SYMMETRY OF PARASPINAL MUSCLE DENERVATION IN CLINICAL LUMBAR SPINAL STENOSIS: SUPPORT FOR A HYPOTHESIS OF POSTERIOR PRIMARY RAMUS STRETCHING?

ANDREW J. HAIG, MD,[1] ZACHARY LONDON, MD,[2] and DANIELLE E. SANDELLA, BS[1]

[1] Department of Physical Medicine and Rehabilitation, University of Michigan, 325 East Eisenhower, Ann Arbor, Michigan, 48108, USA

[2] Department of Neurology, University of Michigan, Ann Arbor, Michigan, USA

Accepted 4 December 2012

ABSTRACT: *Introduction:* Denervation of the paraspinal muscles in spinal disorders is frequently attributed to radiculopathy. Therefore, patients with lumbar spinal stenosis causing asymmetrical symptoms should have asymmetrical paraspinal denervation. *Methods:* Seventy-three patients with clinical lumbar spinal stenosis, aged 55–85 years, completed a pain drawing and underwent masked electrodiagnostic testing, including bilateral paraspinal mapping and testing of 6 muscles on the most symptomatic (or randomly chosen) limb. *Results:* With the exception of 10 subjects with unilateral thigh pain ($P = 0.043$), there was no relationship between side of pain and paraspinal mapping score for any subgroups (symmetrical pain, pain into 1 calf only). Among those with positive limb EMG (tested on 1 side), no relationship between side of pain and paraspinal EMG score was found. *Conclusion:* Evidence suggests that paraspinal denervation in spinal stenosis may not be due to radiculopathy, but rather due to stretch or damage to the posterior primary ramus.

Muscle Nerve **48**: 198–203, 2013

Lumbar spinal stenosis is the most common reason older persons undergo spine surgery,[1] and its prevalence is expected to rise as the population ages. The disorder has been a diagnostic dilemma, with arbitrary or unproven radiological criteria widely espoused and cited, even in major trials.[2–5]

Recently, electrodiagnostic testing (EDx), especially the quantified paraspinal mapping needle electromyographic protocol for examination of the paraspinal muscles, has been shown to be highly specific and moderately to highly sensitive (depending apparently on severity) in the clinical diagnosis.[6,7] Although studies have shown denervation of paraspinal and limb muscles in clinically apparent stenosis, these did not always occur together. In 1 study of 51 persons with mostly mild to moderate clinical stenosis who underwent paraspinal mapping and examination of 6 limb muscles, 11 had limb fibrillation potentials, 12 had paraspinal fibrillation potentials, and 6 had both.[6] Limb and paraspinal data from a second study were more difficult to sort out, but apparently 12 of 28 subjects had limb fibrillation potentials on a 3-muscle examination and 27 of 28 had paraspinal fibrillation potentials.[7]

A number of possibilities may explain the lack of complete concordance between limb and paraspinal electromyography (EMG). Purely sacral radiculopathies have no representation in the paraspinal muscles. Also, there could be sampling error. Testing of more limb muscles might result in more abnormalities concordant with paraspinal findings. The statistical cut-off for normal paraspinal muscles (paraspinal mapping score <5) may be too stringent. However, it is also possible that the paraspinal muscles are involved in a different pathophysiology than the limb muscles. For example, it is known that paraspinal muscle denervation occurs in asymptomatic subjects.[8,9] This has generated the hypothesis that paraspinal denervation might be caused by stretch of the posterior primary ramus that innervates these muscles, but not the limb muscles.[10] This stretch and paralysis is thought to predispose to segmental instability, and the instability causes further stretch in a cycle of denervation and joint degeneration.

If the problem is due to sampling error, we would still expect subjects with symptoms on 1 side only to have paraspinal denervation primarily on that side. However, if the problem is stretch of the posterior primary ramus, this may be a more symmetrical process despite asymmetry of clinical complaints.

The Michigan Spinal Stenosis Study (MSSS), described in numerous publications, but best summarized by Haig et al.,[11] and a subsequent Michigan Spinal Stenosis Study II (MSSS II, which has not yet been described in the literature), contain data on paraspinal mapping bilaterally and on the laterality of symptoms. These data sets can be used to address the question. The purpose of this study was to determine whether paraspinal denervation is more prominent on the more symptomatic side in persons with clinically evident lumbar spinal stenosis.

