and opioid intake

B = bupivacaine; L = lidocaine; M = mepivacaine; N = no; NHP = Nottingham Health Profile; NRS = Numeric Rating Scale; NS = normal saline; ODI = Oswestry Disability Index; P = procaine; RMDI = Roland Morris Disability Index; wk = week; Y = yes; Yr = year

Pharmacological therapy

Calcitonin

Calcitonin has received considerable interest in the management of LSS because of its direct analgesic properties (through release of β -endorphin).²⁴ Alternately, calcitonin can decrease the vascular supply to the bone by lowering its metabolic activity, thus allowing more blood to reach the compromised neural tissues.²⁵ To date, four RCTs have investigated the use of calcitonin in LSS.

In 1992, Eskola *et al.*²⁵ conducted a double-blind, crossover RCT on 39 patients suffering from LSS. The subjects initially received a subcutaneous injection of

calcitonin or placebo every other day for four weeks. After a two-month washout period, the alternate solution was administered for another four weeks. When comparing with baseline levels, these authors observed that statistically greater decreases in static and dynamic pain scores were seen in the calcitonin group for up to three months irrespective of the order of administration. However, increases in walking distance and jumping time did not survive the crossover. Furthermore, at one year, none of the two groups experienced residual benefits.²⁵ In 2004, Podichetty *et al.*²⁶ recruited 47 patients and compared a six-week regimen of intranasal calcitonin with placebo. At six weeks, no differences in pain, walking distance, walking time, and physical or emotional function were found between the

Table 3 Trials pertaining to physical therapy for lumbar spinal stenosis

Authors (year)	Blinded Assessment/ Sample Size justification	Description	Number of Patients/ Groups	Primary Outcome and Duration of Follow-Up	Main Findings
Koc <i>et al.</i> ²² (2009)	Y/N	Control vs PT (2-wk course of hot pack, TENS, and US application) vs fluoroscopy-guided interlaminar epidural (B and triamcinolone)	29/3	Pain (VAS), walk test, sit-to-stand test, weight-carrying test, RMDI, NHP until 6 months after treatment	PT: no differences compared with control Epidural: greater improvement in VAS, RMDI, and NHP than in Control at 2 wks
Whitman <i>et al.</i> ⁴⁶ (2006)	Y/Y	Twice weekly, 6-wk regimen: lumbar flexion exercise/ walking program/ US (Gp I) vs manual therapy/ exercise/ walking program (Gp II)	55/2	GRC until 1 yr after start of treatment	Gp II: higher perceived recovery (GRC \geq 3) at 6 wks No difference in perceived recovery at 1 yr No differences in ODI, NPRS, SSS Satisfaction Subscale, walking distance
Pua <i>et al.</i> ⁴⁷ (2007)	Y/Y	Twice weekly, 6-wk regimen: cycling vs treadmill ambulation with body weight support	42/3	ODI until 6 wks after start of treatment	No differences in ODI, RMDI, VAS, perceived benefit and ability to walk \geq 800 m

B = bupivacaine; Gp = group; GRC = global rating of change; N = no; NHP = Nottingham Health Profile; NPRS = numeric pain rating scale; ODI = Oswestry Disability Index; PT = physiotherapy; RMDI = Roland Morris Disability Index; SSS = spinal stenosis scale; TENS = transcutaneous electrical nerve stimulation; US = ultrasound; VAS = visual analogue scale; wk = week

treatment and control groups. Subsequently, in 37 subjects with LSS, Tafazal *et al.*²⁷ compared a four-week regimen of intranasal calcitonin with placebo. Again, no intergroup differences were noted in terms of pain and walking distance. The Oswestry Disability Index (ODI) and Low Back Outcome Score were also similar between the two groups.

