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Advertising Editor, Journal of the Canadian Chiropractic Association
186 Spadina Avenue, Suite 6, Toronto, Ontario M5T 3B2
Tel: 416-585-7902 877-222-9303 Fax: 416-585-2970

Email: Dr. Allan Gotlib<AGotlib@jcca.ca>
Website: www.jcca-online.org

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Beyond the Spine: A New Clinical Research Priority

James Donovan, BSc, DC
J. David Cassidy, DC, PhD, DrMedSc
Carol Cancelliere, DC, MPH
Erik Poulsen, DC, PhD
Mette Jensen Stochkendahl, DC, PhD
Jørgen Kilsgaard, DC, MPH
Marc-André Blanchette, DC, MSc
Jan Hartvigsen, DC, PhD

Over the past two decades, clinical research within the chiropractic profession has focused on the spine and spinal conditions, specifically neck and low back pain. However, there is now a small group of chiropractors with clinical research training that are shifting their focus away from traditional research pursuits towards new and innovative areas. Specifically, these researchers are now delving into areas such as brain injury, work disability prevention, undifferentiated chest pain, hip osteoarthritis, and prevention of pain in children and adolescents to name a few. In this paper, we highlight recent research in these new areas and discuss how clinical research efforts in musculoskeletal areas beyond

Au cours des deux dernières décennies, les recherches cliniques dans le domaine de la chiropratique se concentrent sur la colonne vertébrale et les conditions connexes, en particulier les douleurs cervicales et lombaires. Toutefois, un petit groupe de chiropraticiens formés en recherches cliniques écarte maintenant les sujets de recherche habituels pour privilégier de nouveaux domaines novateurs. Plus précisément, ces chercheurs se concentrent maintenant sur différents sujets, notamment les lésions cérébrales, la prévention de l'incapacité découlant du travail, les douleurs thoraciques indistinctes, l'arthrose de la hanche, et la prévention de la douleur chez les enfants et les adolescents. Dans cet article, on souligne les récentes recherches dans ces nouveaux domaines et discute de la manière dont les efforts de recherche clinique réalisés dans les domaines musculosquelettiques au-delà de

From the Division of Health Care and Outcomes Research, Toronto Western Research Institute, University Health Network, University of Toronto, Canada (Donovan, Cancelliere, Cassidy); Institute of Health Policy, Management and Evaluation, University of Toronto, Canada (Cancelliere, Cassidy); Nordic Institute of Chiropractic and Clinical Biomechanics, Odense, Denmark (Stochkendahl, Poulsen, Hartvigsen), School of Public Health, University of Montreal, Montreal QC, Canada (Blanchette); KIApro Worklife Healthcare, Herlev, Denmark (Kilsgaard); Institute of Sports Science and Clinical Biomechanics, Faculty of Health, University of Southern Denmark, Odense, Denmark (Cassidy, Hartvigsen)

Correspondence to James Donovan, Toronto Western Research Institute, University Health Network, University of Toronto, LuCliff Place, 700 Bay Street, Suite 602, Toronto, Ontario, Canada, M5G 1Z6. Telephone: 416-201-1785. Email: jamesd@uhnresearch.ca

the spine can benefit patient care and the future of the chiropractic profession.

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KEY WORDS: brain injury, work disability, chest pain, OA, research, chiropractic

la colonne vertébrale peuvent améliorer les soins aux patients et contribuer à l'avenir de la chiropratique.

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MOTS CLÉS : lésion cervicale, incapacité découlant du travail, douleur thoracique, O.P., recherche, chiropratique

Introduction

Chiropractors with clinical research training have traditionally focused on the spine and its related disorders and especially neck and low back pain. Examples include the recent Decade of the Bone and Joint 2000-2010 Task Force on Neck Pain and Its Associated Disorders[1] as well as several excellent randomized trials of spinal manipulative therapy (SMT) for neck[2] and low back pain[3-5]. Despite these and many other research successes, from 1990 to 2010 disability from spine-related pain has significantly increased, with low back pain now the leading cause of global disability, affecting 10% of the population or more than 600 million people worldwide.[6] Over the same two decades, disability from other musculoskeletal disorders has also increased by 44.6%[7], and with an aging and increasingly sedentary society this trend is likely to continue and so too will the demand for improved care and prevention. Even patients seeking care for neck and low back pain rarely have pain isolated to just the spine and frequently report co-occurring non-spinal pain, not to mention other co-morbid diseases.[8] Chiropractors already commonly manage a variety of musculoskeletal disorders and at different anatomical sites, not just those related to the spine. Taken together, these facts provide a good basis to promote the growth of clinical research efforts in other non-spinal musculoskeletal areas.

Moreover, with the growing burden of musculoskeletal disorders there is a need for chiropractors to become more involved and integrated in interdisciplinary collaborative research efforts aimed at improving the understanding and care of such complex disorders. Increasing multidisciplinary clinical research collaboration was among the top priorities in the recently published research agenda for the chiropractic profession in Europe.[9] Further, a recent letter to the editor from this publication, opined that the fate of the chiropractic profession depends on research and education as well as the capacity for chiropractors to function and thrive in interdisciplinary collaboration.[10] One way to secure the future growth of the chiropractic profession may be to prioritize support for clinical research in musculoskeletal areas beyond the spine and more specifically, clinical research that's interdisciplinary and collaborative in nature. Presently, a group of chiropractors with post-graduate clinical research training are involved in innovative, collaborative research efforts in important, but less traditional areas of research such as mild traumatic brain injury (MTBI), work disability prevention, undifferentiated chest pain, hip osteoarthritis, and prevention of spine pain in children and adolescents to name a few. The aim of this commentary is twofold: to highlight recent findings from several examples of collaborative clinical research and discuss how clinical research efforts in areas beyond the spine can enhance the capacity for interdisciplinary collaboration, improve outcomes for patients and solidify the future growth of the chiropractic profession.

Discussion

Research Examples: MTBI

In 2004 the WHO Collaborating Centre for Neurotrauma, Prevention, Management and Rehabilitation (WHO Task Force) published the first-ever systematic review on the course and prognosis of MTBI.[11] Ten years later, the International Collaboration on Mild Traumatic Brain Injury Prognosis (ICoMP) undertook a series of systematic reviews and best-evidence syntheses to update the WHO Task Force findings.[12] This 21-member collaboration was led by Dr. J. David Cassidy, a chiropractor and epidemiologist, and included 5 other chiropractors as well as other top international clinician/research scientists in brain injury.

Overall, the ICoMP results indicate post-traumatic symptoms including neck pain and headache are common sequelae after MTBI.[13] Spinal-related pain appears strongly associated with overall MTBI recovery[14] and reducing MTBI-associated somatic pain (e.g., spine and head pain) may help improve recovery. One ICoMP paper by Jan Hartvigsen, another chiropractor/epidemiologist and his colleagues showed that those suffering on-going MTBI symptoms after a traffic collision-related MTBI sought more care from allied health professionals, including chiropractors, over the course of the first year after the injury.[15] The overall findings from the ICoMP suggest that chiropractors can make important contributions, both from a research and a clinical perspective in the area of MTBI.

Athletes are a particular group in need of better evidence-informed care for sport-related concussion. Head-injuries to high-level athletes and the ensuing media attention has sensationalized issues surrounding concussion for the general public, including amateur and recreation level athletes, and this creates the potential for confusion and misinformation. An ICoMP systematic review on prognosis after sport concussion led by Carol Cancelliere, a chiropractor pursuing a PhD degree in clinical epidemiology addressed issues and concerns surrounding concussions in athletes, including immediate vs. delayed return to play, the possible risks associated with repeat concussions, physical and cognitive sequelae of concussions, and others.[16] Chiropractors involved in the care of athletes are well-positioned to incorporate the ICoMP's evidence-based findings to help make better informed decisions and improve the outcome for concussed athletes of all levels, as well as better educate parents and coaches.

Finally, prognostic research on MTBI is now shedding light on possible similarities between MTBI and other traumatic injuries. For instance, similar post-traumatic symptoms can occur after whiplash, or MTBI or other orthopedic-related injuries; these symptoms include headache, dizziness, nausea, fatigue, concentration and memory problems and spinal pain, to name a few.[13,14,17] This suggests that these symptoms are not specific or unique to either MTBI or whiplash, but may be a non-specific response to trauma-related physical or psychological stress in general. This is underscored by the fact that predictors of recovery are less related to injury type, but more related to, for instance, patients' expectations and beliefs about recovery.[14,18] In other words, whether a patient has sustained a MTBI or a whiplash injury, those who expect to recover more slowly have a worse prognosis than those who are more optimistic about their recovery. In addition, previous research in whiplash suggests that those who rely on passive coping strategies recover more slowly than those that don't.[19] One of the ICoMP reviews identified predictors strongly associated with self-reported recovery after MTBI, and they appear quite similar to those determining whiplash recovery, or patients suffering from other traumatic conditions or non-specific spinal pain.[13] Further clinical research would help to develop the evidence-base needed to better define the relationship between MTBI and other traumatic injuries.

Research Examples: Work Disability Prevention

Managing RTW can be difficult for clinicians because the determinants of RTW can be far reaching, extending well beyond those of the injury and may involve psychosocial issues such as depression and job dissatisfaction, workplace issues such as lack of modified duties, unsupportive supervisor and co-workers and insurance issues such as complicated compensation programs and delayed benefits.[20-23] With various influences on the RTW process, a multidisciplinary rehabilitation approach using clinical and occupational interventions can be beneficial. One of us (JK) developed and designed a coordinated and tailored work rehabilitation (CTWR) program that was later tested in a randomized controlled trial in Denmark. The trial compared the effectiveness of the CTWR program to conventional case management and included an interdisciplinary team of chiropractors and others that collaboratively screened and tailored RTW specific rehabilitation plans for participants.[24] Overall the CTWR group showed reduced sickness-absence-hours after 6 months and the results indicate that effective CTWR recommendations include psychological therapy to address RTW barriers, promoting workplace supervisor support and attending roundtable work-related discussions. This important piece of clinical research highlights how chiropractors can be effective multidisciplinary RTW team members, skilled in effectively guiding injured workers back to sustained employment. Dr. Patrick Loisel a leading WDP researcher, who pioneered RTW interventions at the workplace, largely influenced this work.[25] Dr. Loisel

is currently offering training to chiropractors in work disability prevention through the Canadian Memorial Chiropractic College.

Research Examples: Undifferentiated chest pain

An attack of acute chest pain can have many causes, not all of which are dire. In fact, after serious pathology such as myocardial infarction has been ruled out, these patients are often discharged from the emergency department (ED) with the diagnosis of undifferentiated chest pain, i.e. chest pain of unknown origin. Though seemingly benign, the pain associated with this type of chest pain and the lack of available treatment options makes managing these patients a challenge for medical professionals and they often re-present to the ED with the same problem.[26] An often over-looked cause of acute undifferentiated chest pain is pain from the cervico-thoracic muscles and joints, creating a subtype of this condition termed musculoskeletal chest pain. A recent Danish randomized controlled trial by Mette Jensen Stochkendahl, a chiropractor and PhD clinical researcher and her colleagues compared chiropractic care, including SMT of the thoracic and/or cervical spine to the normal self-management program for patients presenting to the Odense University Hospital in Denmark with acute musculoskeletal chest pain.[27,28] The results demonstrated a positive change in self-perceived chest pain and an improved change in pain intensity in favour of chiropractic care. Additionally, patients receiving chiropractic care reported significantly less thoracic spine and shoulder-arm pain. This study suggests that chiropractic care may help speed recovery for patients with acute musculoskeletal chest pain presenting to the ED. While these results are indeed significant for patients, there are also conceptual aspects of this study important for chiropractors: the intervention was delivered by eight community-based chiropractors, making this study highly relevant to those in everyday practice; it demonstrates how inter-professional collaboration, in this case chiropractic, cardiology, nuclear medicine and biostatistics can improve the outcomes for patients with challenging musculoskeletal conditions where medical treatments are limited or non-existent; and, this study serves as an example of how chiropractors may play a role in an otherwise unknown or poorly known area of musculoskeletal patient care.

Research Examples: OA of the hip

The societal burden of OA is substantial and estimated to continue to increase over the coming decades[29,30] In Canada alone, an estimated 4.4 million were living with the disease in 2010 resulting in significant reduced quality of life and risk of increased mortality.[31,32] Contrary to popular belief, not all patients diagnosed with hip and knee OA will require joint replacement surgery. A recent study documented that only 20% of patients have had hip replacement surgery up to 28 years after the initial radiographic diagnosis.[33] Further, in primary care the average time from initial radiographic diagnosis until referral for an orthopedic evaluation has been estimated at 82 months (i.e., 6 years and 10 months).[34] These important studies call for cost-effective and safe interventions for primary care patients who do not require or want an operation. The current evidence-based clinical guidelines for hip and knee OA[35-37] recommend an initial combined core intervention of non-pharmacological treatment focusing on patient education, exercise and if indicated, weight loss. Furthermore, recently published clinical trials[38-40] have demonstrated manual therapy having a clinically significant effect either as a mono or as co-intervention for patients with hip OA.

Last year, a Danish multidisciplinary three-arm parallel group randomized clinical trial by Eric Poulsen, a chiropractor and PhD clinical researcher and colleagues demonstrated that patient education (PE) combined with manual therapy (MT) was more effective than usual care in reducing pain and improving self-reported function and quality of life for patients with hip OA.[40] Nearly 80% of the patients receiving the MT + PE intervention classified themselves as improved versus only 22% in a group receiving only PE and 13% in the usual care groups. Even more, within the MT + PE group, 62% of patients experienced a 25% or more reduction in pain from baseline to 6 weeks, resulting in a number needed to treat (NNT) of just three. These results have important implications for patients: one out of every three patients who suffer OA of the hip would experience clinically significant reductions in pain after 6 weeks of a combined intervention of MT and a PE program. The study was completed at Odense University Hospital and the University of Southern Denmark where chiropractors and physicians are trained together; the practical aspects of the project involved collaboration between general medical practitioners, physiotherapists, orthopaedic surgeons and chiropractors.

Research Examples: Prevention and early treatment of musculoskeletal problems in children and adolescents

Pain from musculoskeletal disorders can start early in life[41,42] and children and adolescents with pain have a higher risk of experiencing pain as adults[43]. Therefore research into prevention and early effective treatment should remain a high priority for chiropractors.[44] Indeed two Danish landmark research projects will contribute with important new knowledge and help to define the role of chiropractors in this area.[45,46] One of those projects, the Childhood Health, Activity, and Motor Performance School Study (CHAMPS), which is headed by an orthopaedic surgeon, is a school-based study where 1,800 children in schools in the town of Svendborg, Denmark are cluster randomized offering either the normal two hours per week of physical education or six hours of physical education.[46] Four chiropractors are involved in the CHAMPS study as PhD students and several other chiropractors are involved either as senior scientists and supervisors or as clinicians. A large number of diverse research projects run by medical specialists from a range of fields, including physiotherapists and chiropractors are involved with this project and chiropractors will lead research that closely maps the occurrence and course of musculoskeletal problems in school children as well as evaluating the effect of age-specific physical education on back pain.

Conclusions

Chiropractors trained as clinical researchers are making substantial scientific contributions in major non-spinal musculoskeletal areas such as MTBI, arthritis, prevention, WDP and public health. These new collaborative clinical research examples serve to demonstrate the capacity for research success in clinical areas beyond the spine, which is encouraging news for musculoskeletal patients who are in need of better evidence-informed management, but also for the chiropractic profession, which will be able to play a stronger and more integrated role in improving the outcomes for these patients.

A recent commentary on the global challenges for the chiropractic profession suggested the need to prioritize the limited available research funds in order to both maximize the capacity for success and achieve measureable outcomes clinicians can actually use.[47] Over the past nearly 40 years, up to $80 million has reportedly been spent (across health professions) on the traditional research pursuit of determining the effectiveness of SMT for acute and chronic non-specific low back pain (LBP).[48] SMT is at present a well established part of evidence-based clinical practice guidelines for LBP[49,50] and these represent the informed application of research evidence to clinical care. That's not to suggest it's time to close the book on SMT research; however, to determine further effects of treatment will likely require research methods beyond what has been previously applied, including for example using advanced trial designs that are large enough in size to detect treatment-specific effects in well-defined subgroups within the cluster of patients with non-specific low back pain.[9,51] This example serves to highlight the need to challenge the existing state of affairs and possibly for the chiropractic profession to establish a new clinical research priority and more specifically to focus on innovative and higher yield research investment opportunities. To start, prioritizing funds for clinical research, that is to say research that directly benefits and informs the care of patients, in particular clinical research in other non-spinal musculoskeletal areas and that involves interdisciplinary collaboration may garner better and earlier returns per research dollar (i.e., outcomes clinicians can use). With increased funding, clinical researchers can continue to innovate in these and other non-traditional research areas, thereby benefiting patients and the future of the profession.