METHODS

Our investigation draws data from the MSSS, which is a masked study of EDx and MRI in

Abbreviations: AANEM, American Association of Neuromuscular and Electrodiagnostic Medicine; EMG, electromyography; EDx, electrodiagnostic testing; MSSS, Michigan Spinal Stenosis Study

This study was supported by the Eunice Kennedy Shriver National Institute of Child Health & Human Development (R01HD059259).

Disclaimer: The content is solely the responsibility of the authors and does not necessarily represent the official views of the Eunice Kennedy Shriver National Institute of Child Health & Human Development or the National Institutes of Health.

Key words: back pain; electrodiagnosis; multifidus; paraspinal mapping; segmental instability; spinal stenosis

Correspondence to: A.J. Haig; e-mail: andyhaig@umich.edu

Published online 13 June 2013 in Wiley Online Library (wileyonlinelibrary.com). DOI 10.1002/mus.23750

subjects with no symptoms, low back pain without stenosis, and clinically evident stenosis. The general methodology of the MSSS and some of the data relating to the current work has been best described in 3 previous articles.[6,11,12] However, previous work focused primarily on those subjects who were not going to have surgery for their stenosis (and who thus had generally mild to moderate symptoms). A second trial has been undertaken, the MSSS II, which recruits asymptomatic volunteers and patients who have been offered surgery for spinal stenosis. Although the purposes and details of those studies are complex, the methodology related to the current investigation is similar between studies.

All potential subjects were screened prior to enrollment in the study. To qualify, the prospective participant had to be between 55 and 90 years of age. Exclusion criteria were: prior back surgery; any history of severe lower limb nerve injury; severe swelling in the legs; a known personal or familial history of polyneuropathy or other neuromuscular disease; a consumer of greater than 12 alcoholic drinks per week; a weight >300 pounds; having any implanted electrodes (such as defibrillators), surgical staples, or metal implants considered not MRI-safe; taking prescription anticoagulants; or with a condition, such as severe heart disease or poor balance, that would make it unsafe to complete a walking test. In addition, potential subjects who were not recruited as part of the vascular group could not have diabetes. Specific requirements for each group were described next.

Subjects with apparent clinical lumbar spinal stenosis were recruited through review of records of the physical medicine and rehabilitation, orthopedic surgery, and neurosurgery clinics of a university spine program. Potential subjects had a clinical diagnosis of spinal stenosis and symptoms of neurogenic claudication, and claimed difficulty walking 200 yards due to the symptoms of stenosis. To ensure a certain level of disease severity for the MSSS II study, subjects were required to have been offered surgery by an orthopedic or neurologic surgeon. A decision to proceed with surgery was not required.

Subjects with non-specific low back pain were recruited from clinic schedules at the university's spine program. Patients being seen at the spine program qualified for the study if they had a primary complaint of low back pain without leg pain, were not thought by their clinicians to have clinical spinal stenosis, had not had previous back surgery, and met all of the eligibility criteria as outlined earlier.

Subjects were recruited for the vascular group through review of medical records in the vascular surgery and diagnostic vascular clinics. Potential participants had pain while walking attributed to vascular disease, and were limited to $\leq$200 yards of walking by their symptoms. Vascular subjects did not have any known spinal stenosis and did not have back pain; due to scarcity of potential vascular subjects who had neither diabetes nor anticoagulation, diabetes subjects were permitted inclusion in this study group.

Asymptomatic volunteers were recruited through postings on the university's website as well as in the community. Asymptomatic volunteers were volunteers who met all of the eligibility criteria and who did not have a complaint of back or leg pain.

All subjects filled out an extensive questionnaire. Pertinent to the current study is a pain drawing. These were coded as having pain in the back, gluteal region, thighs, legs, or feet on the right and left. Subsequently, the pain drawing results were reclassified as non-localized, weakly localized, or strongly localized pain. The criteria for each class were as follows: non-localized included those whose pain drawings showed markings on the back only (no leg pain), or markings on both legs including 1 thigh and the opposite lower leg (calf and/or ankle), both thighs, and both lower legs. Weakly localized was defined as pain in 1 thigh only, whereas strongly localized was pain in 1 lower leg only.