Gabapentin

Voltage-sensitive calcium and sodium channels accumulate at sites of axonal injury.²⁸ By binding to the α_2 delta subunit of these channels, gabapentin has been thought to modulate neural transmission and provide analgesia.^{29,30}

In 2007, Yaksi *et al.*³¹ randomized 55 patients with LSS to a four-month regimen of gabapentin combined with conservative management or conservative management alone. The latter included physical therapy (lumbar flexion, pelvic traction, and strengthening of abdominal muscles), the use of a lumbosacral corset, and pharmacological treatment with non-steroidal anti-inflammatory drugs (NSAIDs). Throughout the trial, these authors observed a greater walking distance in the gabapentin group. Furthermore, pain scores were also significantly lower in the latter at the end of the third and fourth months (3.6 ± 2.2 vs 4.8 ± 2.2 ; $P = 0.039$; and 2.9 ± 2.6 vs 4.7 ± 2.2 , respectively; $P = 0.006$). After four months, a greater

reduction in sensory deficits was seen with gabapentin compared with conservative management (decrease of 28.6% vs 7.4%, respectively; $P = 0.04$).³¹

Limaprost

The pathogenesis of LSS may be multifactorial. Mechanical compression of the spinal cord can lead to a decrease in the vascular supply of neural tissues.³² Limaprost, an alprostadil (prostaglandin E1) analogue, possesses vasodilatory, antiplatelet, and cytoprotective properties.³³

In 66 patients, Matsudaira *et al.*²¹ compared limaprost with etidolac, a NSAID. After eight weeks, subjects receiving limaprost displayed better scores pertaining to Standard Form-36 (SF-36) subscales of physical function, physical role, bodily pain, vitality, and mental health. Furthermore, greater improvements were also noted in terms of walking distance, leg numbness, and patient satisfaction. However, the two groups did not differ in terms of low back and leg pain.²¹

Methylcobalamin

Since high doses of vitamins, such as B6, have been used in nerve entrapment syndromes,³⁴ Waikakul *et al.*³⁵ set out to investigate the role of methylcobalamin, a methyl-vitamin

B12, in LSS. These authors randomized 152 patients to a six-month regimen of methylcobalamin or control. All subjects also received patient education, core strengthening exercises, physiotherapy, oral analgesics, NSAIDs, muscle relaxants, and supplemental vitamins. During the entire study period (two years), there were no intergroup differences observed in terms of pain, limitations of motion, straight leg raise test, or neurological findings. However, patients receiving methylcobalamin experienced greater improvement in ambulation at six, 12, and 18 months.³⁵

Interpretation

The available evidence suggests that parenteral calcitonin, but not intranasal calcitonin, can lead to transient benefits (\leq three months) in patients with LSS. Although gabapentin, limaprost, and methylcobalamin have been shown to improve parameters, such as analgesia, walking distance, or sensory deficits, further trials are required to validate these findings because of the small number of RCTs involved.

Epidural blockade and adhesiolysis

Epidural blockade

In LSS, pain may be due to transient ischemia of the cauda equine.³⁶ Thus, epidural injection of local anesthetics is commonly used to provide sympathetic blockade and vasodilation, thereby increasing blood flow to neural tissues.³⁷ Furthermore local anesthetic agents can also exert beneficial effects by curtailing pain-induced neuronal sensitization and release of neurotransmitters involved in pain pathways.³⁸⁻⁴¹ Alternately, administration of corticosteroids in the epidural space is thought to reduce inflammatory edema of the injured nerve root,⁴² decrease sensitization of the dorsal horn neurons,⁴³ and suppress the transmission of nociceptive C fibres.⁴⁴ In clinical practice, both agents are often combined.³⁷