References

1. Haldeman S, Carroll L, Cassidy JD, Schubert J, Nygren A, Bone et al. The Bone and Joint Decade 2000-2010 Task Force on Neck Pain and Its Associated Disorders: executive summary. Spine (Phila Pa 1976). 2008;33(4 Suppl):S5-7.
2. Maiers M, Bronfort G, Evans R, Hartvigsen J, Svendsen K, Bracha Y et al. Spinal manipulative therapy and exercise for seniors with chronic neck pain. Spine J. 2014;14(9):1879-99.
3. Giles LG, Muller R. Chronic spinal pain: a randomized clinical trial comparing medication, acupuncture, and spinal manipulation. Spine (Phila Pa 1976). 2003;28(14):1490-503.
4. Haas M, Vavrek D, Peterson D, Polissar N, Neradilek MB. Dose-response and efficacy of spinal manipulation for care of chronic low back pain: a randomized controlled trial. Spine J. 2014;14(7):1106-16.
5. Hoiriis KT, Pfleger B, McDuffie FC, Cotsonis G, Elsangak O, Hinson R et al. A randomized clinical trial comparing chiropractic adjustments to muscle relaxants

for subacute low back pain. J Manipulative Physio Ther. 2004;27(6):388-98.
6. Murray CJ, Vos T, Lozano R, Naghavi M, Flaxman AD, Michaud C et al. Disability-adjusted life years (DALYs) for 291 diseases and injuries in 21 regions, 1990-2010: a systematic analysis for the Global Burden of Disease Study 2010. Lancet. 2012;380(9859):2197-223.
7. Smith E, Hoy DG, Cross M, Vos T, Naghavi M, Buchbinder R et al. The global burden of other musculoskeletal disorders: estimates from the Global Burden of Disease 2010 study. Ann Rheum Dis. 2014;73(8):1462-9.
8. Hartvigsen J, Natvig B, Ferreira M. Is it all about a pain in the back? Best Pract Clin Rheumatol. 2013;27(5):613-23.
9. Rubinstein SM, Bolton J, Webb AL, Hartvigsen J. The first research agenda for the chiropractic profession in Europe. Chiropr Man Therap. 2014;22(1):9.
10. Blanchette MA, Hartvigsen J. RE: Chiropractors as Primary Spine Care Providers: precedents and essential measures. [letter]. JCCA. 2014;58(1):96-97.
11. Carroll LJ, Cassidy JD, Peloso PM, Borg J, von Holst H, Holm L et al. Prognosis for mild traumatic brain injury: results of the WHO Collaborating Centre Task Force on Mild Traumatic Brain Injury. J Rehabil Med. 2004;(43 Suppl):84-105.
12. Cancelliere C, Cassidy JD, Côté P, Hincapié CA, Hartvigsen J, Carroll LJ et al. Protocol for a systematic review of prognosis after mild traumatic brain injury: an update of the WHO Collaborating Centre Task Force findings. Syst Rev. 2012;1:17.
13. Cassidy JD, Cancelliere C, Carroll LJ, Côté P, Hincapié CA, Holm LW et al. Systematic Review of Self-Reported Prognosis in Adults After Mild Traumatic Brain Injury: Results of the International Collaboration on Mild Traumatic Brain Injury Prognosis. Arch Phys Med Rehabil. 2014;95(3S):S132-S51.
14. Cassidy JD, Boyle E, Carroll LJ. Population-based, inception cohort study of the incidence, course, and prognosis of mild traumatic brain injury after motor vehicle collisions. Arch Phys Med Rehabil. 2014;95(3 Suppl):S278-85.
15. Hartvigsen J, Boyle E, Cassidy JD, Carroll LJ. Mild traumatic brain injury after motor vehicle collisions: what are the symptoms and who treats them? A population-based 1-year inception cohort study. Arch Phys Med Rehabil. 2014;95(3 Suppl):S286-94.
16. Cancelliere C, Hincapié CA, Keightley M, Godbolt AK, Côté P, Kristman VL et al. Systematic Review of Prognosis and Return to Play After Sport Concussion: Results of the International Collaboration on Mild Traumatic Brain Injury Prognosis. Arch Phys Med Rehabil. 2014;95(3S):S210-S29.
17. Ferrari R, Russell AS, Carroll LJ, Cassidy JD. A re-examination of the whiplash associated disorders (WAD) as a systemic illness. Ann Rheum Dis. 2005;64(9):1337-42.
18. Holm LW, Carroll LJ, Cassidy JD, Skillgate E, Ahlbom A. Expectations for recovery important in the prognosis of whiplash injuries. PLoS Med. 2008;5(5):e105.
19. Carroll LJ, Cassidy JD, Côté P. The role of pain coping strategies in prognosis after whiplash injury: passive coping predicts slowed recovery. Pain. 2006;124(1-2):18-26.
20. MacEachen E, Clarke J, Franche RL, Irvin E, Workplace-based Return to Work Literature Review Group. Systematic review of the qualitative literature on return to work after injury. Scand J Work Environ Health. 2006;32(4):257-69.
21. Ammendolia C, Cassidy JD, Steensta I, Soklaridis S, Boyle E, Eng S et al. Designing a workplace return-to-work program for occupational low back pain: an intervention mapping approach. BMC Musculoskelet Disord. 2009;10:65.
22. Loisel P, Durand MJ, Berthelette D, Vézina N, Baril R, Gagnon D, et al. Disability Prevention: New paradigm for the management of occupational back pain. Dis Manage Health Outcomes. 2001;9(7):351-60.
23. Iles RA, Davidson M, Taylor NF. Psychosocial predictors of failure to return to work in non-chronic non-specific low back pain: a systematic review. Occup Environ Med. 2008;65(8):507-17.
24. Bültmann U, Sherson D, Olsen J, Hansen CL, Lund T, Kilsgaard J. Coordinated and tailored work rehabilitation: a randomized controlled trial with economic evaluation undertaken with workers on sick leave due to musculoskeletal disorders. J Occup Rehabil. 2009;19(1):81-93.
25. Loisel P, Abenhaim L, Durand P, Esdaile JM, Suissa S, Gosselin L et al. A population-based, randomized clinical trial on back pain management. Spine (Phila Pa 1976). 1997;22(24):2911-8.
26. Marks EM, Chambers JB, Russell V, Bryan L, Hunter MS. The rapid access chest pain clinic: unmet distress and disability. QJM. 2014;107(6):429-34.
27. Stochkendahl MJ, Christensen HW, Vach W, Høilund-Carlsen PF, Haghfelt T, Hartvigsen J. Chiropractic treatment vs self-management in patients with acute chest pain: a randomized controlled trial of patients without acute coronary syndrome. J Manipulative Physio Ther. 2012;35(1):7-17.
28. Christensen HW, Vach W, Gichangi A, Manniche C, Haghfelt T, Høilund-Carlsen PF. Manual therapy for patients with stable angina pectoris: a nonrandomized open prospective trial. J Manipulative Physio Ther. 2005;28(9):654-61.
29. Murphy L, Helmick CG. The impact of osteoarthritis in the United States: a population-health perspective. Am J Nurs. 2012;112(3 Suppl 1):S13-9.

30. Cross M, Smith E, Hoy D, Nolte S, Ackerman I, Fransen M et al. The global burden of hip and knee osteoarthritis: estimates from the Global Burden of Disease 2010 study. Ann Rheum Dis.2014;73(7):1323-30.
31. Bombardier C, Hawker G, Mosher D. The impact of arthritis in Canada: today and the next 30 years. Toronto: Arthritis Alliance of Canada, 2011:1-51.
32. Nüesch E, Dieppe P, Reichenbach S, Williams S, Iff S, Juni P. All cause and disease specific mortality in patients with knee or hip osteoarthritis: population based cohort study. BMJ. 2011;342:d1165.
33. Franklin J, Ingvarsson T, Englund M, Ingimarsson O, Robertsson O, Lohmander LS. Natural history of radiographic hip osteoarthritis: A retrospective cohort study with 11-28 years of followup. Arthritis Care Res (Hoboken). 2011;63(5):689-95.
34. Paans N, van der Veen WJ, van der Meer K, Bulstra SK, van den Akker-Scheek I, Stevens M. Time spent in primary care for hip osteoarthritis patients once the diagnosis is set: a prospective observational study. BMC Fam Pract. 2011;12:48.
35. National Institute for Health and Care Excellence. Osteoarthritis: Care and management in adults (CG177). London: National Institute for Health and Care Excellence, 2014:1-36.
36. Fernandes L, Hagen KB, Bijlsma JW, Andreassen O, Christensen P, Conaghan PG et al. EULAR recommendations for the non-pharmacological core management of hip and knee osteoarthritis. Ann Rheum Dis. 2013;72(7):1125-35.
37. Zhang W, Moskowitz RW, Nuki G, Abramson S, Altman RD, Arden N et al. OARSI recommendations for the management of hip and knee osteoarthritis, Part II: OARSI evidence-based, expert consensus guidelines. Osteoarthritis Cartilage. 2008;16(2):137-62.
38. Abbott JH, Robertson MC, Chapple C, Pinto D, Wright AA, Leon de la Barra S et al. Manual therapy, exercise therapy, or both, in addition to usual care, for osteoarthritis of the hip or knee: a randomized controlled trial. 1: clinical effectiveness. Osteoarthritis Cartilage. 2013;21(4):525-34.
39. Brantingham JW, Parkin-Smith G, Cassa TK, Globe GA, Globe D, Pollard H et al. Full kinetic chain manual and manipulative therapy plus exercise compared with targeted manual and manipulative therapy plus exercise for symptomatic osteoarthritis of the hip: a randomized controlled trial. Arch Phys Med Rehabil. 2012;93(2):259-67.
40. Poulsen E, Hartvigsen J, Christensen HW, Roos EM, Vach W, Overgaard S. Patient education with or without manual therapy compared to a control group in patients with osteoarthritis of the hip. A proof-of-principle three-arm parallel group randomized clinical trial. Osteoarthritis Cartilage. 2013;21(10):1494-503.
41. Rathleff MS, Roos EM, Olesen JL, Rasmussen S. High prevalence of daily and multi-site pain – a cross-sectional population-based study among 3000 Danish adolescents. BMC Pediatr. 2013;13:191.
42. Aartun E, Hartvigsen J, Wedderkopp N, Hestbaek L. Spinal pain in adolescents: prevalence, incidence, and course: a school-based two-year prospective cohort study in 1,300 Danes aged 11-13. BMC Musculoskelet Disord. 2014;15:187.
43. Hestbaek L, Leboeuf-Yde C, Kyvik KO, Manniche C. The course of low back pain from adolescence to adulthood: eight-year follow-up of 9600 twins. Spine (Phila Pa 1976). 2006;31(4):468-72.
44. Hartvigsen J, Hestbaek L. Children and chiropractic care: a window of opportunity. J Manipulative Physio Ther. 2009;32(8):603-5.
45. Aartun E, Degerfalk A, Kentsdotter L, Hestbaek L. Screening of the spine in adolescents: inter- and intra-rater reliability and measurement error of commonly used clinical tests. BMC Musculoskelet Disord. 2014;15:37.
46. Wedderkopp N, Jespersen E, Franz C, Klakk H, Heidemann M, Christiansen C et al. Study protocol. The Childhood Health, Activity, and Motor Performance School Study Denmark (The CHAMPS-study DK). BMC Pediatr. 2012;12:128.
47. Brown R. Climate change: global challenges for the chiropractic profession. JCCA. 2013;57(2):106-10.
48. Menke JM. Do Manual Therapies Help Low Back Pain? A comparative effectiveness meta-analysis. Spine (Phila Pa 1976). 2014;39(7):E463-72.
49. Chou R, Qaseem A, Snow V, Casey D, Cross JT, Jr., Shekelle P et al. Diagnosis and treatment of low back pain: a joint clinical practice guideline from the American College of Physicians and the American Pain Society. Ann Intern Med. 2007;147(7):478-91.
50. Savigny P, Kuntze S, Watson P, Underwood M, Ritchie G, Cotterell M, et al. Low Back Pain: early management of persistent non-specific low back pain. London: National Collaborating Centre for Primary Care and Royal College of General Practitioner, 2009:1-209.
51. Foster NE, Dziedzic KS, van der Windt DA, Fritz JM, Hay EM. Research priorities for non-pharmacological therapies for common musculoskeletal problems: nationally and internationally agreed recommendations. BMC Musculoskelet Disord. 2009;10:3.

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Tuberculosis of the neuromusculoskeletal system: a review of two cases presenting as chiropractic patients

Ismat Kanga, BSc, DC, FCCS(C)[1]
John A. Taylor, DC, DACBR[2]
Craig Jacobs, MSc, DC, FCCS(C)[3]
Geoff Outerbridge, MSc, DC[4]

Tuberculosis caused by Mycobacterium tuberculosis is a major public heath problem world-wide, particularly in low-income countries. Increased number of immunocompromised patients and immigration from countries where tuberculosis is endemic has resulted in increased number of cases in high-income countries. Tuberculosis can affect any organ system, but is of particular interest to chiropractors when it affects the neuromusculoskeletal system. Patients with tuberculosis of the neuromusculoskeletal system can present with mechanical low back pain or with complex neurologic deficits. The aim of this paper is to highlight the importance of considering a diagnosis of tuberculosis in susceptible populations and the devastating consequences of the disease. The epidemiology, clinical

La tuberculose causée par le bacille de Koch représente un problème de santé publique important dans le monde, surtout dans les pays à faible revenu. Le nombre grandissant de patients immunovulnérables et l'immigration provenant de pays où la tuberculose est endémique entraînent une hausse des cas de maladie dans les pays à revenu élevé. La tuberculose peut toucher n'importe quel système d'organe, mais les chiropraticiens s'y intéressent lorsqu'elle affecte le système neuromusculosquelettique. Les patients atteints de tuberculose du système neuromusculosquelettique peuvent souffrir de douleurs lombaires mécaniques ou de déficits neurologiques complexes. L'objectif du présent article est de souligner l'importance de prendre en considération la possibilité d'un diagnostic de tuberculose chez les populations à risque, et les conséquences dévastatrices de cette maladie. On présente également l'épidémiologie et les caractéristiques cliniques de la tuberculose, ainsi que

[1] Department of Graduate Studies, Clinical Sciences, Canadian Memorial Chiropractic College, Toronto, Ontario, Canada
[2] Professor, Chiropractic Department, D'Youville College, Buffalo, NY
[3] Director, Clinical Education and Patient Care, Division of Clinical Education, Canadian Memorial Chiropractic College (CMCC), Toronto, Ontario, Canada
[4] Clinic Director, World Spine Care and Adjunct Faculty, Canadian Memorial Chiropractic College, Toronto, Ontario, Canada
Corresponding author: Ismat Kanga
ismat.kanga@gmail.com
T: (416) 482-2340 F: (416) 482-2560
6100 Leslie Street, Toronto, Ontario, Canada, M2H 3J1
Consent: Written consent was obtained from both patients to use information and images from their files for this case report.

features and management of tuberculosis will also be presented to facilitate early diagnosis, appropriate referral and multidisciplinary care of these patients.

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KEY WORDS: tuberculosis, tuberculous spondylodiscitis tuberculosis radiculomyelitis, HIV, chiropractic

la lutte contre celle-ci, afin de favoriser un diagnostic précoce, le choix de ressources appropriées et les soins multidisciplinaires donnés à ces patients.

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MOTS CLÉS : tuberculose, spondylodiscite tuberculeuse, radiculomyélite tuberculeuse, VIH, chiropratique

Introduction

It is estimated that 2.3 billion people worldwide are infected with tuberculosis (TB).[1] Despite advances in health care, TB continues to be a major public health problem worldwide, particularly in low-income countries.[1–3] Increased numbers of immunocompromised patients and immigration of people from countries where TB is endemic has resulted in a rise in the number of cases in high-income countries.[3,4] Tuberculosis caused by *Mycobacterium tuberculosis,* most commonly affects the lungs but can affect any organ system.[1–3] TB also affects the neuromusculoskeletal system. The spine is afflicted in 1 to 5% of all patients infected with TB and is the most common site, occurring in approximately 50% of the cases of skeletal TB.[2–5] TB of the spine or TB spondylodiscitis is also known as Pott's disease. It was named after Sir Percival Pott, a British surgeon who first described spinal TB and the surgical treatment of paravertebral abscesses in his monograph in 1779.[3,6,7] The central nervous system is also involved in approximately 10% of all patients with TB.[8]

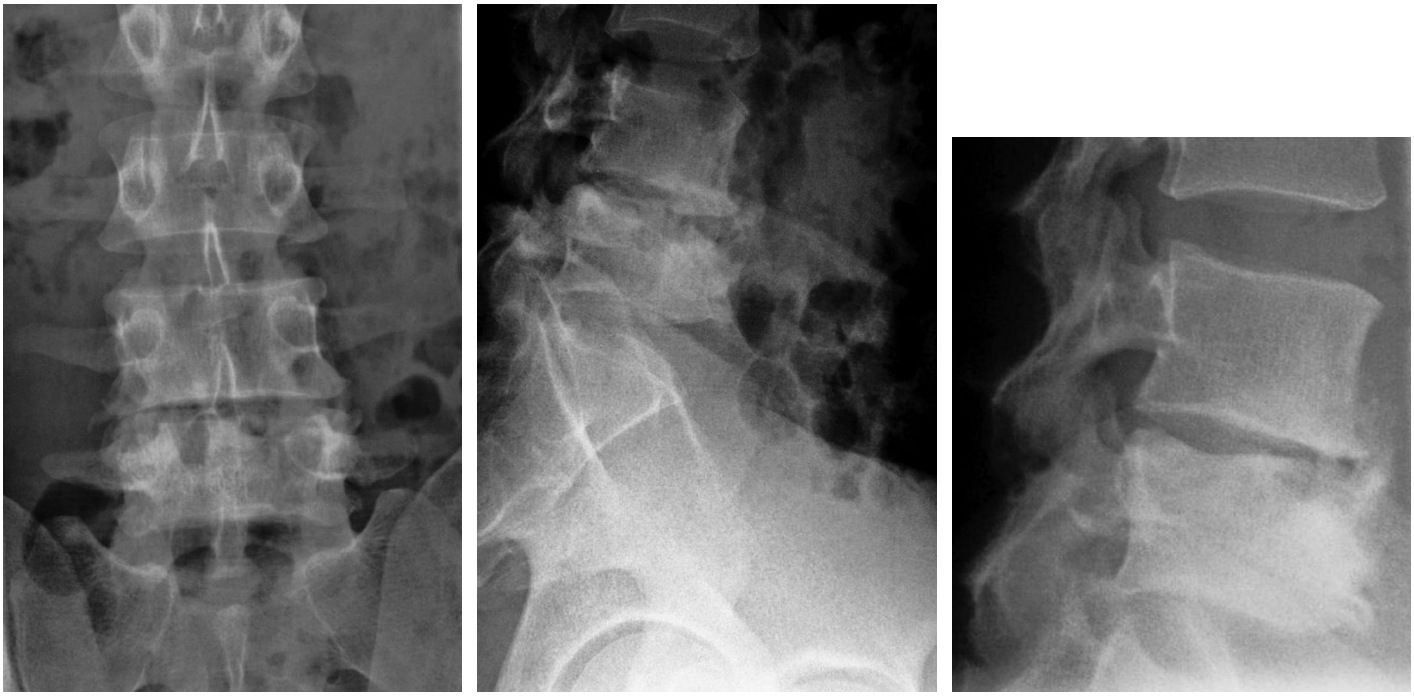
These two cases describe TB in two HIV-positive (Human immunodeficiency virus) patients. The first case chronicles a young male with a history of chronic low back pain, who presented to the chiropractic clinic for treatment. The second patient presented after TB involving the central nervous system (CNS) was diagnosed and treated. This patient suffered complex sequelae as a consequence of the infection resulting in paraparesis. The aim of this paper is to emphasize the importance of considering tuberculosis in the differential diagnosis in patients presenting with presumed mechanical back pain and also to remind chiropractors of the devastating consequences of the disease. The epidemiology, pathogenesis, imaging features and clinical presentation of TB will be presented in order to highlight the disease in order to facilitate appropriate management of these patients.