In addition to questionnaires, subjects underwent an electrodiagnostic examination by the physical medicine and rehabilitation or neurology specialists, who were either board-certified or board-eligible in neuromuscular and electrodiagnostic medicine. The physicians were not allowed to discuss clinical issues or to perform a physical examination with the subjects, and were thus effectively masked to the subjects' clinical presentation by a process validated elsewhere.[12]

The technical examination included paraspinal mapping needle EMG on both sides. Paraspinal mapping is a quantified, codified needle examination of the L2-, L3-, L4-, and L5-innervated multifidus and muscles sampled incidentally during the approach to these muscles.[13,14] Persons who are interested in performing the procedure are advised to read the AANEM course handout.[14] Briefly, the procedure involves insertion of a 50–75-mm monopolar EMG needle through the skin at 4 locations, which are 2.5 cm lateral to the spinous process, 1 level below each of these vertebrae. The needle is directed toward the spinous process at a 45;–60° depth in short insertions. Reproducible abnormal spontaneous activity is scored 0–4^+ in the early part of the insertion, and separately in the last 1 cm of insertion. On contact with the spinous process the needle is withdrawn to the surface and redirected 45° cranially, then on contact with bone again withdrawn and redirected 45°

Table 1. Demographics of the sample.

Age (years) [mean (SD)]	65.4 (8.1)
Gender	43.3% male
Race	
White	86.6%
Black	8.2%
Other	5.2%
Body mass index	29.0 (5.3)
Duration of symptoms (years)	4.4 (6.8)
Severity of symptoms	
Pain Disability Index total	4.4 (2.4)
Visual Analog Pain Scale [average for week (cm)]	26.4 (16.5)
Limb denervation	30.6% positive

caudally. This results in 4 skin punctures, 12 insertions to the bone, and 24 scores of 0–4, with a total possible score of 96 on 1 side. Paraspinal mapping has been shown to have excellent test–retest and interrater reliability with age-related ranges of normal defined through masked testing of asymptomatic volunteers.[15]

In addition, a 6-muscle EMG on the side determined by a research assistant to be more symptomatic (or if symptoms were symmetrical, by coin toss not observed by the electrodiagnostician). A needle was inserted 6 times in 4 directions to seek abnormal spontaneous activity in each of the following muscles: gluteus maximus; tensor fascia lata; vastus medialis; tibialis anterior; fibularis longus; and medial gastrocnemius. Spontaneous activity was scored 0–4^{+} in each muscle using the Daube criteria.[16] A minimum of 10 motor unit's were observed in each muscle and the number of polyphasic motor unit's, subjectively scored, was recorded. EMG testing also included ipsilateral sural and fibular motor nerve conduction studies and bilateral H-waves.

RESULTS

Two hundred forty-two subjects participated in the MSSS studies, including 99 with clinical stenosis, 57 with mechanical low back pain, 17 with peripheral vascular disease, and 69 asymptomatic volunteers. The current study included the group with lumbar spinal stenosis only (total = 73 subjects) with complete, usable data. Table 1 presents subject demographics.

There was no significant difference in paraspinal mapping scores on the asymptomatic side when compared with the symptomatic side in any of the groups, except the weakly localized group, which showed a significant difference between scores ($N = 10$, $P = 0.043$) (Table 2).

Additional analysis was performed on the group of subjects who were found to have at least 1 leg muscle with denervation. A total of 22 subjects were included in this analysis. The group as a whole showed symmetrical paraspinal mapping scores, as did all of the subgroups (Table 3).

DISCUSSION

The study set out to determine whether paraspinal denervation was higher on the side of worse symptoms in persons with lumbar spinal stenosis. In large part, this was found to be not true. Methodologic strengths and limitations need to be understood. Implications of this finding, both theoretical and clinical, should be discussed.