To date, two RCTs have compared epidural injection of local anesthetics with placebo or conservative management (NSAIDs, physiotherapy). In 53 patients suffering from LSS, Fukusaki *et al.*³⁷ performed a series of two non fluoroscopy-guided interlaminar epidural injections and randomized the injectate to saline, local anesthetic, and local anesthetic with steroid (methylprednisolone). Compared with placebo, these authors observed a greater walking distance at one week in both treatment groups ($87-92 \pm 58-66$ vs 23 ± 19 m; $P < 0.05$). However, this beneficial effect did not persist, as no differences were found at one and three months. Interestingly, steroids did not add any clinical benefits.³⁷ In 2009, Koc *et al.*²²

randomized 29 subjects with LSS to control, physiotherapy (two-week course of hot pack, transcutaneous electrical nerve stimulation, and ultrasound application), or fluoroscopy-guided interlaminar epidural injection (bupivacaine and triamcinolone). In addition, all patients received a six-month home-based exercise program of muscle stretching/ strengthening and a two-week course of diclofenac. Intergroup analysis revealed that, compared with controls, epidural injection resulted in a greater improvement in pain intensity, Roland Morris Disability Index, and Nottingham Health Profile at two weeks.²²

In addition to Fukusaki *et al.*'s study,³⁷ two other RCTs have investigated the role of steroids in epidural blockade. In 1985, Cuckler *et al.*⁴⁵ randomized 37 patients to an epidural injection containing procaine combined with saline or methylprednisolone. Success was defined as a 75% improvement compared with baseline. No difference in success rates was found between the two groups at the 24 hr (17.7-25.0%) and the 13-30 month follow-ups (10.5-25%). In 2007, Manchikanti *et al.* set out to recruit 120 subjects with LSS. The intended goal was to compare fluoroscopy-guided caudal injection of local anesthetic (lidocaine) with or without corticosteroid (betamethasone). In 2008, these authors published their preliminary findings in 40 patients (20 per group). Manchikanti *et al.*¹⁹ observed that the addition of betamethasone to lidocaine did not improve analgesia, employment status, ODI scores, and opioid intake after three, six, and twelve months.

Epidural adhesiolysis

In 2006, Manchikanti *et al.* set out to recruit 120 subjects with LSS. The intended goal was to compare fluoroscopy-guided caudal injection of lidocaine and betamethasone with percutaneous epidural adhesiolysis. In 2009, these authors published their preliminary findings in 50 patients (25 per group). Manchikanti *et al.*²⁰ observed that patients receiving adhesiolysis exhibited lower scores for pain and ODI at three, six, and twelve months. Furthermore, after adhesiolysis, the average duration of relief was also longer for patients with back pain (12.3 ± 10.9 vs 3.2 ± 3.7 weeks; $P < 0.05$) and leg pain (12.5 ± 11.0 vs 3.1 ± 3.8 weeks; $P < 0.05$). However, no intergroup differences in opioid intake and employment status were found.

Interpretation

Although epidural injection of local anesthetics has been shown to improve pain and function in LSS, these benefits seem short-lived ($<$ one month). The available evidence does not support the addition of steroids to local anesthetic agents. Despite promising early results, further studies are required to validate the use of epidural adhesiolysis in LSS.

Physical therapy

Passive physical therapy has been studied in one trial. Koc *et al.*²² randomized 29 subjects with LSS to control, physiotherapy (two-week course of hot pack, transcutaneous electrical nerve stimulation, and ultrasound application), or epidural injection (bupivacaine and triamcinolone). In addition, all patients received a six-month home-based exercise program of muscle stretching and strengthening as well as a two-week course of diclofenac. No difference was found between the physiotherapy and control groups. In contrast, as previously stated, compared with controls, epidural injection resulted in a greater improvement in pain intensity, Roland Morris Disability Index, and Nottingham Health Profile at two weeks.²²