Cases:

Case 1

History: A 32-year-old male presented to the World Spine Care Clinic in Mahalapye, Botswana with a complaint of chronic low back pain. He attributed his pain to lowering a 50kg bag from waist height to the ground around 11 months prior. The onset of pain was immediate. He characterized the nature of his pain as a dull ache and a cramp down the back of his right thigh, but no neurologic symptoms were reported. On presentation, he reported constant pain and rated his pain as 10/10 on a scale of 0 to 10 (0 representing no pain at all and 10 being the worst pain ever). Aggravating factors included bending, lifting and prolonged sitting. Lying down helped reduce the intensity of pain somewhat. Prior to his attending the chiropractic clinic, previous treatment for his pain included analgesics (paracetamol), non-steroidal anti-inflammatory (diclofenac) injections and tricyclic antidepressant medication (amitriptyline). He reported no constitutional symptoms (fevers, weight loss or chills). Inquiry into his medical history revealed that he was diagnosed with HIV seven years ago. His last CD4 count, assessed a few months prior, was 465 cells per microliter (Normal range 500-1000 cells per microliter).

Physical examination: Examination revealed severely restricted and painful lumbar ranges of motion, most notably in rotation and flexion. Kemp's test was positive bilaterally. Intersegmental joint restrictions and tenderness were found in the lower lumbar spine from L3 to L5 and bilateral sacroiliac joints. Spinous percussion revealed tenderness at the lower lumbar levels. His lumbar



1A 1B 1C

Figure 1:
Patient 1. Frontal (A), and lateral (B, C) radiographs reveal severe L4-5 disc space narrowing, erosion and partial collapse of the anterior-superior and anterior-inferior L5 vertebral endplates and to a lesser extent the inferior L4 endplate, and prominent sclerosis of the L5 vertebral body.

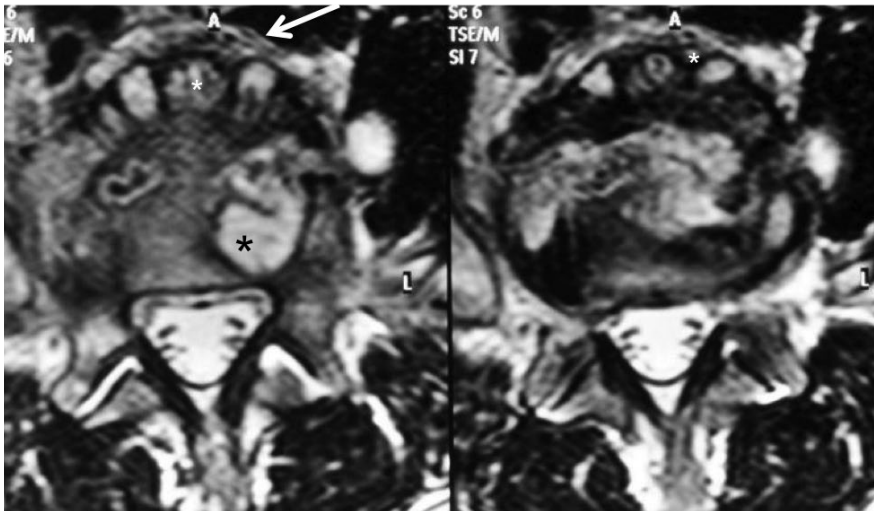
spine, gluteal and posterior thigh musculature were tender to direct palpation. Upper and lower limb neurologic examination including reflexes, motor and sensory examination were within normal limits.

Diagnostic Imaging: The patient brought in radiographs, which revealed severe disc space narrowing at L4-5 with erosion and partial collapse of the anterior-superior and anterior-inferior endplates of the L5 vertebral body, and to a lesser extent the inferior endplate of L4. In addition, prominent sclerosis of the L5 vertebral body was also evident. (Figure 1) Based on radiographic findings consistent with infectious spondylodiscitis, the patient was referred to an orthopedic surgeon. The patient was started on immediate anti-tubercular therapy and referred for magnetic resonance imaging (MRI) of the lumbar spine.

An MRI examination was performed two weeks later. The fluid-sensitive (T2-weighted) images revealed extensive high signal intensity with anterior destruction and collapse of the L4 and L5 vertebral bodies. In addition, the L4-5 disc space was destroyed and a hyperintense inflammatory abscess extended into the anterior soft tissues coursing in a subligamentous distribution resulting in bulging of the anterior longitudinal ligament anteriorly. (Figures 2A, B, C) T1-weighted images obtained following intravenous injection of gadolinium revealed significant enhancement of the subligamentous abscess. (Figure 2D) The patient history combined with imaging findings confirmed the presence of tuberculous spondylodiscitis of the spine (Pott's spine).

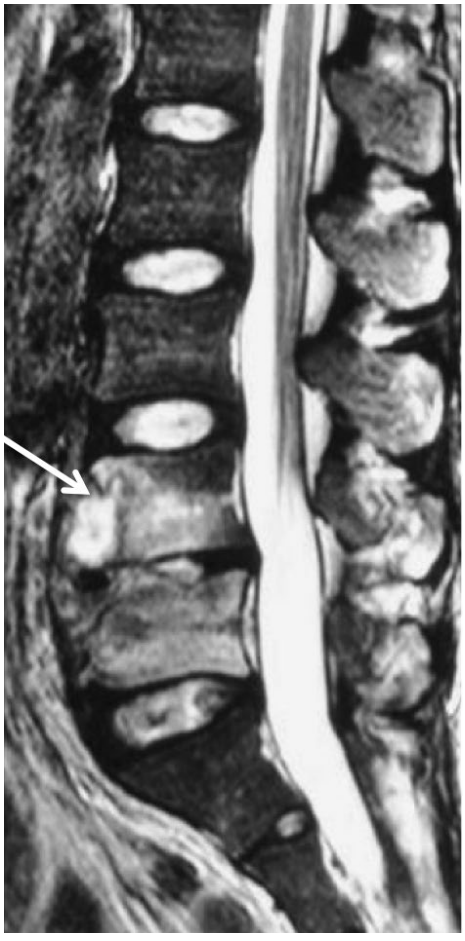
Case 2

History: A 40-year old male was admitted to a Toronto area hospital with a fever and postictal (altered consciousness) status after having a seizure. Serological testing revealed his serum Venereal Disease Research Laboratory (VDRL) test and Fluorescent Treponemal Antibody-Absorption (FTA-ABS) test were positive,



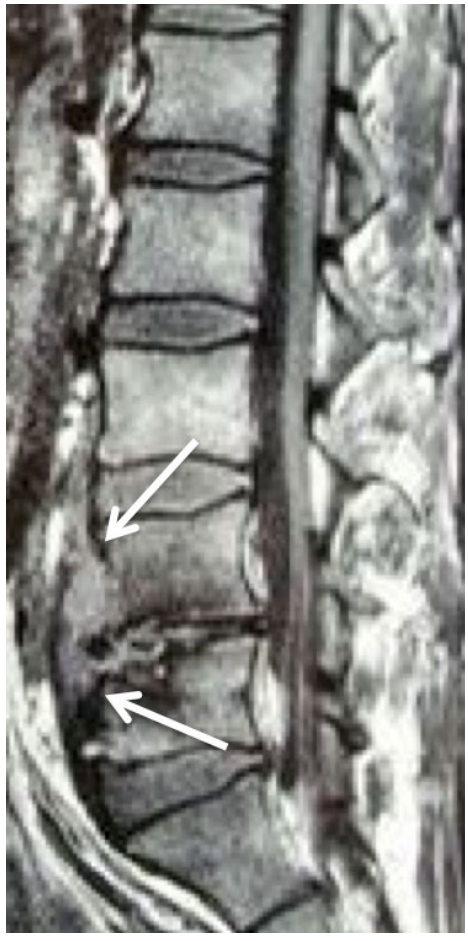
2A

Figure 2:
Patient 1. Fluid-sensitive (T2-weighted) axial (A) and sagittal (B,C) images show extensive high signal intensity (asterisk) with anterior destruction and collapse of the L4 and L5 vertebral endplates, L4-5 disc space destruction, and a hyperintense abscess extending into the anterior soft tissues coursing in a subligamentous distribution resulting in bulging of the anterior longitudinal ligament anteriorly (arrow). T1-weighted images obtained following intravenous injection of gadolinium (D) reveals significant enhancement of the abscess (arrows) and clearly defines its subligamentous location.



2B

2C



2D

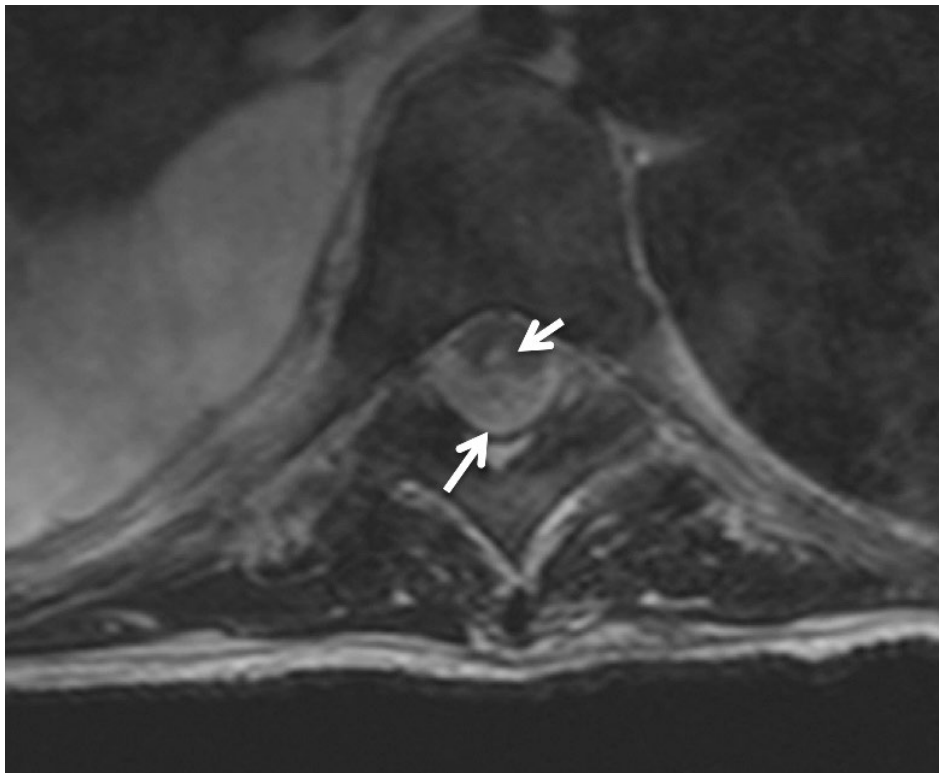


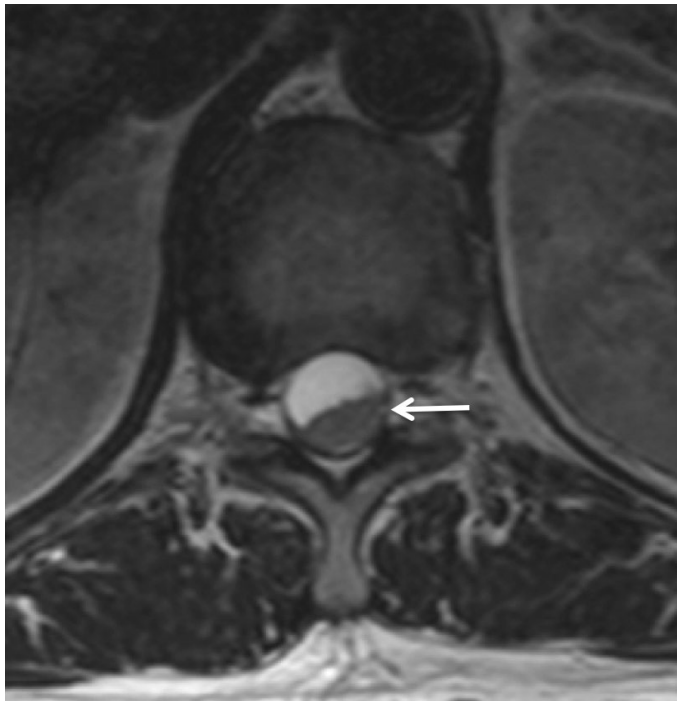
Figure 3:
Patient 2. T2-weighted sagittal image (A) reveals a large linear plaque (arrows) on the dorsal surface of the cord extending from the T3 to T11 segments, resulting in diffuse ventral deviation and cord compression. The T2-weighted axial image (B) shows increased signal intensity in the cord (arrows) due to edema or ischemia.

3A 3B

indicating neurosyphilis. At that time he was also diagnosed with HIV with a CD4 count of 77 cells per microliter. Two weeks later, while still at the hospital the patient developed shortness of breath. He was found to have pleural effusion and his pleural fluid tested positive for Acid-Fast Bacilli, confirming pulmonary tuberculosis. He was started on anti-tubercular therapy and antiretroviral medications. During his hospital stay, he started to experience weakness in his lower extremities and subsequently developed a neurogenic bladder. MRI revealed a large plaque located on the dorsal surface of the spinal cord extending from T3 to T11 and increased signal intensity of the cord suggesting edema or ischemia. (Figures 3A, B) Multiple small tubercular granulomas were also visualized on the surface of the brain and cervical spinal cord. The MRI also showed assimilation of the odontoid process of C2 and the C1 vertebral body. The patient suffered neurologic sequelae from his disseminated TB including TB meningitis, TB arachnoiditis and TB granulomata.

Follow-up one year after revealed severe pyramidal weakness bilaterally. Hip flexors were graded as 2/5, hamstrings 4/5 and ankle dorsiflexors 1/5. Other neurologic findings included reduced proprioception and vibration sense at the ankles and sensory loss over the surface of the thoracic cage. A repeat MRI revealed anterior displacement of the superior thoracic cord and posterior displacement of the inferior thoracic cord. The T2-weighted sequences revealed a hyperintense signal with alteration

4A



4B

Figure 4:
Patient 2. T2-weighted (A) sagittal image reveals anterior displacement of the cord at upper segments of the thoracic with posterior displacement of the lower thoracic cord with hyperintensity and alteration in the contour of the cord (arrows). Axial T2-weighted image (B) shows posterior crowding of the cauda equina.

of the contour of the cord, adhesions and formation of CSF pockets. (Figures 4 A, B)

This patient presented to a Canadian Memorial Chiropractic College teaching clinic four years after his disseminated TB, with complaints of neck pain, low back pain and headaches. His neck pain was also associated with radiation of pain into his left arm and into the third digit. He attributed the onset of his pain to his long hospitalization associated with his TB. He reported the pain was constant and rated the intensity of his neck pain as a 10 out of 10. Sitting for prolonged periods aggravated his low back pain, while movement of his neck aggravated his neck pain. Hot showers, acupuncture and manual therapy were reported to relieve his pain. His medical history is significant for Type 2 diabetes, HIV, neurogenic bladder, obstructive sleep apnea, epistaxis, dyslipidemia, hypertension, cognitive impairment and anemia. Past medical history was significant for neurosyphilis with seizures, gastritis and acute respiratory distress syndrome.

Physical Examination: Observation revealed a paretic gait. Active ranges of motion testing in his cervical and lumbar spine were painful and restricted by 25% globally. Upper limb neurologic examination revealed normal sensation, motor strength and deep tendon reflexes (DTRs). Hoffman's test (flicking terminal phalanx of the third or fourth digits) was positive bilaterally. Lower limb DTRs revealed 3+ bilaterally at L4 and 1+ bilaterally at S1. Motor strength was diminished and was rated as 3/5 at L2 and 4/5 in L3-S1 bilaterally. Sensation, plantar reflex and clonus were within normal limits. Tenderness was noted

Figure 5:
Patient 2. Lateral cervical radiograph reveals assimilation of a C2 os odontoideum with the C1 anterior tubercle, degenerative joint and disc disease and lateral stenosis from C5 through C7.

bilaterally in his lumbar and cervical paraspinal, trapezius and levator scapulae muscles.

Diagnostic Imaging: Radiographs of his cervical spine revealed mild cervical degenerative disc disease and moderate foraminal stenosis at the C5-7 levels. The atlantoaxial articulation was anomalous with an apparent os odontoideum and assimilation of the odontoid process ossicle with the anterior tubercle of C1. (Figure 5) Flexion-extension radiographs revealed marked segmental instability with 6 mm of translation of this anomalous C1 and C2 ossicle in relation to the C2 vertebral body.

Diagnosis: The patient was diagnosed with mechanical low back pain, degenerative disc and joint disease and lateral stenosis from C5 to C7, in addition to his persistent neurologic deficits as a result of his TB, neurosyphilis and HIV.

Chiropractic Management: The patient was treated with myofascial release of the cervical and lumbar paraspinal muscles, mobilization of the lumbar spine and active rehabilitation including neck and lower limb isometric strengthening and education. The patient responded well to chiropractic care and noticed significant improvement in his strength. Though still requiring a wheelchair for a significant amount of the time, he was able to use a walker for some activities of daily living and accomplish his daily hygiene routine without assistance.