Methodology and Findings. The study methodology has numerous strengths, including masking, reasonable clinical criteria for diagnosis of clinical spinal stenosis, and quantification of EMG findings. Pain drawings are well-established measures of pain location for persons with spinal disorders.[17] By combining the data from the original MSSS with the second trial, we have included a fairly large number of subjects with diverse presentations in addition to those with back pain and asymptomatic controls. These subjects met typical criteria for spinal stenosis—clinical impression plus imaging. There remain no validated clinical criteria for stenosis, however, so generalization to populations defined differently may be limited. For example, although neurogenic claudication is considered by many to be the hallmark of stenosis, it has not been declared the *sine qua non* of the diagnosis.

Table 2. Paraspinal mapping scores in subjects with spinal stenosis based on localization of pain.

	N	Symptomatic side/ testing side [mean (SD)]	Asymptomatic side [mean (SD)]	Paired *t*-test *P*-value
Non-localized	45	4.02 (7.03)	4.29 (7.14)	0.707
Non-localized, above knee only	17	4.65 (9.6)	4.53 (8.2)	0.923
Weakly localized	10	5.10 (5.04)	0.50 (0.97)	0.043[a]
Strongly localized	18	3.61 (4.88)	3.94 (5.06)	0.786
Localized pooled	28	3.79 (4.85)	2.71 (4.39)	0.294

Non-localized defined as: (a) pain in the low back without pain in either leg or (b) pain in both thighs or (c) pain in both lower legs (calf and ankle/foot) or (d) pain in one calf and other thigh. Weakly localized defined as: 1 leg without pain, other leg with pain in thigh. Strongly localized defined as: 1 leg without pain, other leg with pain in lower leg (calf and ankle/foot).

Table 3. Paraspinal mapping scores in subjects with spinal stenosis based on localization of pain in subjects with limb denervation based on EMG.

	N	Symptomatic side/ testing side [mean (SD)]	Asymptomatic side [mean (SD)]	Paired *t*-test (*P*-value)
Total	22	7.92 (9.9)	7.96 (9.7)	0.977
Non-localized	13	7.23 (8.0)	9.38 (10.4)	0.180
Weakly localized	3	6.67 (7.4)	0.0 (0.0)	0.258
Strongly localized	6	3.50 (4.14)	5.5 (7.5)	0.482
Localized pooled	9	4.56 (5.2)	3.67 (6.5)	0.736

Non-localized defined as: (a) pain in the low back without pain in either leg or (b) pain in both thighs or (c) pain in both lower legs (calf and ankle/foot) or (d) pain in 1 calf and other thigh. Weakly localized defined as: 1 leg without pain, other leg with pain in thigh. Strongly localized defined as: 1 leg without pain, other leg with pain in lower leg (calf and ankle/foot).

In our study, EMG was not performed on the limb muscles contralateral to the symptomatic side. If these asymptomatic limb muscles were studied, denervation changes may have supported the theory that stenosis is truly a bilateral process, even when symptoms are not. If the contralateral limb muscles were normal, however, it would lend credence to the hypothesis that the paraspinal denervation on the asymptomatic side is due to isolated stretch of the posterior primary ramus. This is an important area for future exploration.

Scientific and Clinical Implications. The finding that paraspinal denervation is relatively symmetrical in persons with clinical lumbar spinal stenosis has a number of implications with regard to the pathophysiology of spinal stenosis. Over time, the pathophysiology of this apparently straightforward disorder has been found to be more difficult to understand. The earliest studies[18] attributed classic symptoms, such as neurogenic claudication, to a small spinal canal, as demonstrated on X-ray. For half a century anatomical measures have been used to "diagnose" stenosis by myelography, followed later by computerised tomography, and most recently magnetic resonance imaging. These include measures of the dimensions or areas of the spinal canal or thecal sac. A number of recent studies, 1 of which includes some individuals in the current database, have shown that none of these measures discriminate individuals with symptoms from asymptomatic volunteers of the same age group, and that perhaps half the older people in the community met the previously touted criteria for spinal stenosis.[6,7,19,20] In retrospect, it appears illogical that a static prone test (MRI, computed tomography, or myelography) would demonstrate the pathophysiology of a disorder when its symptoms often occur only upon standing or walking.