To date, two RCTs have attempted to determine the optimal regimen for (active) physical therapy. In 2006, Whitman *et al.*⁴⁶ randomized 55 patients with LSS to two six-week physiotherapy regimens. The first group (Group I) received lumbar flexion exercises, a progressive treadmill ambulation program, and subtherapeutic pulsed ultrasound. The second group (Group II) received manual physical therapy (spine, pelvis, and lower extremities), exercises (designed to improve mobility, strength and coordination), and a body weight-supported treadmill ambulation program. In addition, all subjects received a home exercise program, and they were asked to take a daily walk. Whitman *et al.*⁴⁶ defined perceived recovery as a global rating of change score ≥ 3 . At six weeks, Group II presented a higher rate of perceived recovery (79 vs 41% of patients; $P = 0.0015$). However, no statistical differences were found at one year and on long-term telephone follow-up (27.4–29.0 months). Furthermore, throughout the study period, the authors reported no intergroup differences in terms of pain, walking distance, ODI, and Spinal Stenosis Scale Satisfaction Subscale scores.⁴⁶ In 2007, Pua *et al.*⁴⁷ randomized 42 patients to a six-week physiotherapy regimen of cycling or treadmill ambulation with body weight support. All subjects also received a home exercise program of flexion/ neural mobilization exercises. At three and six weeks, no differences were found in terms of disability (ODI, Roland Morris Disability Index) and pain.⁴⁷

Interpretation

The limited evidence available suggests that passive physical therapy provides minimal benefits. The optimal regimen for active physiotherapy remains unknown. Although the combination of manual therapy/ exercise/ body weight-supported ambulation results in a higher rate of perceived recovery than the combination of flexion

exercise/ progressive treadmill ambulation/ ultrasound, these benefits did not persist beyond six weeks and did not translate into an improvement of objective indices. Furthermore, no differences were found between cycling and treadmill ambulation with body weight support.

Discussion

In terms of pharmacological treatment, the available evidence suggests that parenteral calcitonin, but not intranasal calcitonin, can transiently decrease pain in patients with LSS. Although benefits have been reported with gabapentin, limaprost, and methylcobalamin, further trials are required to validate these findings. In the setting of epidural blocks, local anesthetic agents can improve pain and function, but the benefits seem short-lived. The available evidence does not support the addition of steroids to local anesthetics. Despite promising results, further studies are required to validate the use of epidural adhesiolysis for patients with LSS. Based on the limited evidence available, passive physical therapy seems to provide minimal benefits in LSS. The optimal regimen for active physiotherapy remains unknown.

A critical survey of the available RCTs can provide an effective tool to establish focused recommendations pertaining to the nonsurgical treatment of LSS. For instance, in this review, no evidence was found to support the use of epidural steroid injection (ESI). This is stark contrast with recently published review articles. Using the pooled results of case series, observational trials and RCTs, 70% of the latter recommended ESI. Furthermore, despite the unclear benefits and limited efficacy associated with active and passive physiotherapy, respectively, 90% of recent reviews advocated the use of physiotherapy. Alarming, a device that can be potentially harmful (through muscle deconditioning), such as a stabilization brace, was recommended for occasional use by 40% of reviews despite the absence of RCTs (Appendix). Since our review article incorporated RCTs missing from previous publications, new and promising therapeutic modalities (limaprost, methylcobalamin, and epidural adhesiolysis) have also been identified. Although the limited evidence available does not permit their clinical implementation at this time, further investigation is certainly warranted.

For practical reasons, a decision was taken to limit this review to RCTs published in the English language. Although such a restriction may constitute a methodological limitation, we believe that its impact on the paper's conclusions is small, since expansion of our search criteria (using the same databases and time periods) to languages

Table 4 Nonsurgical management strategies warranting further clinical investigation

Pharmacotherapy	<ul style="list-style-type: none"> • Efficacy of acetaminophen, NSAIDs, selective COX 2 inhibitors, and opioids • Confirmatory trials for gabapentin, methylcobalamin, and limaprost • Pregabalin vs gabapentin
Physical therapy	<ul style="list-style-type: none"> • Optimal regimen for active physical therapy
Interventional Treatment	<ul style="list-style-type: none"> • Palpation- vs fluoroscopy-guided interlaminar epidural injection of LA • Fluoroscopy-guided interlaminar vs transforaminal vs caudal epidural injection of LA • Confirmatory trials for epiduroscopic adhesiolysis
Multimodal Treatment	<ul style="list-style-type: none"> • Efficacy of multimodal treatment • Best combination for multimodal management