Discussion:

Epidemiology

The global prevalence of TB is increasing at a rate of 1.1 % per year.[2,9] TB kills 1.4 to 2 million people every year and is second only to HIV as the leading cause of death from infectious disease.[1,3] Countries in Africa and South-East Asia have the greatest burden of disease globally.[10] Immigration from these regions represents the majority of cases in high-income countries. Risk factors for TB include advanced age, malnutrition, poor sanitation, homelessness, alcohol and substance abuse, immunocompromised states and diabetes.[2,3,11] Infection occurs through the respiratory tract, by inhalation of droplet nuclei. Individuals with active pulmonary TB spread droplet nuclei when coughing, sneezing or talking. Droplet nuclei are able to penetrate the alveoli, where they multiply before spreading to various organ systems.[2]

Association with Immunocompromised patients

Immunocompromised patients, particularly those with HIV are at a higher risk of infection with TB, reactivating latent TB and also acquiring a concurrent pyogenic infection.[3] Of the nine million new cases of active TB annually, 13 to 15% of all cases are associated with HIV.[10,12] The incidence rates of TB in patients with AIDS (Acquired Immunodeficiency Syndrome) are 500 times greater than in HIV-negative patients.[3] HIV results in dysfunction of macrophage, monocyte and lymphocyte function, which play a pivotal role in the body's defense against *Mycobacterium tuberculosis*.[13] Patients with a CD4 count of less than 500 per microliter and total lymphocyte counts of between 1000-2000 per microliter have a comprom-

ised immune system and are at greater risk of infection with TB.[13] It should also be noted that certain patients might be immunocompromised as a result of iatrogenic influences. The immunoinhibitory effects of medications and biologic substances such as corticosteroids and anti-tumour necrosis factor (TNF) biologics are well documented.[14] Anti-TNF-α agents (infliximab, etanercept and adalimumab), used in the treatment of rheumatoid arthritis, ankylosing spondylitis, psoriatic arthritis and juvenile idiopathic arthritis increase the risk of TB and should be considered in the etiology of tuberculosis infections.[15]

Tuberculous spondylodiscitis

Pathogenesis: The spread of TB to the vertebral column occurs as a result of haematogenous dissemination via the Batson's venous plexus or lymphatic drainage via the para-aortic lymph nodes.[4,11,16] Once it has reached the vertebral column the infection usually arises in the anterior portion of the body.[9] The tubercle bacillus begins gradual trabecular destruction and demineralization, progressing to the cortex, adjacent disc and vertebral body. Within the vertebral body a granulomatous lesion develops, comprised of leukocytes, caseous material, bone debris and tubercle bacilli.[4,11,16] The collapse and wedging of multiple adjacent vertebral bodies generate the characteristic gibbus deformity.[4,5] Paraspinal abscesses are located in 70% of patients. Psoas abscesses are common and can extend deep to the iliopsoas fascia into the proximal thigh, resulting in a groin mass.[11,17] Differential diagnoses for TB spondylodiscitis include pyogenic and fungal infections, brucellosis, compression fractures and metastatic disease.[9,11]

Clinical Presentation: TB spondylodiscitis has an insidious onset of symptoms and slow progression, making early diagnosis of TB fundamentally important in preventing deformity and paraplegia. The mean duration between infection and clinical presentation can range from a few days to three years.[11,18] The typical presentation of TB spondylodiscitis is low back pain over the affected vertebral bodies, low-grade fever, chills, weight loss.[16] Clinical examination may reveal local tenderness and limitation in spinal motion.[11] Neurologic complications such as radicular symptoms, cauda equina and paraplegia can arise from edema, vascular engorgement and retropulsed debris from vertebral body collapse.[11]

Diagnostic imaging features of tuberculous spondylodiscitis: TB spondylodiscitis occurs most frequently in the lower thoracic and thoracolumbar spine.[9,11,16,18] Conventional radiographs are not sensitive in detecting early TB of the spinal column, as vertebral body destruction is not visualized until 50% of the trabeculae have been destroyed, corresponding to approximately six months through the course of the disease.[5,11] At later stages in the disease, loss of vertebral height, disc space narrowing, erosions, paravertebral masses and vertebral body collapse may be visualized on the radiographs.[5,11] Paravertebral abscesses are often visualized as areas of soft tissue swelling adjacent to the spine.[19] Changes on computed tomography (CT) and MRI are detectable as early as six weeks after infection.[4] CT scans are particularly useful in revealing destruction of the vertebral endplates, fragmentation of the vertebral body, paravertebral calcifications and in facilitating image-guided biopsy to confirm a diagnosis.[5,11] Calcification within the paraspinal abscess, seen on CT images are pathognomonic for TB.[11] TB spondylodiscitis can also present as isolated involvement of the posterior elements, infection limited to one vertebral segment or multiple nonadjacent vertebrae and an ivory vertebrae.[11,17] MRI is most sensitive in revealing early changes within the bone and endplate and in demonstrating the extent of spinal cord compromise.[5,11] TB lesions appear as low signal intensity on T1-weighted images and heterogeneous increased signal on T2-weighted images.[11]

Tuberculosis of the Central Nervous System (CNS).

TB of the CNS arises from haematogenous spread secondary to disease in another part of the body. CNS TB is the most dangerous form of systemic TB, responsible for high mortality and severe disability.[20] Small tuberculous lesions or Rich's foci develop in the meninges, surface of the brain or spinal cord. Growth or rupture of these lesions results in the numerous types of CNS tuberculosis.[8] Tuberculous meningitis (TBM) is the most common presentation of CNS tuberculosis.[20] The clinical presentation of TBM includes fever, headache and altered level of consciousness and other meningeal signs such as neck stiffness, photophobia and vomiting.[20]

Spinal tuberculous arachnoiditis, more commonly known as tuberculous radiculomyelitis (TBRM) is a rare complication of tuberculous meningitis and should be considered when a TBM patient develops spinal cord symptoms.[21,22] TBRM is an inflammatory condition in-

volving the arachnoid lining or leptomeninges of the spinal cord.[21]

Pathogenesis: Three separate mechanisms may be responsible for the development of TBRM. It may result from 1. a primary tuberculosis lesion developed in the central nervous system; 2. direct extension from the vertebral body; or 3. descent from intracranial TB meningitis (most common).[21,22] The thoracic spine is most frequently involved, and less often the lumbar and cervical spine.[22,23] TBRM is characterized by a gross granulomatous reaction with associated histiocytic proliferation, caseation and tubercle formation that expands and fills the space between the spinal dura mater and the leptomeninges.[21] It often extends over several segments, envelops the spinal cord and compresses on it and the nerve roots, but does not penetrate into either.[8,20–22] The neurologic symptoms experienced by patients occur as a result of direct compression of the cord and ischemia.[8] Ischemia often develops in the spinal arteries as a result of vasculitis and thrombosis.[20,21] Within the parenchyma of the cord syringomyelia and myelomalacia may develop as a result of vacuolization, atrophy, and central necrosis.[21,22]

Clinical Presentation: TBRM may manifest at any point after infection with TB meningitis. Clinical presentation of TBRM includes paraparesis, paraplegia, radicular pain, paresthesias, muscle atrophy and neurogenic bladder.[21,22] Absence of deep tendon reflexes, weakness in the lower limbs and upgoing plantar response are also commonly seen.[21] Diagnosis is based on clinical features, presence of tuberculous meningitis, CSF analysis and advanced imaging.[24] CSF analysis displays an active inflammatory response with lymphocytosis, decreased glucose content and abnormally high protein levels.[21]

Diagnostic imaging features of tuberculosis radiculomyelitis: Loculation and destruction of the subarachnoid space, loss of contour of the spinal cord, myelomalacia and adhesions (thickening, clumping or matting) of the nerve roots particularly in the lumbar spine are common MRI findings of TBRM.[21] TBRM on T1-weighted images display increased intensity of the CSF fluid and loss of the contour of the spinal cord, due to increased protein within the CSF. Adhesions of the nerve roots may also be visualized. Fluid-sensitive T2-weighted images display increased signal intensities within the cord as a result of myelitis, edema and ischemia.[23] During the chronic phase of the infection, MRI sequences may not exhibit enhancement but continue to show signs of arachnoiditis such as the adhesions of the nerve roots. Development of syringomyelia and myelomalacia can also be clearly visualized on MRI.[21,22]

Management

Treatment for TB includes a combination of anti-tubercular medications including isoniazid, rifampin, pyrazinamide, ethambutol, streptomycin, ethionamide and cycloserine, administered over a span of 12 to 13 months.[25] Patients with HIV treated with a course of anti-tubercular drugs display the same clinical, radiological and microbiological response as HIV-negative patients.[2] Surgery was previously utilized as a treatment for TB, however it is now used more sparingly and selectively. It is used less for controlling disease and more for reducing or rectifying spinal deformities and neurologic complications.[3] The use of anti-tubercular drugs has resulted in multidrug resistant strains creating a further complication in the management of TB.[26] The first patient presented in the late of stage of infection, after significant vertebral body destruction, but demonstrating only symptoms of mechanical low back pain. He had substantial lower back pain, tenderness and restriction in range of motion but no signs of TB such as weight loss, fever, chills or neurologic symptoms. The diagnosis of TB can easily be missed, especially in patients at early stages of infection with similar signs and symptoms to those of our patient, particularly in the absence of constitutional symptoms or radiographic findings of infection. When patients present to chiropractors with low back pain, infection, both pyogenic and non-pyogenic should be considered in the differential diagnosis and should be ruled out during history and physical examination. Red flags suggesting a more serious underlying condition should also be excluded. Any history of constant progressive pain, past history of cancer, trauma, prolonged use of corticosteroids, unexplained weight loss, fever and immunocompromised status warrants further investigation.[27]

Our first patient presented with constant pain and HIV, which were subtle indicators that raised the suspicion of a non-mechanical cause of low back pain. The second patient presented with musculoskeletal pain years after disseminated TB. Though the patient suffered from the neurosyphillis prior to the onset of TB, his clinical presentation and diagnostic imaging findings suggested TB

radiculomyelitis as the principal cause of his neurologic deficits. This case is an excellent reminder that TB is a world-wide epidemic and can result in death and disability. It is a common differential consideration in low-income countries, however its recognition is difficult in high-income nations. Because chiropractors encounter it so infrequently, it is often overlooked as a cause of back pain and often mistaken for metastasis or simple compression fracture owing to similar clinical presentation and imaging findings.[16] Tuberculous spondylitis presenting as mechanical neck pain has been previously described the in the literature.[28] A 21 year-old male presented to a chiropractic office with neck pain, stiffness and difficulty swallowing. Cervical radiographs taken were unremarkable, and the patient was treated with spinal manipulation, trigger point therapy and stretching. Though the patient regained all active ranges of motion, the treatment was unable to alleviate the patients' persistent tenderness in his suboccipital region. As a result the patient was referred to a physiatrist and subsequently hospitalized with a presumptive diagnosis of tuberculous spondylitis. [28]

Despite the low prevalence of TB in North America, these patients – especially immigrants and immunocompromised patients – may indeed present to our offices. Patients with TB spondylodiscitis present with tenderness, hypertonicity of the paraspinal muscles and limited mobility. Soft tissue therapy, education and mobilization/manipulation of unaffected vertebral bodies may be safely applied. Affected vertebral bodies have compromised integrity and are a contra-indication to high-velocity low-amplitude spinal manipulation. Patients who are afflicted with CNS TB despite early treatment are often left with neurologic deficits including paraparesis and paraplegia. Rehabilitation is important for these patients to maintain and improve strength and to treat soft tissue contractures. Patients are treated with anti-tubercular drugs for a period of a year or longer and therefore may even present to our clinics while being on concurrent treatment. It would be important for chiropractors to assist in monitoring these patients for disease progression or onset of new symptoms and collaborate with other health care professionals in the care of these patients.

Summary:

It is important to include TB as a differential diagnosis in elderly and immunocompromised patients and immigrants from areas where TB is endemic. As it is uncommon in high-income countries, its recognition is often difficult and often presents with similar signs and symptoms as mechanical low back pain, metastasis or compression fractures. TB may affect multiple organ systems within the body, but chiropractors should be aware of the clinical presentation of neuromusculoskeletal TB. Presentation of these two cases aims to heighten the awareness of the global burden of disease and remind chiropractors that TB may present in high-income nations as well. Furthermore, these cases emphasize the role of chiropractors in the diagnosis and management of the disease.

References

1. CDC Grand Rounds: the TB/HIV syndemic. MMWR Morb Mortal Wkly Rep. 2012;61(26):484–9.
2. Harries AD, Dye C. Tuberculosis. Ann Trop Med Parasitol. 2014;100(5-6):415–31.
3. Tuli SM. Tuberculosis of the spine: a historical review. Clin Orthop Relat Res. 2007;460:29–38.
4. Tuli SM. General principles of osteoarticular tuberculosis. Clin Orthop Relat Res. 2002;(398):11–9.
5. Moore SL, Rafii M. Imaging of musculoskeletal and spinal tuberculosis. Radiol Clin North Am. 2001;39(2):329–42.
6. Dass B, Puet TA, Watanakunakorn C. Tuberculosis of the spine (Pott's disease) presenting as "compression fractures". Spinal Cord. 2002;40(11):604–8.
7. Garg RK, Somvanshi DS. Spinal tuberculosis: a review. J Spinal Cord Med. 2011;34(5):440–54.
8. Garg RK. Tuberculosis of the central nervous system. Postgrad Med J. 1999;75(881):133–40.
9. Burrill J, Williams CJ, Bain G, Conder G, Hine AL, Misra RR. Tuberculosis: a radiologic review. Radiographics. 2007;27(5):1255–73.
10. Zumla A, Raviglione M, Hafner R, von Reyn CF. Tuberculosis. N Engl J Med. 2013;368(8):745–55.
11. De Backer AI, Mortelé KJ, Vanschoubroeck IJ, Deeren D, Vanhoenacker FM, De Keulenaer BL, et al. Tuberculosis of the spine: CT and MR imaging features. JBR-BTR. 2005;88(2):92–7.
12. Hanekom WA, Lawn SD, Dheda K, Whitelaw A. Tuberculosis research update. Trop Med Int Health. 2010 Aug;15(8):981–9.
13. Jellis JE. Human immunodeficiency virus and osteoarticular tuberculosis. Clin Orthop Relat Res. 2002;(398):27–31.
14. Delogu G, Goletti D. The spectrum of tuberculosis infection: new perspectives in the era of biologics. J Rheumatol Suppl. 2014;91:11–6.
15. Cantini F, Niccoli L, Goletti D. Tuberculosis risk in patients treated with non-anti-tumor necrosis factor-α (TNF-α) targeted biologics and recently licensed TNF-α

inhibitors: data from clinical trials and national registries. J Rheumatol Suppl. 2014;91:56–64.
16. Dass B, Puet T a, Watanakunakorn C. Tuberculosis of the spine (Pott's disease) presenting as "compression fractures". Spinal Cord. 2002;40(11):604–8.
17. Griffith JF, Kumta SM, Leung PC, Cheng JCY, Chow LTC, Metreweli C. Imaging of musculoskeletal tuberculosis: a new look at an old disease. Clin Orthop Relat Res. 2002;(398):32–9.
18. Abou-Raya S, Abou-Raya A. Spinal tuberculosis: overlooked? J Intern Med. 2006;260(2):160–3.
19. Shanley DJ. Tuberculosis of the spine: imaging features. AJR Am J Roentgenol. 1995;164(3):659–64.
20. Bernaerts A, Vanhoenacker FM, Parizel PM, Van Goethem JWM, Van Altena R, Laridon A, et al. Tuberculosis of the central nervous system: overview of neuroradiological findings. Eur Radiol. 2003;13(8):1876–90.
21. Hernández-Albújar S, Arribas JR, Royo A, González-García JJ, Peña JM, Vázquez JJ. Tuberculous radiculomyelitis complicating tuberculous meningitis: case report and review. Clin Infect Dis. 2000;30(6):915–21.
22. Chotmongkol V, Kitkuandee A, Limpawattana P. Tuberculous radiculomyelitis (arachnoiditis) associated with tuberculous meningitis. Southeast Asian J Trop Med Public Health [Internet]. 2005;36(3):722–4.
23. Du Plessis J, Andronikou S, Theron S, Wieselthaler N, Hayes M. Unusual forms of spinal tuberculosis. Childs Nerv Syst [Internet]. 2008;24(4):453–7.
24. Sharma A, Goyal M, Mishra NK, Gupta V, Gaikwad SB. MR imaging of tubercular spinal arachnoiditis. AJR Am J Roentgenol [Internet]. 1997;168(3):807–12.
25. Huelskamp L, Anderson S, Bernhardt M. TB of the spine: Pott's disease. Orthop Nurs. 2000;19(4):31–5.
26. Nathanson E, Nunn P, Uplekar M. MDR tuberculosis – critical steps for prevention and control. NEJM. 2010;363(11):1050-8
27. Van Tulder M, Becker A, Bekkering T, Breen A, del Real MTG, Hutchinson A, et al. Chapter 3. European guidelines for the management of acute nonspecific low back pain in primary care. Eur Spine J. 2006;15 Suppl 2:S169–91.
28. Thiel H, Gotlib A. Tuberculous Spondylitis: a case report. J Can Chiropr Assoc. 1986;29(2):139–42.