Denervation of the limbs and especially of the paraspinal muscles also does not exactly reflect the pathophysiology of the symptom of neurogenic claudication. EMG has been shown to be a highly specific diagnostic test, with moderate to high sensitivity depending perhaps on the severity of symptoms.[6,7] However, denervation as detected on needle EMG is also not something that comes and goes with standing and lying down. Fibrillation potentials represent spontaneous depolarization of a muscle fiber, a phenomenon that only occurs after axonal loss, Wallerian degeneration, and reconfiguration of the neuromuscular junction. Fibrillation potentials in the lumbar paraspinal muscles are not thought to appear for 10–14 days after axonal loss, although this has not been studied prospectively in spinal disorders.[21] The timing of reinnervation and cessation of fibrillation has not been well established. Because fibrillation's follow degeneration of the synapse, muscles do not fibrillate and stop fibrillating when a person walks and rests. Denervation is either a consequence of repeated injury or an epiphenomenon, or both. Furthermore, a process of minor denervation and reinnervation may be a more accurate reflection of this chronic disorder.

We found that paraspinal denervation does not relate well to the side of a pain complaint in patients with spinal stenosis. Spinal stenosis is typically a central phenomenon, so there is no reason to believe that all of the nerve damage would be on 1 side, such as one finds with lateral disk herniations. Also, when the pain is in the back or gluteal region, one may suspect that this is because the pain is not from nerve, but is instead associated facet or sacroiliac or musculo-ligamentous pain. However, pain below the knee is typically neurogenic, yet persons in this study with unilateral pain below the knee had symmetrical paraspinal denervation. We conclude that the process of paraspinal denervation is different from the process of neurogenic pain, at least in these cases.

We suspect that the mechanism of this finding may be isolated denervation of the posterior primary ramus. This branch of each lumbar nerve root travels posteriorly, then splits into 3 branches.[22] The lateral branch innervates the iliocostalis and the overlying skin. The intermediate

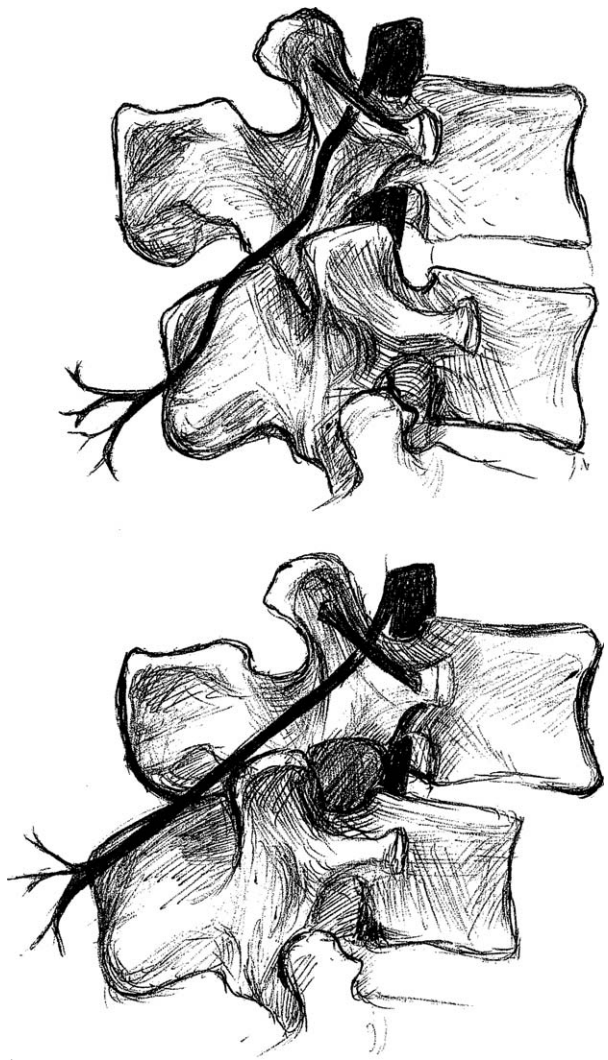

FIGURE 1. Potential explanation for symmetrical paraspinal denervation in stenosis. Segmental hypermobility both compromises the area of the spinal canal and creates tension where the posterior primary ramus travels under the mammilo-accessory ligament (figure courtesy of Karin Roszell).

branch goes to the longissimus muscle. Highly relevant to the current discussion, the medial branch travels to the multifidus muscle, the facet joint, and other structures that are prominent with regard to pain. As Figure 1 shows, the posterior primary ramus passes under the mammilo-accessory ligament, an area where some have proposed it can be entrapped or tethered and stretched.[23,24] Segmental hypermobility may symmetrically stretch this posterior primary ramus, again illustrated in Figure 1. It is not clear whether this in itself causes some aspect of the symptoms of stenosis. However, it is apparently not related to damage to the ventral ramus that goes down the leg.