COX = cyclooxygenase; LA = local anesthetic agent; NSAID = non-steroidal anti-inflammatory drug

other than English yielded only three additional RCTs.⁴⁸⁻⁵⁰ No attempt was made in this review to produce a meta-analysis. In our view, given the wide array of modalities used for pharmacological and physical therapy, patient enrolment would have been insufficient to support a systematic pooling of data. For interventional treatments, the heterogeneity in techniques (palpation- vs fluoroscopy-guided epidural block vs adhesiolysis) would have constituted an obstacle. In this narrative review, no RCT was excluded based on factors such as sample size justification, statistical power, blinding, definition of intervention allocation, or clinical outcomes. This may represent a limitation to our article, as it may serve to overemphasize evidence derived from “weaker” RCTs. Most importantly, if trials lacked sample size justification, provided limited enrolment, and found no difference between study groups, we cannot exclude the possibility that they were inadequately powered to answer the question they sought to investigate.

Despite current best evidence, many issues regarding nonsurgical modalities remain unresolved and, thus, require elucidation through well-designed and meticulously conducted RCTs (Table 4). Future trials should use sample size justification and blinded assessment. Furthermore, the duration of LSS prior to enrolment and the length of follow-up should be rigorously controlled. For trials investigating interventional treatment, evidence-based standardized conservative management should be implemented in the control group. Lastly, most studies have focused thus far on single or dual therapeutic modalities—the role of multimodal therapy warrants investigation.

Appendix

See Table 5

Table 5 Recently published reviews of lumbar spinal stenosis

Authors (reference number) (year)	Focused on Treatment	Restricted to RCTs	Inclusion of All Contemporary RCTs* Pertaining to Nonsurgical Treatment	Contemporary RCTs Pertaining to Nonsurgical Treatment Not Included in Review (reference numbers)	Recommended Nonsurgical Treatment
Katz <i>et al.</i> ² (2008)	N	N	N	25, 26, 27, 31, 35, 46, 47	Stabilization brace (occasional), PT (core-strengthening exercises), pharmacotherapy (acetaminophen, NSAIS, mild opioid), ESI
Kim <i>et al.</i> ¹⁰ (2005)	N	N	N	25, 26, 35, 37, 45	Rest, restricted movement, stabilization brace (occasional), PT (massage, heat, cold, US, TENS, core-strengthening exercises), acupuncture, exercises (bike, treadmill, aquatic ambulation), balance training, patient education, biofeedback, relaxation training, pharmacotherapy (acetaminophen, aspirin, NSAID, tramadol, opioid, muscle relaxant, antidepressant, anticonvulsant), ESI

Table 5 continued

Authors (reference number) (year)	Focused on Treatment	Restricted to RCTs	Inclusion of All Contemporary RCTs* Pertaining to Nonsurgical Treatment	Contemporary RCTs Pertaining to Nonsurgical Treatment Not Included in Review (reference numbers)	Recommended Nonsurgical Treatment
Yuan <i>et al.</i> ¹¹ (2005)	Y	N	N	26, 35, 37	Rest, PT (flexion-based exercises), aquatic training, exercise, patient education (posture, ADL), pharmacotherapy (aspirin, NSAID, muscle relaxant, antidepressant, oral corticosteroid, calcitonin), ESI
Atlas <i>et al.</i> ¹² (2006)	Y	N	N	25, 35, 37, 45	PT, exercise, patient education, analgesics
Babb <i>et al.</i> ¹³ (2006)	N	N	N	25, 26, 35, 37, 45	Stabilization brace (occasional), PT, weight loss program (if overweight), exercises (bicycle, aquatic therapy, inclined treadmill), pharmacotherapy (NSAID, oral steroid), ESI
Englund ¹⁴ (2007)	N	N	N	25, 35, 37, 46	PT (core-strengthening exercises), exercises (bicycle, aquatic therapy, inclined treadmill), pharmacotherapy (acetaminophen, NSAID, anticonvulsant, oral corticosteroid)
Chad ¹⁵ (2007)	N	N	N	25, 26, 35, 37, 45, 46	Stabilization brace (occasional), PT (back/ leg lengthening exercises, US, TENS), weight loss program (if overweight), exercises (bicycle, walking), pharmacotherapy (NSAID, muscle relaxant), ESI
Markman <i>et al.</i> ¹⁶ (2008)	N	N	N	25, 27, 31, 35, 37, 45, 46, 47	No clear recommendation
Rahman <i>et al.</i> ¹⁷ (2008)	N	N	N	25, 27, 31, 35, 37, 45, 46, 47	PT (back/ leg lengthening exercises, mobility training, US, TENS), pharmacotherapy (NSAID, muscle relaxant), ESI
Aliabadi <i>et al.</i> ¹⁸ (2009)	N	N	N	19, 25, 26, 27, 31, 35, 45, 47	PT (manual PT, body weight-supported treadmill). No clear recommendation pertaining to ESI