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Evidence-based prognostication in a case of sciatica

Peter C. Emary, BSc, DC[1,2]

Objective: *To present an evidence-based case report on the prognosis of a patient with sciatica.*

Case: *A 43-year-old man presented with right-sided buttock and lower extremity pain and numbness of 10 weeks' duration. Magnetic resonance imaging revealed a lumbosacral disc herniation. Straight leg raise testing provoked the patient's right sciatic pain, and neurologic examination revealed a diminished right Achilles tendon reflex and mild hypoesthesia along the patient's outer right foot.*

Outcome: *PubMed was searched and two cohort studies relevant to sciatic prognosis were found. These articles were critically appraised for their validity, importance, and applicability in making a prognostic estimate for this particular patient. Based on the appraised research evidence, and the confidence intervals calculated therein, the overall prognosis for sciatic pain recovery with conservative care was estimated as favourable for this patient, though sensory recovery (even with surgical care) was not.*

Summary: *This case report illustrates how to use research literature in estimating the clinical prognosis*

Objectif : *Présenter une étude de cas fondée sur des éléments probants au sujet du pronostic d'un patient souffrant de sciatique.*

Cas : *Un homme de 43 ans souffre de douleurs et d'engourdissements au niveau de la fesse droite et des membres inférieurs depuis dix semaines. L'imagerie par résonance magnétique permet de constater une hernie discale lombo-sacrée. Lorsqu'on lève la jambe, tenue droite, du patient, le mouvement provoque chez lui une douleur sciatique, et un examen neurologique permet de constater une perte de réflexe au niveau du tendon d'Achille droit et une légère hypoesthésie sur l'extérieur du pied droit.*

Résultat : *À la suite d'une recherche dans PubMed, on a trouvé deux études par cohortes pertinentes sur le pronostic sciatique. Ces études ont fait l'objet d'un examen critique afin de déterminer leur validité, leur importance et leur applicabilité en matière de pronostic pour ce patient. Selon les preuves de recherche évaluées et les intervalles de confiance calculés, on a estimé le pronostic global de soulagement de la douleur sciatique au moyen de traitements conservateurs comme favorable pour ce patient, mais c'est l'inverse pour le rétablissement sensoriel (même avec des soins chirurgicaux).*

Résumé : *Dans cette étude de cas, on montre la façon d'utiliser les ouvrages sur la recherche pour établir un pronostic clinique d'un patient, et l'utilité de cette*

[1] Master of Science (MSc) Candidate, MSc Advanced Professional Practice (Clinical Sciences), Anglo-European College of Chiropractic, 13-15 Parkwood Road, Bournemouth, Dorset, BH5 2DF, United Kingdom
[2] Private Practice, 201C Preston Parkway, Cambridge, ON, N3H 5E8, Canada
Phone: 519-653-2101. E-mail: pcemary@hotmail.com

Consent: The patient has provided written consent to having his personal health information, including radiographs, published.

for an individual patient, and how this can be useful towards clinical decision-making concerning treatment.

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KEY WORDS: sciatica, prognosis, chiropractic, evidence based practice, case reports

approche par rapport aux décisions cliniques relatives au traitement.

(JCCA 2015; 59(1):24-29)

MOTS CLÉS : sciatique, pronostic, chiropratique, pratique basée sur des données probantes, études de cas

Introduction

Sciatica is a disorder characterized by radiating leg pain that follows a dermatomal pattern, accompanied at times by sensory symptoms.[1] In about 90% of cases, it is caused by a herniated disc with nerve root compression, and generally is considered to have a favourable prognosis.[1] However, most prognostic estimates of sciatica are based on data from individual studies, as systematic reviews on this topic are scarce. Moreover, evidence regarding specific prognostic factors for sciatic recovery, particularly in non-surgically treated patients, is conflicting.[2] For the chiropractor, communicating patient prognoses is nevertheless a routine part of proper informed consent procedure.[3] Presented here in an 'evidence-based' format,[4] this case report will chronicle how research literature was used in estimating the clinical prognosis of a patient with a lumbar disc herniation and sciatica.

Case Report

A 43-year-old man presented with a chief complaint of right-sided buttock and lower extremity pain of 10 weeks' duration. It began two days after working out at the gym. The pain was constant and described as if the nerves in his entire leg from the buttock down were being "pulled apart," accompanied by "numbness" along his right posterolateral thigh, lower leg, and foot. The pain

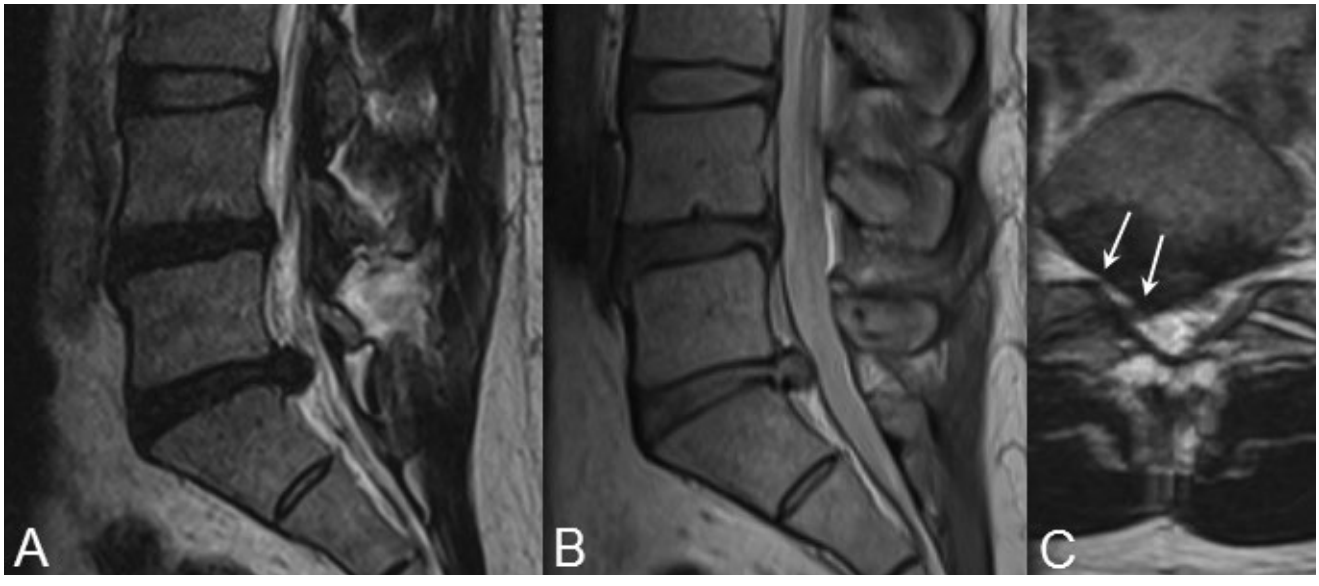

Figure 1.

*T2-weighted **(A)** and proton density **(B)** sagittal MR images of the patient's lower lumbar spine showing a contained intervertebral disc herniation at L5-S1. Mild-to-moderate disc dessication is also evident at both the L4-5 and L5-S1 levels. **(C)** T2-weighted coronal spot view of L5-S1 revealing a right paracentral disc protrusion, resulting in both displacement of the right S1 nerve root and intervertebral foraminal encroachment at L5-S1 on the right **(arrows)**.*

severity was graded as a seven out of 10; and his overall Bournemouth Questionnaire[5] score totalled 40 out of 70, where zero equals no disability and 70 equals complete disability. The buttock and lower extremity symptoms were most intense first thing in the morning (e.g. when getting out of bed). Standing up and walking around was somewhat palliative. Other provocative features included prolonged sitting and/or twisting movements (e.g. turning over in bed and getting in/out of a car). Two weeks earlier, a magnetic resonance imaging (MRI) study had been performed, revealing a lumbosacral disc herniation with impingement of the right S1 nerve root (Figure 1). Because heat therapy and over-the-counter anti-inflammatories (Ibuprofen) had not provided any relief, his nurse practitioner had prescribed ice and a stronger anti-inflammatory drug (Celebrex), and referred him for chiropractic treatment.

On examination, the patient's lumbar spine range of motion was normal, except for some mild right-sided low back pain experienced during seated extension. Prone motion palpation of the lumbosacral and sacroiliac joints revealed bilateral hypomobility, along with pain and gluteal muscle spasm on the right. Hypertonicity was also noted within the right quadratus lumborum muscle. The Straight Leg Raise test[6] (at approximately 30° of hip flexion) and the Yeoman's test each elicited pain and paraesthesia down the patient's right leg, while the Well Leg Raise, Double Leg Raise, Nachlas', and Hibb's tests were negative. Lower limb neurologic examination also revealed a diminished Achilles tendon reflex on the right (graded as +1), as well as mild hypoesthesia along the patient's outer right foot (i.e. S1 dermatome). Based on his age, history, physical exam, and MRI findings, the working diagnosis was a right-sided lumbosacral disc herniation with sciatica.

Clinical Question

Cognitive dissonance refers to the mental and emotional responses experienced by a clinician when a patient scenario calls for knowledge that he/she does not possess.[7] In this case, the author was uncertain about the prognosis for recovery in a patient with a lumbar disc herniation and sciatica. Clinical experience had taught that the natural course for this condition was variable – some patients improved, while others did not. Counselling such patients on expected outcomes, therefore, had often proved difficult. Adding to this uncertainty was the fact that the current patient had already been suffering with sciatica for 10 weeks. As a consequence, what was the likelihood that his symptoms would resolve? How long would it take? And would conservative treatment help, or would he require surgery? To answer these questions using an evidence-based approach, a more focused clinical question was needed.[7] Because the main issue here was regarding prognosis, the following foreground question was posed: *In a 43-year-old man with a lumbar disc herniation and sciatica, what is his prognosis for recovery with non-surgical/conservative care?*

Using the mnemonic, *PICO* (i.e. **P**atient/problem, **I**ntervention, **C**omparison, and **O**utcome[s] of interest),[7] this question can be broken down as follows:

P = A 43-year-old man with sciatica.
I = Non-surgical/conservative care.
C = Natural course.
O = Prognosis for recovery.

Literature Search

The best evidence to answer a clinical question about prognosis is a systematic review of cohort studies.[8] To begin, therefore, a search of the English language literature (from inception to March 5, 2014; limited to human studies) was conducted using the PubMed database. The medical subject headings '*sciatica*' and '*prognosis*' were combined using the Boolean operator '*AND*', yielding 392 hits. After filtering the search to '*systematic reviews*,' 23 total citations were produced. Based on their titles and abstracts, two of these were deemed pertinent.[2,9] Inclusion criteria consisted of systematic reviews of cohort studies containing information on the prognosis of non-surgically treated patients with sciatica. Studies of surgical cohorts were excluded. Similar searches of the Cochrane Library and Cumulative Index to Nursing and Allied Health Literature were conducted, but no additional articles pertinent to this case were found. Therefore, two systematic reviews on the prognosis of conservatively treated patients with sciatica were identified and retrieved.

Critical Evaluation of the Evidence

Atop the hierarchy of research evidence are systematic reviews (Table 1).[10] In the review on prognosis by Pen-

Table 1.
A hierarchy of research evidence (adapted from Haneline[10]).

Study designs (in decreasing order of evidence strength):
1. Systematic reviews and meta-analyses
2. Randomized controlled trials
3. Cohort studies
4. Case-control studies
5. Case series
6. Case reports
7. Editorials and expert opinion
8. Animal research and laboratory studies

gel *et al*.,[9] 15 articles of inception cohorts with acute low back pain or sciatica were evaluated. Upon further review, however, only one of these actually included patients with sciatica. In this sample, leg pain and disability decreased over the first month by an average of 69% and 57%, respectively. Unfortunately, no long-term follow-up data were obtained. In a more recent systematic review, Ashworth *et al*.[2] evaluated seven prospective cohorts of non-surgically treated sciatic patients. The data on individual prognostic factors for recovery were conflicting, however; and the natural course of sciatica (short-term or long-term) was not assessed. Therefore, both systematic reviews were unable to adequately answer the current clinical question. As a consequence, the next highest level of evidence for prognosis was required – that of individual cohort studies.

For this, a quick PubMed search was conducted combining the medical subject heading '*sciatica*' with the text words '*natural course OR inception cohort study*.' This produced only 14 hits, but the first one[11] appeared quite relevant to the current case. A second paper[12] from this same inception cohort was also found in the 'related citations' section of PubMed. Before applying the results from these two studies to the current patient, however, they were first appraised according to the standards proposed by Sackett *et al*.[7].

Are the results of these prognosis studies valid?

Both studies by Suri *et al*.[11,12] used a prospective inception cohort design, and included a clearly defined sample of consecutive patients (n = 154) with MRI-confirmed lumbar disc herniation and sciatica. In order to capture those at a common and early point in the course of their disease, symptom onset upon entry to the study was 12 weeks or less. Patient follow-up was also sufficient, and was carried out over the course of two years. Data were obtained for 73%, 70%, and 77% of the entire cohort for motor weakness, sensory deficit, and pain/disability levels, respectively. (Follow-up rates >70% are within the range of those generally considered acceptable for epidemiologic cohort studies.[13]) The investigators also used validated patient-report outcome measures in their follow-up assessments. To reduce bias, the questionnaires were mailed to each patient for completion. Finally, some prognostic factors for sciatic recovery were identified in both studies, but further validation was required. Nevertheless (based on the above criteria), the results of both cohort studies were deemed valid.

Are the valid results of these prognosis studies important?

The results were deemed important for two reasons. First

Table 2.
Calculating a confidence interval (CI) around a measure of prognosis.[7]

Clinical measure	**Standard error (SE)**	**Calculation of SE and CI**
Proportion (e.g. % of patients recovered from sciatica[11])	SE = $\sqrt{\{p \times (1-p) / n\}}$ • where p is proportion and n is number of patients	If p = 79/97 = 0.81 (or 81%) • SE = $\sqrt{\{0.81 \times 0.19 / 79\}}$ = 0.044 (or 4.4%) • 95% CI is 81% ± 1.96 x 4.4%, i.e. 72.4% to 89.6%

of all, they showed that the prognosis for sciatic pain and motor recovery with non-surgical treatment was good. Secondly using the data provided, 95% confidence intervals (CIs) could be calculated for the results (see Table 2). This is important from an evidence-based standpoint because from these, a clinician can estimate the likelihood of sciatic outcomes over time, including the precision of these prognostic estimates for an individual patient. For example, 81% (95% CI, 72-90%) of patients in this cohort experienced resolution of leg pain, defined as a pain-free period for ≥1 month, following non-surgical treatment.[12] The average time to resolution was six months. Extrapolating these findings to the general population, one can be 95% confident that had the total population of sciatic patients with disc herniations been included in this study, between 72-90% of those undergoing non-surgical treatment would have recovered. By two years, ongoing muscle weakness was also reported by only 25% (95% CI, 15-35%) of those who, treated surgically or non-surgically, had a motor deficit at baseline.[11] The prognosis for sensory recovery was not as good, however, as 47% (95% CI, 32-62%) of patients with baseline deficits reported continuing sensory loss at two-year follow-up.[11] Twenty-five percent (95% CI, 15-35%) of those who underwent non-surgical treatment also reported a recurrence of leg pain within one year after resolution.[12]

Can we apply this valid, important evidence about prognosis in caring for our patient?

When comparing demographic and clinical characteristics, there were many similarities between the current patient and the study sample[12,14] including age, clinical features (i.e. MRI-confirmed disc herniation with sciatica), duration of symptoms, and leg pain severity. Conservative treatment in the study sample[12,14] also consisted of the following: education, physiotherapy, chiropractic, massage, over-the-counter and/or prescription drugs, and/or cortisone injections (if necessary); a minority of patients (n = 21) were referred for surgery. Within the current multidisciplinary context,[15] the patient in this case would have had access to these same therapies as well. Based on all these factors, therefore, the results of the two cohort studies were deemed applicable.

Application of the Evidence

Using the aforementioned results and their calculated 95% CIs, an estimation of the patient's sciatic prognosis was made. As for his leg pain, there was between a 72-90% chance he would recover within six months with non-surgical/conservative care;[12] however, there was also between a 15-35% chance his pain would recur within a year.[12] Concerning his sensory symptoms, there was between a 32-62% probability he would still perceive sensory loss, whether treated surgically or non-surgically, even after two years of follow-up.[11] Thus despite a chance of recurrence, his overall prognosis for pain recovery with conservative care was good, though sensory recovery (even with surgical care) was not as good.

Evaluation of the Outcome

Because this case report was written as part of an 'observation-only' clinical placement,[16] the above prognostic estimates were not actually communicated to the patient. Had they been, the discussion would subsequently have revolved around whether to treat him conservatively or to refer him for surgery. Currently, there is general consensus that in the absence of progressive neurologic deterioration, initial sciatic treatment should be conservative for at least 6-8 weeks.[1] Early surgery may provide a faster recovery of leg pain, but no clear differences in outcome have been shown after one or two years.[1,17] Similarly, Suri *et al*.[11] found no significant differences in baseline characteristics or treatment outcome for patients treated surgically or non-surgically. Regarding efficacy of individual conservative therapies, no one type has been shown to be clearly superior to another (including no treatment) for sciatica.[18] In this case and others, therefore, clinical experience and patient preference – hallmarks of evidence-based medicine[7] – would be important features in the decision-making process.

Limitations

This case report has some limitations. First, only two cohort studies were selected for final appraisal; consequently, other articles relevant to the prognosis of sciatica and/or disc herniations may have been omitted (e.g. Jensen *et al*.[19]). Second, non-response bias in the studies by Suri *et al*.[11,12] could have somewhat compromised the validity of their results. For instance, if all non-responders in their study had undergone non-surgical/conservative care without sciatic resolution (i.e. 'worst-case' scenario), only 79 out of 133 total patients would have actually recovered

within that first year;[12] the prognostic estimate of recovery would therefore have reduced from 81% (95% CI, 72-90%) to 59% (95% CI, 48-70%). In a 'best-case' scenario, however, if all non-responders *had* recovered, the recovery rate would have been as high as 115 out of 133 or 87% (95% CI, 80-94%). Finally, the prognostic estimates in this case report may not be generalizable to other chiropractic patients or practices. For example, the study cohort[11,12,14] and current patient[15] each presented within primary care medical facilities. Treatment in these settings may differ from that of more traditional chiropractic clinics. As such, the applicability of the aforementioned prognostic estimates may be limited toward these patients.

Summary

Prognostic information that is valid, precise, and generalizable can be very useful when counselling patients on the likely course of their disorder and/or when making decisions concerning treatment.[7] As for sciatica, systematic reviews on its prognosis are few, and further validation studies on prognostic factors for non-surgically treated patients are needed. Nevertheless, the prognoses of pain and sensory recovery were estimated for a patient with a lumbar disc herniation and sciatica, in this case using evidence from two individual cohort studies.