One theory about spinal stenosis is that symptoms occur as a result of segmental instability. When standing, gravity pulls 1 vertebra forward on the vertebra below. This can be resisted by passive resistance of ligaments or active resistance by muscles, either 1 of which can change as a person stands for a long time or walks. However, if there is a shift, the spinal canal becomes smaller, perhaps causing the venous congestion that Porter claims is the pathophysiological mechanism of neurogenic claudication, and that Laban has observed in clinical cases of worsening leg pain at night in patients with congestive heart failure.[25,26] We have proposed that this vicious cycle of stretch, causing weakness, and then causing more stretch, may be part of the process of lumbar spinal stenosis. This theory was not supported, however, in a study that showed no relationship between paraspinal denervation and radiological or clinical progression of stenosis over 22 months.[10] However, this is a relatively short time-frame for spinal degenerative processes. Our study has provided other evidence that somewhat supports this hypothesis. Scientists may find other ways in the laboratory or clinic to monitor the paraspinal muscles and the posterior primary ramus in relation to clinical spinal stenosis.

The clinical implications of this study are less clear. Regardless of theoretical concerns it remains true that EMG is a good diagnostic test for clinically apparent lumbar stenosis.[19] There is also increasing evidence that using direct epidural injections toward the denervation found on EMG is more effective than not using the EMG data. One may conclude from this study that paraspinal findings do not necessarily represent the root damage. So, directing an injection or surgery at the level found on paraspinal mapping would not make sense. However, in this study we have not adequately addressed this issue. Posterior primary ramus stretch may still represent the location of the lesion causing symptoms. Also, it remains possible that compression of roots from stenosis preferentially and symmetrically impacts the paraspinal muscles.

From a therapeutic standpoint the data contrast with, but do not conflict with, work by Hodges and colleagues.[27] They found focal asymmetrical atrophy of paraspinal muscles after simple backache (with no apparent neurological involvement), which did not improve after resolution of symptoms, until an exercise program specific to these muscles was implemented.[26] However, in stenosis, at least part of the "atrophy" is denervation. In contrast to at least 1 book written for the public,[28] exercise of the weak paraspinals will not make the nerve root or posterior primary ramus grow back faster. Other goals and other mechanisms of strengthening need to be addressed instead.

In conclusion, paraspinal denervation does not favor the side of increased symptoms in persons

with lumbar spinal stenosis. This supports, but does not prove, the theory that a symmetrical process such as stretch of the posterior primary ramus is the cause of some paraspinal denervation. The pathophysiology of spinal stenosis might include a downward spiral of denervation causing stretch of the posterior primary ramus, thus causing further denervation of the back muscles.