ADL = activities of daily living; ESI = epidural steroid injection; N = no; NSAID = non-steroidal anti-inflammatory drug; PT = physiotherapy; RCT = randomized controlled trial; TENS = transcutaneous electrical nerve stimulation; US = ultrasound; Y = yes

* A contemporary RCT is defined as a RCT published no later than the year prior to the publication of the review article

References

1. Sachs B, Frankel V. Progressive and kyphotic rigidity of the spine. *J Nerv Ment Dis* 1900; 27: 1.
2. Katz JN, Harris MB. Clinical practice. Lumbar spinal stenosis. *N Engl J Med* 2008; 358: 818-25.
3. Arnoldi CC, Brodsky AE, Cauchoix J, et al. Lumbar spinal stenosis and nerve root entrapment syndromes. Definition and classification. *Clin Orthop Relat Res* 1976; 115: 4-5.
4. Deyo RA, Ciol MA, Cherkin DC, Loeser JD, Bigos SJ. Lumbar spinal fusion. A cohort study of complications, reoperations, and resource use in the Medicare population. *Spine (Phila Pa 1976)* 1993; 18: 1463-70
5. Deyo RA, Gray DT, Kreuler W, Mirza S, Martin BI. United States trends in lumbar fusion surgery for degenerative conditions. *Spine (Phila Pa 1976)* 2005; 30: 1441-5
6. Ciol MA, Deyo RA, Howell E, Kreif S. An assessment of surgery for spinal stenosis: time trends, geographic variations, complications, and reoperations. *J Am Geriatr Soc* 1996; 44: 285-90.
7. Taylor VM, Deyo RA, Cherkin DC, Kreuter W. Low back pain hospitalization. Recent United States trends and regional variations. *Spine (Phila Pa 1976)* 1994; 19: 1207-13
8. Watters WC 3rd, Baisden J, Gilbert TJ, et al. Degenerative lumbar spinal stenosis: an evidence-based clinical guideline for the diagnosis and treatment of degenerative lumbar spinal stenosis. *Spine J* 2008; 8: 305-10.
9. Friedly J, Chan L, Deyo R. Increases in lumbosacral injections in the Medicare population. *Spine (Phila Pa 1976)* 2007; 32: 1754-60
10. Kim SL, Lim RD. Spinal stenosis. *Dis Mon* 2005; 51: 6-17.
11. Yuan PS, Booth RE Jr, Albert TJ. Nonsurgical and surgical management of lumbar spinal stenosis. *Instr Course Lect* 2005; 54: 303-12.