References

1. Koes BW, van Tulder MW, Peul WC. Diagnosis and treatment of sciatica. BMJ. 2007; 334:1313-1317.
2. Ashworth J, Konstantinou K, Dunn KM. Prognostic factors in non-surgically treated sciatica: a systematic review. BMC Musculoskelet Disord. 2011; 12:208.
3. Standard of Practice S-013: Consent. Toronto: College of Chiropractors of Ontario. Available from: http://www.cco.on.ca/site_documents/S-013.pdf [Accessed 6 March 2014].
4. Bolton JE. Evidence-based case reports. J Can Chiropr Assoc. 2014; 58(1):6-7.
5. Bolton JE, Breen AC. The Bournemouth Questionnaire: a short-form comprehensive outcome measure. I. Psychometric properties in back pain patients. J Manipulative Physiol Ther. 1999; 22(8):503-510.
6. Evans RC. Illustrated Essentials in Orthopedic Physical Assessment. St. Louis: Mosby; 1994.
7. Sackett DL, Straus SE, Richardson WS, Rosenberg W, Haynes RB. Evidence-based Medicine: How to Practice and Teach EBM. 2nd ed. London: Churchill Livingstone; 2000.
8. Miller PJ, Jones-Harris AR. The evidence-based hierarchy: is it time for change? A suggested alternative. J Manipulative Physiol Ther. 2005; 28(6):453-457.
9. Pengel LHM, Herbert RD, Maher CG, Refshauge KM. Acute low back pain: systematic review of its prognosis. BMJ. 2003; 327:323-325.
10. Haneline MT. Evidence-Based Chiropractic Practice. MA: Jones and Bartlett Publishers Inc; 2007.
11. Suri P, Rainville J, Gellhorn A. Predictors of patient-reported recovery from motor or sensory deficits two years after acute symptomatic lumbar disk herniation. PM R. 2012; 4(12):936-944.
12. Suri P, Rainville J, Hunter DJ, Li L, Katz JN. Recurrence of radicular pain or back pain after nonsurgical treatment of symptomatic lumbar disk herniation. Arch Phys Med Rehabil. 2012; 93(4):690-695.
13. Fewtrell MS, Kennedy K, Singhal A, Martin RM, Ness A, Hadders-Algra M, Koletzko B, Lucas A. How much loss to follow-up is acceptable in long-term randomised trials and prospective studies? Arch Dis Child. 2008; 93(6):458-461.
14. Suri P, Hunter DJ, Jouve C, Hartigan C, Limke J, Pena E, Li L, Luz J, Rainville J. Nonsurgical treatment of lumbar disk hernation: are outcomes different in older adults? J Am Geriatr Soc. 2011; 59(3):423-429.
15. Langs Community Health Centre: About Langs. Available from: http://www.langs.org/about-langs/ [Accessed 5 December 2013].
16. MSc Advanced Professional Practice (Clinical Sciences). Anglo-European College of Chiropractic: Bournemouth. Available from: http://www.aecc.ac.uk/cpd/postgrad/app-clinical.aspx [Accessed 19 March 2014].
17. Jacobs WCH, van Tulder M, Arts M, Rubinstein SM, van Middelkoop M, Ostelo R, Verhagen A, Koes B, Peul WC. Surgery versus conservative management of sciatica due to a lumbar herniated disc: a systematic review. Eur Spine J. 2011; 20(4):513-522.
18. Luijsterburg PAJ, Verhagen AP, Ostelo RWJG, van Os TAG, Peul WC, Koes BW. Effectiveness of conservative treatments for the lumbosacral radicular syndrome: a systematic review. Eur Spine J. 2007; 16(7):881-899.
19. Jensen TS, Albert HB, Soerensen JS, Manniche C, Leboeuf-Yde C. Natural course of disc morphology in patients with sciatica: an MRI study using a standardized qualitative classification system. Spine (Phila Pa 1976). 2006; 31(14):1605-1612.

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Chiropractic management of postpartum pubic symphysis diastasis: A case report

Lucian Henry, BSc, DC*

This case report describes the chiropractic management of a 30-year-old female patient with severe postpartum pelvic pain secondary to pubic symphysis diastasis. No literature was found on the chiropractic management of postpartum symphysis pubis diastasis. The existing literature concerning chiropractic care for symphysis pubis dysfunction during pregnancy is limited and indicates a potential benefit. Separation of the pubic symphysis may include ligamentous injury to the sacroiliac joints and may lead to chronic pain. Pubic symphysis separation of 17 millimeters was present on digital radiograph. Management consisted of chiropractic adjustments, trigger point release, electrical stimulation, moist heat, sacroiliac belt, and specific stabilizing exercises. The patient's pain improved immediately following treatment on the initial visit. Pain was reduced from 8/10 VAS at the first visit to 2/10 at the fourth visit. She was able to resume normal activities and reached a final pain level of 1/10. The diastasis was reduced by 7 millimeters at 14-weeks post radiograph for a final separation of just under 10 millimeters.

Cette étude de cas décrit le traitement chiropratique d'une patiente de 30 ans souffrant de douleurs pelviennes post-partum secondaires à une symphyse pubienne avec diastasis. Aucun ouvrage n'a été trouvé sur le traitement chiropratique d'une symphyse pubienne post-partum avec diastasis. Les ouvrages au sujet des soins chiropratiques d'un dysfonctionnement symphyse pubienne durant la grossesse sont rares et indiquent un bienfait potentiel. La séparation de la symphyse pubienne peut entraîner une lésion ligamenteuse à l'articulation sacro-iliaque et causer des douleurs chroniques. Une radiographie numérique montre une séparation de la symphyse pubienne de 17 mm. Le traitement consistait à des ajustements chiropratiques, à un relâchement de points gâchettes, à de la stimulation électrique, à une chaleur humide, à une ceinture sacro-iliaque et à des exercices adaptés de stabilisation. La douleur de la patiente a diminué immédiatement après le traitement de la première rencontre. La douleur est passée de 8/10 à l'EVA à la première rencontre à 2/10 à la quatrième rencontre. La patiente a réussi à reprendre ses activités habituelles et son niveau de douleur a diminué à 1/10. Le diastasis a diminué de 7 mm 14 semaines après la radiographie pour une séparation définitive inférieure à 10 mm. On recommande une

*Private practice
1314 Pelham Road, Greenville, South Carolina 29615
(864) 288-7797
Email: info@henryclinic.com
There are no disclaimers.
The author has obtained the patient's written consent to publish the case.
There was no external funding or support.

Collaboration between obstetricians, midwives and chiropractors may be warranted.

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KEY WORDS: chiropractic, diastasis, spinal manipulation, postpartum pelvic pain, symphysis pubis

collaboration entre les obstétriciens, les sage-femmes et les chiropraticiens.

(JCCA 2015; 59(1):30-36)

MOTS CLÉS : chiropratique, diastasis, manipulation vertébrale, douleur pelvienne post-partum, symphyse pubienne

Introduction

Symphysis pubis diastasis is a rare cause of pelvic pain in pregnancy but may be underdiagnosed.[1,2] It is a complication of pregnancy and vaginal delivery in which the pubic symphysis separates, resulting in acute pelvic pain, and may lead to severe long-term consequences.[1] This separation may occur during delivery from a rapid birth, forceps delivery, in late pregnancy or post-natal. The incidence has been variously estimated from 1 out of 300 to 1 of 30,000.[1-4] The pubic articulation should not exceed 8 mm in non-pregnant adults or 10 mm in children.[5] Bahlman and colleagues found pubic symphysis width in pregnant women increased from a mean of 4 mm to a mean of 7 mm at term, with an average increase in pubic symphysis width of 3 mm during pregnancy.[6] Garagiola found a mean pubic symphysis width of 6.5 mm in women within 24 hours of uncomplicated vaginal delivery, with widths ranging from 3 to 11 mm.[7]

Diagnosis of diastasis is made based on symptoms and radiographic examination. The patient presents with pain and swelling. Crepitus may be present on walking. Visible deformity may be present on physical examination. Pubic symphysis separation greater than 10 to 13 millimeters on radiograph is diagnostic and represents a subdislocation. A diastasis greater than 14 millimeters indicates attendant damage to the sacroiliac joint.[6] Conservative treatment is recommended, with surgery sometimes necessary for separations greater than 25 millimeters.[4] Surgical treatment may consist of debridement or fusion. Surgical management with plate and screws necessitates cesarean section in the event of future pregnancy. In the absence of appropriate care, diastasis may lead to chronic pain. Surgical management interferes with breast-feeding due to analgesics, antibiotics, and thromboembolic prophylaxis.[8] Scriven et al found a direct relation between permanence of the pubic separation and chronic pain. There does not seem to be a consensus on the natural history of pubic symphysis diastasis nor does the literature suggest that this condition is self-limiting. Pubic symphysis diastasis can lead to incontinence, dyspareunia, chronic pain and / or disability and there is little evidence to guide the clinician.[9] It is uncertain what percentage of patients will end up with poor long-term outcomes. Out of a series of nine patients with diastasis treated by Scriven and colleagues, four were pain free at last follow up, one required surgical fusion, two ended up with severe disability (one in a wheelchair), and the rest had some combination of low back pain or pubic pain and / or dyspareunia.[1] Due to the rarity of the condition and limited literature available, what constitutes appropriate treatment for pubic symphysis diastasis remains controversial.[10]

Since there is a lack of literature on the treatment of postpartum pubic symphysis diastasis, the author reviewed the literature on the conservative treatment of postpartum pelvic pain in general to aid in his clinical decision making. The evidence of treatment effectiveness for postpartum pelvic pain is weak.[11] Various conservative treatments, including physiotherapy, exercises and sacroiliac belt, have been recommended but there does not seem to be a consensus. Nillson-Wikmar and colleagues compared home exercises, in-office exercises and sacroiliac belt in women with postpartum pelvic pain, finding improvement in all three groups with no significant difference between the groups. The authors concluded that home or in-office exercise provided no additional benefit beyond that achieved by a non-elastic sacroiliac belt and giving information.[12] In contrast, Depledge and associates concluded that sacroiliac belts did not add to the results achieved with exercise and advice for symphysis pubis dysfunction.[13] Haugland and colleagues found no significant difference between a control group and those treated with intervention for pregnancy related pelvic pain. The

intervention group was given education on ergonomics, exercises, pain management, and advice for daily life movement, pelvic belt/crutches, and information about delivery. The control group was free to seek other advice or interventions so it is possible that they may have received benefit from other treatment or information. However, the utility of care was rated higher by patients in the intervention group.[14] Stuge and associates compared specific stabilizing exercises with physical therapy without specific stabilizing exercises in a long-term study on postpartum pelvic pain. The group that received specific stabilizing exercises demonstrated lower levels of pain and disability 2 years after delivery.[11]

A literature search was conducted using the Index to Chiropractic Literature and PubMed. A search on PubMed using the keywords, "chiropractic symphysis pubis diastasis," "chiropractic symphysis pubis dysfunction" and "chiropractic postpartum pelvic pain" was performed. No previous literature was found concerning the chiropractic care of a patient with diastasis. Two articles describing three cases of pelvic pain during pregnancy were found.

Panarello reported on the case of a 32-year-old female with severe pubic and groin pain that began when she was 28 weeks pregnant. The pain caused sleep disturbance and difficulty sitting. The patient reported some pain relief following the first chiropractic adjustment and was able to sleep that night. She was pain free by the fifth adjustment. The patient had an uncomplicated vaginal delivery, with no recurrence of pubic pain.[15]

Howell reported on two cases of symphysis pubis dysfunction with successful chiropractic management. Treatment included soft tissue therapy, pregnancy support belt, side-lying mobilizations, pelvic blocks and instrument-assisted pubic symphysis adjustments. Postpartum rehabilitation exercises were done to restore muscular endurance, control and pelvic stability. The two patients were given home care advice to apply ice, stay active, move as a unit, stretch, use a pillow between the knees while sleeping, take regular breaks from sitting and do pelvic floor (Kegel) exercises. Both patients were 30 weeks pregnant. Both patients were mostly pain free on long-term follow up (eleven months for one patient and twelve months for the other).[16]

Intervention and Outcome

The author has obtained the patient's written consent to publish this case report. Patient care, including radiographic procedures were rendered in compliance with applicable law.

A 30-year-old female presented with severe pelvic pain seven days after giving birth to her first child by normal vaginal delivery at home with midwife attending. Her midwife referred her for chiropractic care. She had prior treatment by her primary care physician, who also practiced obstetrics. She was diagnosed with pelvic separation and was treated with ibuprofen.

The patient described constant dull ache at the pelvic area with the pain feeling crushing at times. She indicated pain at the pubic and sacroiliac areas on both sides. She rated the pain 8/10 visual analog scale (VAS). She reported: a) that the pain interfered with walking and lifting either leg, b) crepitus at the sacroiliac area with walking, c) pain at the lower back and radiating to both thighs posteriorly, worse on the right, and d) paresthesia and swelling at both legs with prolonged standing or sitting. The pain did not follow a dermatomal pattern.

The patient's past medical history included left knee surgery and tonsillectomy. She had a past history of T11 fracture. The patient was married and worked as a missionary. Hobbies included biking, tennis and walking. Family history included hypertension. Review of systems was negative other than the chief complaint and associated symptoms already described. Medications included ibuprofen as necessary.

The patient was 162.5 cm height and weighed 59 kg. Vital signs: pulse was 76, blood pressure was 137/89 and respiration rate was 20. She was alert and oriented to person, place, and time, with appropriate mood and affect. The patient appeared in pain. Ambulation was impaired to the point that she required assistance from her husband to walk more than a few steps and her movements were antalgic. Straight leg raise was negative. Lower extremity motor was grade 5/5. Deep tendon reflexes were 2+ at the lower extremities. Dermatome testing at the lower extremities was normal. All lumbosacral ranges of motion were limited. There was tenderness and hypertonicity at the right sacroiliac area. Palpation revealed asymmetry and restriction at L4, L5, sacrum and both sacroiliac joints. Leg length inequality was evaluated with the patient in the prone and supine positions with knees extended, which revealed a right short leg in the prone position and a left short leg in the supine position. Various estimates

Figures 1 and 2, both without SI belt, were taken at the author's office.

Figure 1: *Pre radiograph showing 17 mm separation at pubic symphysis.*

Figure 2: *Post radiograph at 14 weeks, showing reduced separation at pubic symphysis to just under 10 mm.*

exist for the specificity, sensitivity, reliability, and clinical significance of leg length inequality. Shambaugh and colleagues demonstrated inter and intraexaminer reliability of the Derifield-Thompson test for leg length inequality to less than 3mm.[17] In a review of the literature relevant to leg length inequality measures Mannello found that no definitive conclusions could be made because of the variation in estimates of reliability.[18] Knutson investigated the relationship between supine leg length asymmetry and self-reported back pain in a group of 74 volunteers and found sensitivity of 74%, specificity 78%, and positive predictive value of 82%.[19] Schneider et al studied the interexaminer reliability of prone leg length analysis using two chiropractors to examine 45 patients. The authors found 82% interexaminer reliability for determining the short leg in the prone position with knees extended, with 67% agreement for the amount of leg length inequality. The authors concluded that the two clinicians showed good reliability in determining which leg was short but poor reliability in measuring the exact amount of the leg length discrepancy.[20] Triano and colleagues reviewed the literature concerning methods used by chiropractors to determine the site for manipulation, screening 2594 titles, with 201 articles meeting the inclusion criteria. The authors found high quality evidence supporting the use, with limitations, of leg length inequality to assess the pelvis, while the literature has not demonstrated validity for relationship to symptoms.[21]

The author reviewed the radiology report from prior AP pelvic radiograph (single view) taken by the patient's

primary care physician at 5 days post-partum using a source-image distance (SID) of 40 inches. The radiologist's report indicated a 24 mm diastasis at the public symphysis and indicated likely ligamentous injury to the sacroiliac joints. The author consulted the limited literature available and in the author's opinion further imaging was necessary to assess the safety and appropriateness of chiropractic treatment. This was due to the severity of the patient's pain, the failure of symptoms to improve with prior medical treatment, in consideration of the risk of developing incontinence, dyspareunia, chronic pain and disability, and due to the possible need for immediate surgical referral in the event that the diastasis had increased. AP and lateral digital radiographs were taken of the lumbopelvic region at 7 days post-partum with the patient standing at a 40 inch SID and collimated to 14 x 17 inches. Gonadal shielding was not used because it would have obscured part of the area of interest. For the purposes of this paper the patient marker and date (part of the overlay) were removed to maintain patient confidentiality. There were no bony abnormalities and no congenital anomalies were present. The lumbar lordosis was increased with anterior pelvic tilt. There was a mild left convex scoliosis at the lumbar spine. A 17 mm pubic symphysis separation was noted (Figure 1).

The patient was diagnosed with postpartum diastasis of the pubic symphysis, accompanied by injury to the sacroiliac joints and segmental dysfunction of the lumbar, sacral and pelvic regions. Transcutaneous electrical nerve stimulation (TENS) and moist heat therapy were applied to the lower lumbar and sacral regions to modulate pain.

The analgesic effect of TENS for acute pain has been consistently demonstrated by randomized controlled trials but remains controversial for specific conditions such as low back pain due to poor study designs and small sample sizes.[22] A review by French et al found limited evidence for the common practice of applying superficial heat or cold for low back pain, while moderate evidence in a small number of trials supported the use of superficial heat for the short-term reduction of pain and disability in patients with acute or subacute low back pain. The addition of exercise further reduced pain and improved function.[23] Low force chiropractic adjustments of L4, L5, sacrum, left and right innominate bones were done using an Activator (chiropractic percussive instrument). Contact points were the lumbar mamillary processes, sacral apex and ischial tuberosity. The patient was fitted for a sacroiliac (SI) belt. She was instructed on specific stabilizing therapeutic exercises, which were done in-office and instructions were given for home exercises. Exercises included Kegel's, pelvic tilt and bridge, progressing to core strengthening using a stability ball. Myofascial trigger points were found at the hip flexors by palpatory examination with the patient in a supine position with the hips slightly flexed. The location of the trigger points on either side was ascertained as the iliopsoas due to location inferior to the inguinal ligament and was confirmed by asking the patient to actively flex the hips. Trigger points were identified by palpation of a localized tender point within a taut band of muscle and the observance of a local twitch response.[24] Manual compression was applied until a release was felt and reduced tenderness was perceived by the patient. No treatment was applied directly to the pubic symphysis. The patient's pain improved immediately following treatment on the initial visit and was reduced to 2/10 VAS at the fourth visit. She was able to drive herself for the first time in approximately one month after three weeks of care.