REFERENCES

1. Deyo RA. Trends and variations in the use of spine surgery. Clin Orthopaed Rel Res 2006;443:139–146.
2. Atlas SJ, Deyo RA, Keller RB, Chapin AM, Patrick DL, Long JM, et al. The Maine Lumbar Spine Study, Part III: 1-Year outcomes of surgical and nonsurgical management of lumbar spinal stenosis. Spine 1996;21:1787–1794.
3. NASS. Evidence based clinical guidelines for multidisciplinary spine care: diagnosis and treatment of degenerative lumbar spinal stenosis. Burr Ridge, IL: NASS; 2007.
4. Steurer J, Nydegger A, Held U, Brunner F, Hodler J, Porchet F, et al. LumbSten: the lumbar spinal stenosis outcome study. BMC Musculoskel Disord 2010;11:254.
5. Weinstein JN, Tosteson TD, Lurie JD, Tosteson A, Blood E, Herkowitz H, et al. Surgical versus nonoperative treatment for lumbar spinal stenosis four-year results of the spine patient outcomes research trial. Spine 2010;35:1329–1338.
6. Haig AJ, Geisser ME, Tong HC, Yamakawa KS, Quint DJ. Hoff JT, et al. Electromyographic and magnetic resonance imaging to predict lumbar stenosis, low-back pain, and no back symptoms. J Bone Joint Surg Am 2007;89:358–366.
7. Yagci I, Gunduz OH, Ekinci G, Diracoglu D, Us O, Akyuz G. The utility of lumbar paraspinal mapping in the diagnosis of lumbar spinal stenosis. Am J Phys Med Rehabil 2009;88:843–851.
8. Date ES, Mar EY, Bugola MR, Teraoka JK. The prevalence of lumbar paraspinal spontaneous activity in asymptomatic subjects. Muscle Nerve 1996;19:350–354.
9. Haig AJ, LeBreck DB, Powley SG. Paraspinal mapping: quantified needle electromyography of the paraspinal muscles in persons without low back pain. Spine 1995;20:715–721.
10. Haig AJ. Paraspinal denervation and the spinal degenerative cascade. Spine J 2002;2:372–380.
11. Haig AJ, Tong HC, Yamakawa KSJ, Parres C, Quint DJ, Chiodo A, et al. Predictors of pain and function in persons with spinal stenosis, low back pain, and no back pain. Spine 2006;31:2950–2957.
12. Haig AJ, Yamakawa KSJ, Parres C, Chiodo A, Tong H. A prospective, masked 18-month minimum follow-up on neurophysiologic changes in persons with spinal stenosis, low back pain, and no symptoms. PM&R 2009;1:127–136.
13. Haig AJ. Clinical experience with paraspinal mapping. II: A simplified technique that eliminates three-fourths of needle insertions. Arch Phys Med Rehabil 1997;78:1185–1190.
14. Haig AJ. Paraspinal mapping. American Association of Electrodiagnostic Medicine workshop handout. Rochester, MN: AAEM; 2000 (revised edition 2005).
15. Tong HC, Haig AJ, Yamakawa KSJ, Miner JA. Paraspinal electromyography: age-correlated normative values in asymptomatic subjects. Spine 2005;30:E499–E502.
16. Daube JR. AAEM minimonograph #11: Needle examination in clinical electromyography. Muscle Nerve 1991;14:685–700.
17. Margolis RB, Chibnall JT, Tait RC. Test–retest reliability of the pain drawing instrument. Pain 1988;33:49–51.
18. Verbiest H. Pathomorphologic aspects of developmental lumbar stenosis. Orthoped Clin N Am 1975;6:177–196.
19. Haig AJ, Tomkins CC. Diagnosis and management of lumbar spinal stenosis. JAMA 2010;303:71–72.
20. Kalichman L, Cole R, Kim DH, Li L, Suri P, Guermazi A, et al. Spinal stenosis prevalence and association with symptoms: the Framingham Study. Spine J 2009;9:545–550.
21. Preston DC, Shapiro B. Electromyography and neuromuscular disorders: clinical–electrophysiologic correlations. Oxford: Butterworth-Heinemann; 2005. 704 p.
22. Bogduk N, Wilson AS, Tynan W. The human lumbar dorsal rami. J Anat 1982;134:383–397.
23. Fisher MA. Electrodiagnostic examination, back pain and entrapment of posterior rami. Electromyogr Clin Neurophysiol 1985;25:183–189.
24. Wu PB, Huang SQ, Russel K. The dorsal ramus myotome: anatomical description and clinical implications in electrodiagnosis. Muscle Nerve 1991;14:887–888.
25. LaBan MM. Restless legs syndrome associated with diminished cardiopulmonary compliance and lumbar spinal stenosis—a motor concomitant of "Vesper's curse." Arch Phys Med Rehabil 1990;71:384–388.
26. Porter RW. Spinal stenosis and neurogenic claudication. Spine 1996;21:2046.
27. Hodges P, Holm AK, Hansson T, Holm S. Rapid atrophy of the lumbar multifidus follows experimental disc or nerve root injury. Spine 2006;31:2926–2933.
28. Johnson J. Tune up #1. How to make your back much stronger. In: Treat your own spinal stenosis. Indianapolis, IN: Dog Ear Publishing; 2010.