12. Atlas SJ, Delitto A. Spinal stenosis: surgical versus nonsurgical treatment. *Clin Orthop Relat Res* 2006; 443: 198-207.
13. Babb A, Carlson WO. Spinal stenosis. *S D Med* 2006; 59: 103-5.
14. Englund J. Lumbar spinal stenosis. *Curr Sports Med Rep* 2007; 6: 50-5.
15. Chad DA. Lumbar spinal stenosis. *Neurol Clin* 2007; 25: 407-18.
16. Markman JD, Gaud KG. Lumbar spinal stenosis in older adults: current understanding and future directions. *Clin Geriatr Med* 2008; 24: 369-88.
17. Rahman RK, Nowak DD, Gelb DE, Poelstra KA, Ludwig SC. Lumbar spinal stenosis. *Current Orthopaedic Practice* 2008; 19: 351-6.
18. Aliabadi H, Isaacs R. Lumbar spinal stenosis: a brief review. *Neurosurgery Quarterly* 2009; 19: 200-6.
19. Manchikanti L, Cash KA, McManus CD, Pampati V, Abdi S. Preliminary results of a randomized, equivalence trial of fluoroscopic caudal epidural injections in managing chronic low back pain: Part 4—Spinal stenosis. *Pain Physician* 2008; 11: 833-48.
20. Manchikanti L, Cash KA, McManus CD, Pampati V, Singh V, Benjamin R. The preliminary results of a comparative effectiveness evaluation of adhesiolysis and caudal epidural injections in managing chronic low back pain secondary to spinal stenosis: a randomized, equivalence controlled trial. *Pain Physician* 2009; 12: E341-54.
21. Matsudaira K, Seichi A, Kunogi J, et al. The efficacy of prostaglandin E1 derivative in patients with lumbar spinal stenosis. *Spine (Phila Pa 1976)* 2009; 34: 115-20.
22. Koc Z, Ozcakir S, Sivrioglu K, Gurbet A, Kurcukoglu S. Effectiveness of physical therapy and epidural steroid injections in lumbar spinal stenosis. *Spine (Phila Pa 1976)* 2009; 34: 985-9.
23. Porter RW, Miller CG. Neurogenic claudication and root claudication treated with calcitonin. A double-blind trial. *Spine (Phila Pa 1976)* 1988; 13: 1061-4.
24. Fraioli F, Fabri A, Gnessi L, Moretti C, Santoro C, Felici M. Subarachnoid injection of salmon calcitonin induces analgesia in man. *Eur J Pharmacol* 1982; 78: 381-2.
25. Eskola A, Pohjolainen T, Alaranta H, Soini J, Tallroth K, Slati P. Calcitonin treatment in lumbar spinal stenosis: a randomized, placebo-controlled, double-blind, cross-over study with one-year follow-up. *Calcif Tissue Int* 1992; 50: 400-3.
26. Podichetty VK, Segal AM, Lieber M, Mazanec DJ. Effectiveness of salmon calcitonin nasal spray in the treatment of lumbar canal stenosis: a double-blind, randomized, placebo-controlled, parallel group trial. *Spine (Phila Pa 1976)* 2004; 29: 2343-9.
27. Tafazal SI, Ng L, Sell P. Randomised placebo-controlled trial on the effectiveness of nasal salmon calcitonin in the treatment of lumbar spinal stenosis. *Eur Spine J* 2007; 16: 207-12.
28. Moll C, Mourre C, Lazdunski M, Ulrich J. Increase of sodium channels in demyelinated lesions of multiple sclerosis. *Brain Res* 1991; 556: 311-6.
29. Cheng JK, Chiou LC. Mechanisms of the antinociceptive action of gabapentin. *J Pharmacol Sci* 2006; 100: 471-86.
30. Serpell MG, Neuropathic Pain Study Group. Gabapentin in neuropathic pain syndromes: a randomised, double blind, placebo-controlled trial. *Pain* 2002; 99: 557-66.
31. Yaksi A, Ozgonenel L, Ozgonenel B. The efficiency of gabapentin therapy in patients with lumbar spinal stenosis. *Spine* 2007; 32: 939-42.