After five weeks of chiropractic care, the patient had a consultation with an orthopedic surgeon. She indicated that her condition was improving but continued to limit her ability to stand, walk and bend and prevented sexual intercourse. She reported that chiropractic care, moist heat, and ibuprofen provided pain relief. The orthopedist ordered an AP pelvic radiographs with and without SI belt, at a 40 inch SID. The diastasis was reduced to 12 mm without the SI belt and 8 mm with the SI belt. The orthopedist recommended continuing use of SI belt, noting that the SI belt was effective in reducing the diastasis.

After six weeks of chiropractic care, the patient reported progressive functional improvement, with reduced difficulty getting up from sitting, less difficulty walking and climbing stairs, and less difficulty lying on her side.

The patient returned for a follow up appointment at approximately nine weeks and was treated using Activator chiropractic adjustment of L5, sacrum, and the sacroiliac joints and home exercise recommendations. She continued with home exercises and the SI belt. At a fourteen-week follow up, she rated her pain 1/10 VAS and reported generally feeling better with return to normal activity. She reported soreness and tightness at the groin on squatting. A post AP lumbopelvic radiograph was taken per patient

request and because the patient expressed apprehension to returning to physical activity without additional imaging. This radiograph indicated pubic symphysis separation of just under 10 mm (Figure 2) (without the SI belt). The patient was encouraged to gradually return to normal activity and was released to return as needed.

Discussion

Various physiotherapy methods have been recommended for postpartum pelvic pain but the evidence of benefit is weak. This patient's care included specific stabilizing exercises, home care advice and sacroiliac belt, in accordance with approaches described in the existing literature. Additionally, she was treated using specific chiropractic adjustments to reduce joint dysfunction at the lumbar, sacral and pelvic areas, using an ischial tuberosity contact to correct anterior superior pelvic misalignment. In this case chiropractic management appears to have helped reduce pain, reduce pubic symphysis separation, and facilitate a return to normal activities.

The author suggests future research to investigate chiropractic care for patients with pubic symphysis diastasis.

Conclusion

In this case, the patient reported that chiropractic care was effective in reducing pain associated with pubic symphysis diastasis. While the author acknowledges that there is no way to know what intervention, if any, helped in this patient's improvement or whether reduction in pain and decreased diastasis resulted from natural history, the potential for long-term pain and disability and the scarcity of existing literature indicate the need for further investigation. Collaboration between chiropractors, midwives, and obstetricians should be encouraged.

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References

1. Scriven MW, Jones DA, McKnight L. The importance of pubic pain following childbirth: a clinical ultrasonographic study of diastasis of the pubic symphysis. JR Soc Med. 1995; 88:28-30.
2. Musumeci R, Villa E. Symphysis pubis separation during vaginal delivery with epidural anaesthesia. Reg Anaesth. 1994; 19:289-91.
3. Senechal PK. Symphysial pubis separation during childbirth. J Am Board Fam Pract. 1994; 7(2):141-4.
4. Parker J, Bhattacharjee M. Peripartum diastasis of the symphysis pubis N Engl J Med. 2009; 361:1886 November 5, 2009 DOI: 10.1056/NEJMicm0807117.
5. Muecke EC, Currarino G. Congenital widening of the pubic symphysis: associated clinical disorders and roentgen anatomy of affected bony pelves. Am J Roentgenol Radium Ther Nucl Med. 1968 May;103(1):179-85.
6. Bahlmann F, Merz E, Macchiella D, Weber G. Ultrasound imaging of the symphysis fissure for evaluating damage to the symphysis in pregnancy and postpartum. Z Geburtshilfe Perinatol. 1993 Jan-Feb;197(1):27-30.
7. Garagiola DM, Tarver RD, Gibson L, Rogers RE, Wass JL. Anatomic changes in the pelvis after uncomplicated vaginal delivery: a CT study on 14 women. AJR Am J Roentgenol. 1989 Dec;153(6):1239-41.
8. Pedrazzini A, Bisaschi R, Borzoni R, Simonini D, Guardoli A. Post partum diastasis of the pubic symphysis: a case report. Acta Biomed. 2005 Apr;76(1):49-52.
9. Shippey S, Roth J, Gaines R. Pubic symphysis diastasis with urinary incontinence: collaborative surgical management. Int Urogynecol J. 2013 Oct;24(10):1757-9. doi: 10.1007/s00192-013-2120-0. Epub 2013 May 15.
10. Hou Z, Riehl JT, Smith WR, Strohecker KA, Maloney PJ. Severe postpartum disruption of the pelvic ring: report of two cases and review of the literature. Patient Saf Surg. 2011;5:2. Published online 2011 January 13. doi: 10.1186/1754-9493-5-2
11. Stuge B, Laerum E, Kirkesola G, Vøllestad N. The efficacy of a treatment program focusing on specific stabilizing exercises for pelvic girdle pain after pregnancy: a randomized controlled trial. Spine (Phila Pa 1976). 2004 Feb 15;29(4):351-9.
12. Nilsson-Wikmar L, Holm K, Oijerstedt R, Harms-Ringdahl K. Effect of three different physical therapy treatments on pain and activity in pregnant women with pelvic girdle pain: a randomized clinical trial with 3, 6, and 12 months follow-up postpartum. Spine (Phila Pa 1976). 2005 Apr 15;30(8):850-6.
13. Depledge J, McNair PJ, Keal-Smith C, Williams M. Management of symphysis pubis dysfunction during pregnancy using exercise and pelvic support belts. Phys Ther. 2005 Dec;85(12):1290-300.
14. Haugland KS, Rasmussen S, Daltveit AK. Group intervention for women with pelvic girdle pain in pregnancy. A randomized controlled trial. Acta Obstet Gynecol Scand. 2006;85(11):1320-6.
15. Panarello SR. Symphysis pubis subluxation: pre and post partum chiropractic care. J Clinical Chiropr Ped. 2005:6(3):432-435.

16. Howell ER. Pregnancy-related symphysis pubis dysfunction management and postpartum rehabilitation: Two case reports; J Can Chiropr Assoc. Jun 2012;56(2): Online access only p1-2-111.
17. Shambaugh P1, Sclafani L, Fanselow D. Reliability of the Derifield-Thompson test for leg length inequality, and use of the test to demonstrate cervical adjusting efficacy. J Manipulative Physiol Ther. 1988 Oct;11(5):396-9.
18. Mannello DM. Leg length inequality. J Manipulative Physiol Ther. 1992 Nov-Dec;15(9):576-90.
19. Knutson GA. Incidence of foot rotation, pelvic crest unleveling, and supine leg length alignment asymmetry and their relationship to self-reported back pain. J Manipulative Physiol Ther. 2002 Feb;25(2):110E.
20. Schneider M1, Homonai R, Moreland B, Delitto A. Interexaminer reliability of the prone leg length analysis procedure. J Manipulative Physiol Ther. 2007 Sep;30(7):514-21.
21. Triano JJ, Budgell B, Bagnulo A, Roffey B, Bergmann T, Cooperstein R, Gleberzon B, Good C, Perron J, Tepe R. Review of methods used by chiropractors to determine the site for applying manipulation. Chiropr Man Therap. 2013 Oct 21;21(1):36. doi: 10.1186/2045-709X-21-36.
22. DeSantana JM1, Walsh DM, Vance C, Rakel BA, Sluka KA. Effectiveness of transcutaneous electrical nerve stimulation for treatment of hyperalgesia and pain. Curr Rheumatol Rep. 2008 Dec;10(6):492-9.
23. French SD, Cameron M, Walker BF, Reggars JW, Esterman AJ. A Cochrane review of superficial heat or cold for low back pain. Spine (Phila Pa 1976). 2006 Apr 20; 31(9):998-1006.
24. Gerwin RD. Diagnosis of myofascial pain syndrome. Phys Med Rehabil Clin N Am. 2014 May;25(2):341-55. doi: 10.1016/j.pmr.2014.01.011. Epub 2014 Mar 18.

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Treatment of a patient with posterior cortical atrophy (PCA) with chiropractic manipulation and Dynamic Neuromuscular Stabilization (DNS): A case report

Vinicius T. Francio, DC, MS[a]
Ron Boesch, DC[b]
Michael Tunning, DC, ATC[c]

Objective: *Posterior cortical atrophy (PCA) is a rare progressive neurodegenerative syndrome which unusual symptoms include deficits of balance, bodily orientation, chronic pain syndrome and dysfunctional motor patterns. Current research provides minimal guidance on support, education and recommended evidence-based patient care. This case reports the utilization of chiropractic spinal manipulation, dynamic*

Objectif : *L'atrophie corticale postérieure (ACP) est un syndrome neurodégénératif évolutif ayant des symptômes inhabituels, notamment un manque d'équilibre, un trouble d'orientation du corps, des douleurs chroniques et une organisation motrice dysfonctionnelle. Les recherches actuelles offrent peu de renseignements à propos du soutien, de l'éducation et des soins basés sur des données probantes recommandés aux patients. Cette étude de cas analyse l'utilisation de manipulations chiropratiques vertébrales, de stabilisations*

[a] Chiropractic Physician, Researcher
OSSO Oklahoma Spine Surgery and Orthopedics – Community Hospital Campus
3110 SW 89th, Suite 200E Oklahoma City, OK 73159
Palmer Center for Chiropractic Research (PCCR)
741 Brady Street, Davenport, IA 52803
[b] Professor, Faculty Clinician, Dean of Clinics, Palmer Academic Health Center
Palmer College of Chiropractic
741 Brady Street, Davenport, IA 52803
[c] Assistant Professor, Department of Diagnosis & Radiology
Palmer College of Chiropractic
1000 Brady Street, Davenport, IA 52803

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neuromuscular stabilization (DNS), and other adjunctive procedures along with medical treatment of PCA.

Clinical features: *A 54-year-old male presented to a chiropractic clinic with non-specific back pain associated with visual disturbances, slight memory loss, and inappropriate cognitive motor control. After physical examination, brain MRI and PET scan, the diagnosis of PCA was recognized.*

Intervention and Outcome: *Chiropractic spinal manipulation and dynamic neuromuscular stabilization were utilized as adjunctive care to conservative pharmacological treatment of PCA. Outcome measurements showed a 60% improvement in the patient's perception of health with restored functional neuromuscular pattern, improvements in locomotion, posture, pain control, mood, tolerance to activities of daily living (ADLs) and overall satisfactory progress in quality of life. Yet, no changes on memory loss progression, visual space orientation, and speech were observed.*

Conclusion: *PCA is a progressive and debilitating condition. Because of poor awareness of PCA by physicians, patients usually receive incomplete care. Additional efforts must be centered on the musculoskeletal features of PCA, aiming enhancement in quality of life and functional improvements (FI). Adjunctive rehabilitative treatment is considered essential for individuals with cognitive and motor disturbances, and manual medicine procedures may be consider a viable option.*

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KEY WORDS: chiropractic, spinal manipulation, neurodegenerative disease, physical and rehabilitation medicine

neuromusculaires dynamiques et d'autres procédures d'appoint connexes au traitement médical de l'ACP.

Caractéristiques cliniques : *Un homme de 54 ans s'est présenté à une clinique de chiropratique avec des douleurs lombaires non précisées, des troubles de la vue, de légères pertes de mémoire et un contrôle cognitif moteur inadéquat. Après un examen clinique, une IRM du cerveau et une tomographie par émission de positons, l'ACP a été diagnostiquée.*

Intervention et résultats : *On a utilisé des manipulations chiropratiques vertébrales et des exercices de stabilisation neuromusculaire dynamique à titre de soins d'appoint au traitement pharmacologique conservateur de l'ACP. Selon l'évaluation des résultats, le patient voit une amélioration de sa santé de l'ordre de 60 %, soit le retour des fonctions neuromusculaires, l'amélioration de la locomotion, la posture et l'humeur, une diminution de la douleur, la capacité à réaliser les activités de la vie quotidienne, et une satisfaction d'ensemble de la progression de la qualité de vie. Toutefois, on n'a pas observé d'amélioration sur le plan de la progression de la perte de la mémoire, l'orientation visuelle dans l'espace et la parole.*

Conclusion : *L'ACP est un trouble progressif et débilitant. Souvent, les patients souffrant d'ACP ne reçoivent pas tous les soins dont ils ont besoin, car les médecins connaissent mal ou ne connaissent pas l'ACP. On doit investir plus d'efforts dans les caractéristiques musculosquelettiques de l'ACP dans le but d'améliorer la qualité de vie et la fonctionnalité. Le traitement d'appoint de réadaptation est considéré comme essentiel aux patients atteints de troubles cognitif et moteur, et il convient de considérer les procédures de médecine manuelle comme étant une option viable.*

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MOTS CLÉS : chiropratique, manipulation vertébrale, maladie neurodégénérative, médecine physique et de réadaptation

Introduction

Posterior cortical atrophy (PCA) is a rare progressive neurodegenerative syndrome characterized by deficits in higher-order visual processing, in which memory, judgment and insight is preserved.[1] This disorder may be considered a variant of Alzheimer disease, but can also result from corticobasal degeneration, Creutzfeldt-Jakob, dementia with Lewy bodies and/or subcortical gliosis. Typically, the age of onset for PCA is 50-65 years old. However, prevalence and incidence are unknown. Furthermore, etiology often remains uncertain until postmortem examination.[2] Clinical features of PCA constitute a wide variety of signs and symptoms; nevertheless, the most frequent include hemiagnosia (deficit in awareness of one side of space), optic ataxia (lack of coordination between visual inputs and hand movements, resulting in inability to reach and grab objects), visual agnosia (an impairment in recognition of visually presented objects), alexia (difficulty to understand written words), acalculia (difficulty with simple mathematical tasks), and agraphia (loss in the ability to communicate through writing).[3] Functions related to the parietal, occipital, and occipitotemporal regions of the brain may be affected, and may include left-right disorientation, language skills, and space perception deficits.[1-3] Some unusual symptoms are potentially linked to different brain arrangements, such as visuovestibular and pontinomedullary reticular formation interactions, evoking a wide range of phenomena, including disturbance of balance, bodily orientation, chronic pain syndrome, decomposition of motion and dysfunctional motor patterns.[3-4] Early identification and increased awareness is key for appropriate management of PCA, since the condition is debilitating and rapidly progressive in its early years. Nevertheless, current research provides minimal guidance on support, education and recommended evidence-based patient care.[5] Benson and colleagues[1] recognized the clinical presentation of PCA for more than 2 decades and yet, compared to other conditions, it is essentially overlooked by researchers and clinicians.

Therefore, the aim of this study is to describe the case of a 54-year-old male with PCA who sought chiropractic care, as well as how the condition was co-managed along with conservative pharmacological care.

Case report

A 54-year-old male sought care for musculoskeletal pain associated with stiffness, and tightness in his neck and back. The symptoms began 6 months earlier. About that time, he noticed he was bumping into things, forgetting familiar faces, and was having difficulty reaching out to pick up and grasp objects. Activities such as tying his shoes and balancing his cheque book were becoming progressively more difficult. His symptoms were primarily affecting activities of daily living (ADLs), such as the ability to drive, perform household chores, dress, and his leisure activities. Family members also reported fluctuations with mood and behavior. Previous medical history was unremarkable, except for a triple bypass surgery in the past.

Upon physical examination performed by the chiropractic physician, satisfactory mental status was accomplished via the mini-mental status test. However, decreased nasal field, and peripheral vision, mainly in the left eye, was noted. Along with reported blurred vision during eye examination. Glabellar reflex (tap sign) produced persistent response only in his right eye, and finger snap activation in the auditory canal activated bilateral blink response on his right ear, and only right blink response (unilateral) on the left ear. Eye movements were conjugated, but he was unable to follow the examiner with increased speed and specific smooth pursuits, eyes opposite and prosaccade eye exercises were conducted. During the neurological exam, strong concentration and reinforcement was required due to difficulty regarding right-left discrepancy. Presence of hyper-reflexive knee jerks (+3) bilaterally, grossly intact plantar reflex, and sensory examination was found. Manual muscle testing indicated very mild left upper extremity flexor angulation and lower extremity extensor angulation. Bilateral increase pronator drift and right-sided wrist cogwheel rigidity was noted. Hoffman's sign was positive bilaterally. Other pathological reflexes were negative. Inability to perform finger-to-nose on the left, and difficulty with graphognosia, barognosia and sterognosia testing were noted. Spinal exam revealed overall loss of flexibility and range of motion in all planes, with anterior head carriage, and hyperkyphotic antalgic posture. Diffuse superficial tenderness to palpation in the back with spinal core stabilizers weakness and joint hypomobility was found. Functional motor pattern analysis revealed mild akathisia and deconditioning syndrome.