32. Delamarter RB, Bohlman HH, Dodge LD, Biro C. Experimental lumbar spinal stenosis. Analysis of the cortical evoked potentials, microvasculature and histopathology. *J Bone Joint Surg Am* 1990; 72: 110-20.
33. Swainston Harrison T, Plosker GL. Limaprost. *Drugs* 2007; 67: 109-18.
34. Keniston RC, Nathan PA, Leklem JE, Lockwood RS. Vitamin B6, vitamin C, and carpal tunnel syndrome. A cross-sectional study of 441 adults. *J Occup Environ Med* 1997; 39: 949-59.
35. Waikakul W, Waikakul S. Methylcobalamin as an adjuvant medication in conservative treatment of lumbar spinal stenosis. *J Med Assoc Thai* 2000; 83: 825-31.
36. Tsuji H, Tamaki T, Itoh T, et al. Redundant nerve roots in patients with degenerative lumbar spinal stenosis. *Spine (Phila Pa 1976)* 1985; 10: 72-82.
37. Fukusaki M, Kobayashi I, Hara T, Sumikawa K. Symptoms of spinal stenosis do not improve after epidural steroid injection. *Clin J Pain* 1998; 14: 148-51.
38. Katz WA, Rothenberg R. The nature of pain: pathophysiology. *J Clin Rheumatol* 2005; 11(2 Suppl): S11-5.
39. Melzack R,Coderre TJ, Katz J, Vaccarino AL. Central neuroplasticity and pathological pain. *Ann N Y Acad Sci* 2001; 933: 157-74.
40. Pasqualucci A, Varrassi G, Braschi A, et al. Epidural local anesthetic plus corticosteroid for the treatment of cervical brachial radicular pain: single injection versus continuous infusion. *Clin J Pain* 2007; 23: 551-7.
41. Decosterd I, Woolf CJ. Spared nerve injury: an animal model of persistent peripheral neuropathic pain. *Pain* 2000; 87: 149-58.
42. Winnie AP, Hartman JT, Myers HL Jr, Ramamurthy S, Baragan V. Pain clinic. II. Intradural and extradural corticosteroids for sciatica. *Anesth Analg* 1972; 51: 990-1003.
43. Coderre TJ. Contribution of protein kinase C to central sensitization and persistent pain following tissue injury. *Neurosci Lett* 1992; 140: 181-4.
44. Johansson A, Hao J, Sjolund B. Local corticosteroid application blocks transmission in normal nociceptive C-fibres. *Acta Anaesthesiol Scand* 1990; 34: 335-8.
45. Cuckler JM, Bernini PA, Wiesel SW, Booth RE Jr, Rothman RH, Pickens GT. The use of epidural steroids in the treatment of lumbar radicular pain. A prospective, randomized, double-blind study. *J Bone Joint Surg Am* 1985; 67: 63-6.
46. Whitman JM, Flynn TW, Childs JD, Wainner RS, Gill HE, Ryder MG, et al. A comparison between two physical therapy treatment programs for patients with lumbar spinal stenosis: a randomized clinical trial. *Spine (Phila Pa 1976)* 2006; 31: 2541-9.
47. Pua YH, Cai CC, Lim KC. Treadmill walking with body weight support is no more effective than cycling when added to an exercise program for lumbar spinal stenosis: a randomized controlled trial. *Aust J Physiother* 2007; 53: 83-9.
48. Kurihara A, Tanabe T, Mishima Y. Therapeutic effect of OP-1206 α -CD on lumbar spinal canal stenosis: multi-center comparative double blind clinical study (Japanese). *Rinsho Iyaku* 1996; 12: 511-29.
49. Uratsuji M, Kurihara A, Iguchi T. The optimal dose for OP-1206 α -CD on lumbar spinal canal stenosis: multi-center comparative double blind clinical study (Japanese). *Rinsho Iyaku* 1996; 12: 489-509.
50. Cheng P, Ma C, Wang XL, Lang HT. Salmon calcitonin plus rehabilitative therapy for lumbar spinal stenosis (Chinese). *Chin J Clin Rehabil* 2006; 10: 32-4.