Spinal radiographs showed multilevel cervicothoracic

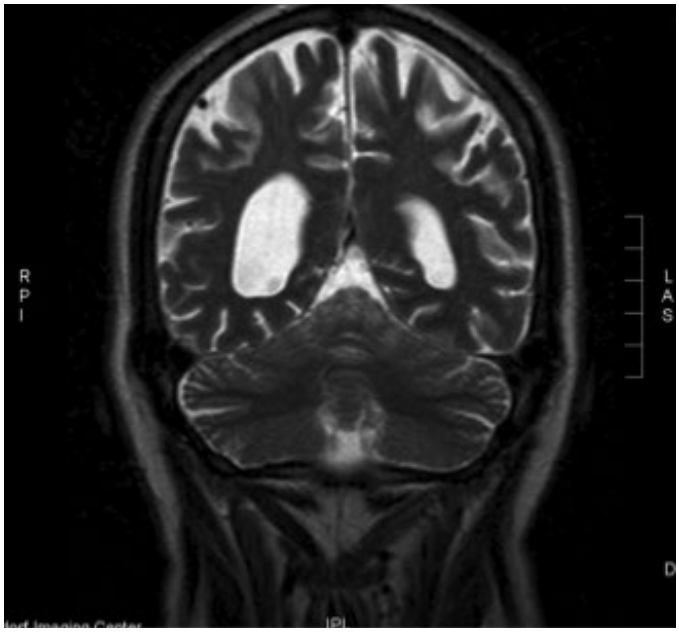

Figure 1.
Coronal plane; Brain magnetic resonance imaging (MRI) revealing enlarged ventricles

Figure 2.
Transverse plane; Brain magnetic resonance imaging (MRI) revealing enlarged ventricles

spine degenerative disc/joint disease, reduction of lordotic curve and moderate anterior head carriage. The neurologist ordered first a magnetic resonance image (MRI) of the brain (see Figure 1 and 2), which revealed mild ventricle enlargement due to ex vacuo dilatation secondary to the diffuse, primarily parieto-occipital cortical atrophy. In addition, a positron emission tomography (PET) study was performed, which indicated lack of radiotracer uptake in the parieto-occipital cortices bilaterally, more pronounced and with a larger volume of involvement on the right (see Figure 3). This study was performed to determine the areas of the brain that were not functioning properly. The clinical diagnosis of posterior cortical atrophy (PCA) was accomplished after a thorough investigation, with corroborative findings from the neurologist consult. Communication between the neurologist and the chiropractic physician regarding exchange of records, treatment and outcomes occurred several times. The neurologist recommended daily dosage of Donepezil, a common medication prescribed for Dementia-like symptoms, which is standard of care for PCA, and regular follow-up regarding the condition's prognosis. The patient decided to adhere to the suggestion, and also pursue chiropractic

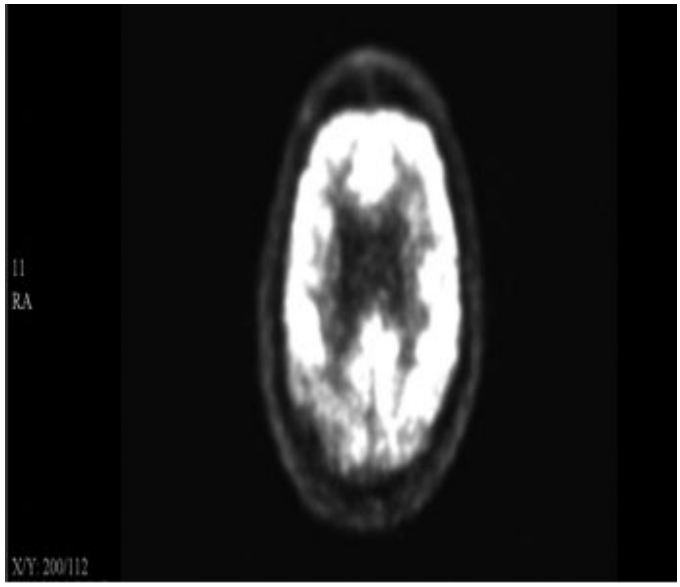

Figure 3.
Brain positron emission tomography (PET) scan with FGD radiotracer. Brain positron emission tomography (PET) scan revealing lack of radiotracer uptake in the parieto-occipital cortices bilaterally, more pronounced and with a larger volume of involvement on the right.

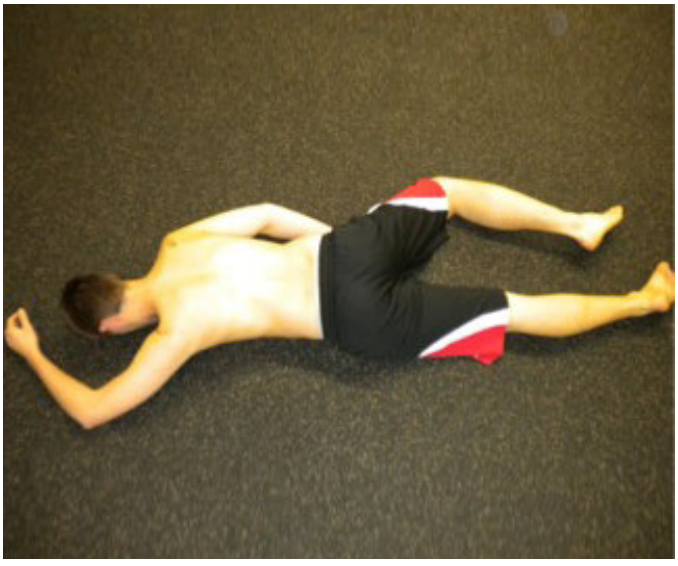

Figure 4.
Dynamic Neuromuscular Stabilization (DNS) procedure utilizing crawl position 1 with treatment points. This is not the actual patient, rather only a model illustration about the procedure sequence.

Figure 5.
Dynamic Neuromuscular Stabilization (DNS) procedure utilizing crawl position 2 with treatment points. This is not the actual patient, rather only a model illustration about the procedure sequence.

manipulative therapy and musculoskeletal rehabilitation hoping to enhance his neuromuscular function.

Our treatment consisted of manual medicine and rehabilitation procedures focused on the neuromuscular features of PCA, in a 42-week period initially, and then another 13-week period. Frequency and duration of treatments were variable throughout these periods. At times, visits were twice a week, others once every two weeks due to conflicting schedule. Treatment visits consisted of the use of vibratory stimuli therapy over the distal extremities (to enhance vibratory and proprioceptive input to the CNS), with the use of a 128htz tuning fork pre and post high-velocity low-amplitude (HVLA) spinal manipulation. Proprioceptive retraining exercises standing on balance training ball performing finger-nose exercise and catching various size objects was also incorporated. DNS therapy was performed in the supine and prone position, with 2 or 3 doctors asking the patient to perform specific motor pattern motions, average approximately 12 minutes (see Figure 4, 5 and 6). At-home therapeutic exercises for thoracic extension, and hamstrings/adductor stretch-hold exercises were encouraged.

Progress evaluation focused on functional improve-

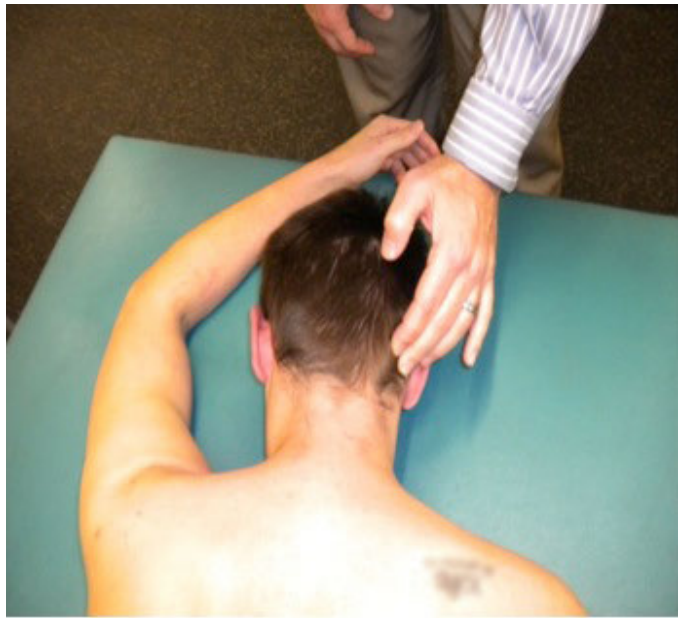

Figure 6.
Dynamic Neuromuscular Stabilization (DNS) procedure utilizing crawl position 3 with treatment points. This is not the actual patient, rather only a model illustration about the procedure sequence.

ments (FI) involving activities such as the ability to drive, dress, perform household chores, climb stairs, play golf, motor behavior, and overall global health status. The response to the DNS therapy, and proprioceptive exercises was used every visit to monitor progress. In a 10-month period, the Health Status Questionnaire (HSQ) demonstrated a 38% functional improvement. Within a 13-month period, HSQ and Back Bournemouth Questionnaire (BBQ) demonstrated a 60% improvement, which is extremely significant regarding musculoskeletal pain and FI. These two outcome measurements are a short, self-reported questionnaire that measures objective improvements with different dimensions in patients with musculoskeletal pain, aiming pain and disability, but also takes the affective and cognitive aspects of pain into account. They are well known for their validity, reliability and responsiveness. Likewise, functional impairments previous noted, such as inability to play golf, self-care (dressing), climbing stairs and motor control were enhanced. Better control of involuntary motion, right and left discrimination, and refined motor skills for reaching objects were observed. No progression in memory loss, space orientation, and speech disturbances was observed.

Significant functional improvement of neuromusculoskeletal control and pain was accomplished. Altered movement patterns between agonists and antagonists secondary to dysfunctional sensory inputs were restored with improvement in the synergistic function between different muscle groups and fasciae. In this case, the integration of chiropractic care with DNS resulted in a number of remarkable musculoskeletal restorations, with improved mechanics and tolerance to activities of daily living. The patient was encouraged to continue with conservative care, in addition to daily routine of exercise, and active rehabilitative program.

Discussion

PCA is a progressive, debilitating, condition characterized by difficulties in visuospatial tasks, writing, and motor control.[6] The loss of cognitive motor function related to PCA, and the presence of prominently asymmetric limb apraxia, illustrates an uncommon feature of this syndrome.[7] Musculoskeletal examination in most cases is unremarkable; however, severe progressive visual and cognitive findings are common. According to Kas and colleagues[8], the visual symptoms are perhaps more likely to be detected than other deficits due to its greater impairment level, nevertheless, neuromuscular dysfunctions are equally as important to address. Because of poor awareness of PCA by physicians, patients usually receive incomplete care. Adjunctive rehabilitative treatment is considered fundamental for individuals with cognitive and motor disturbances. Special efforts must be centered on the neuromuscular features of PCA, and appropriate management with manual medicine, rehabilitation, exercises and cognitive therapy is recommended as an option for care, aiming to enhance quality of life, and improve neuromusculoskeletal functional.[9] According to Kolar[10], influenced by the work of Vojta, Dynamic Neuromuscular Stabilization may be considered an adjunctive therapy in such cases to help enhance specific functions of the musculoskeletal system. Such an approach was chosen by the clinician as the most suitable therapy to facilitate passive movement patterns in this case, while enhancing primitive subcortical kinesiological patterns to stimulate appropriate neuromuscular function.

Dynamic neuromuscular stabilization studies suggest that our motor behavior is predetermined. During motor development, characteristic muscle synergies are stored in the brain as a matrix, responsible for posture, locomotion, and movement patterns.[10] The highest level of integration occurs at the cerebral cortex, which allows us the ability to develop new skills, refines, and predicts specific movements. The mechanism, which the nervous system responds to external stimuli and adjusts future responses based on previous outcomes, is defined as 'functional neural plasticity,' and its understanding is fundamental in the field of neuromusculoskeletal rehabilitation.[10] The function of each part of the nervous system is dependent on the central integrative state of each hemisphere, which is determined to a large extent by the awareness of afferent stimulation from the periphery. This fact supports the concept of functional hemisphericity, which involves asymmetric cortical modulation of various forms of input that can result in decreased postural tones, spinal stiffness, pelvic floor weakness, and inhibition of the intrinsic stabilizer spinal muscles.[10-11] The pontomedullary reticular formation receives nearly 90% of the output dedicated to the neuraxis and cortex modulation, ipsilaterally. The impact that an asymmetrical cortical output has clinically in the neuromusculoskeletal system is evident with symptoms such as, decrease in muscle tone ipsilaterally,

ipsilateral pain syndrome, generalized spinal stiffness and deconditioning syndrome.[11,12,13]

As evidenced by the present case, this clinical presentation of PCA was associated with musculoskeletal dysfunctions and asymmetric cortical modulation. The patient's presentation with ipsilateral flexor angulation of upper limb, extensor angulation of lower limb, spinal stiffness, deconditioning, and imbalance motor patterns supports the application of manual medicine and rehabilitation as adjunctive therapeutic procedures. SMT and DNS were utilized focusing on movement pattern analysis. According to Vacek[14] if the central nervous system (CNS) is not functioning normally, the postural program will change and kinesiological analysis of postural activity and re-actions can be used to evaluate the CNS. Also, mobilization and manipulation of spinal joints and fascia may influence global movement patterns allowing higher levels of cortical function, stimulating appropriate cortex output through balanced peripheral afferent stimuli to the PMRF.[11] Other therapeutic procedures included inhibitory stretching protocols to the facilitated muscles as described in the upper and lower cross syndrome, as well as facilitating exercises to increased activity in the inhibited muscles.[13] Balance, vibration therapy over the extremities and proprioception exercises using solid ground and 2-way wobble boards were used with eyes open and closed, to stimulate appropriate cortical awareness of proprioceptive and cerebral pathways.[14,15]

According to several studies[11,13,16,17], a number of pathways may explain the potential effects of manual medicine procedures on the neuraxis, and therefore theoretically corroborate the positive neuromuscular improvements in this case. These include, but are not limited to, exciting spinoreticular pathways and dorsal column pathways to the PMRF, modulation of vestibulosympathetic pathways and vestibulocerebellar activation of the nucleus tractus solitarius, dorsal motor nucleus of vagus, and nucleus ambiguous. Yet, spinal manipulation and afferent peripheral stimulation may result in brain hemisphere influence via descending excitation of PMRF pathways, inhibitory control of IML cell column, which may alter central integration of the brain stem and hypothalamus via spinoreticular and spinothalamic afferent direct connections.[11 14-19] These manual medicine procedures tend to facilitate global stability and enhance musculoskeletal functional patterns addressing a number of anatomical structures that help maintain appropriate neuromuscular stimulation. Mechanoreceptors, proprioceptors, Golgi tendon organs, muscle spindles, and her sensory organ are all directly influenced by mobilization and directive manual therapy approach.[20-23] Ultimately, extensive sensory input into the CNS contributes to the neuromuscular adaptation demanded by the cortex needs in its process of reorganization. Hence, manual medicine procedures play a fundamental role in adding sensory stimulation to peripheral receptors, known to influence motor response patterns, which directly affect posture, balance, locomotion, and musculoskeletal function.[24-26]

The manual medicine procedures used in this case improved the neuromuscular function and quality of life of this patient. Yet, no changes in memory, visual spatial or writing ability were seen. Furthermore, the results of this case report, and likewise cases [27, 28], where similar procedures were utilized, supports the value of such procedures in the co-management of neurological conditions associated with musculoskeletal dysfunctions. Chiropractic, DNS, and adjunctive therapies can help manage neuromuscular compensations and altered movement patterns, secondary to improper spinal mechanics, and dysfunctional sensory inputs between muscle groups focusing in enhancing structural mobility, postural integrity and enhance neuromuscular function.[29,30] Our clinical approach for the management of musculoskeletal dysfunction, such as in this case, emphasizes the utilization of manual stimulation of specific zones of the body by properly mobilizing/manipulating joints to evoke predetermined efferent motor patterns by the central nervous system, facilitating sensory input to enhance cortex assimilation, and appropriate re-organization or adaptation.

Limitations

This report has the limitations of all case reports that represent the experience of a single patient. Therefore, findings may not be generalizable to other patients.

Conclusion

As evidenced by current literature and substantiated by the present case, PCA is a progressive neurological disorder associated with higher cortical dysfunctions, and impaired neuromusculoskeletal ability. Medical research provides minimal guidance regarding the appropriate management of PCA. Yet, multidisciplinary care is con-

sidered essential for successful results. Chiropractic, DNS and other procedures were utilized in this case. There was no progression in memory loss, speech or visuospatial orientation noted, though significant musculoskeletal improvements were achieved, as endorsed by an improvement in 60%, according to the HSQ, and BBQ outcome assessment tools, especially in quality of life, and musculoskeletal function. Our goal with this study is to increase multidisciplinary management awareness, and include chiropractic care in future research considering its contributory use for functional enhancement in the co-management of neurological disorders with musculoskeletal dysfunctions.

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References

1. Benson DF, Davis RJ, Snyder BD. Posterior cortical atrophy. Arch Neurol. 1988; 45:789-793.
2. Renner JA, Burns JM, Hou CE et al. Progressive posterior cortical atrophy dysfunction: a clinicopathologic series. Neurology. 2004; 63:1175-1180.
3. Crutch SJ, Lehmann M, Schott JM et al. Posterior cortical atrophy. Lancet Neurol. 2012; 11:170-78.
4. Migliaccio R, Agosta F, Rascovsky K et al. Clinical syndromes associated with posterior atrophy: early age at onset AD spectrum. Neurology. 2009; 73:1571-1578.
5. Mendez MF, Ghajarania M, Perryman KM. Posterior cortical atrophy: clinical characteristics and difference compared to Alzheimer's disease. Dement Geriatr Cogn Disord. 2002: 14:33-40.
6. McMonagle P, Deering F, Berliner Y et al. The cognitive profile of posterior cortical atrophy. Neurology. 2006; 66:331-338.
7. Tang-Wai DF, Josephs KA, Boeve BF et al. Visuospatial dysfunction as the presenting feature in corticobasal degeneration. Neurology. 2003; 61:1134-1135.
8. Kas A, de Souza LC, Samri D et al. Neural correlates of cognitive impairment in posterior cortical atrophy. Brain. 2011; 134:1464-78.
9. Roca M, Gleichgerrcht E, Torralva T et al. Cognitive rehabilitation in posterior cortical atrophy Neuropsychol Rehabil. 2010; 20:528-40.
10. Kolar P, Kobesova A. Three levels of motor control in the assessment and treatment of the motor system. J Bodyw Mov Ther. 2014; 18:23-33.
11. Beck RW. Fundamental concepts in functional neurology. In: Functional Neurology for practitioners of manual therapy. Churchill Livingtstone: Philadelphia, 2009: 1-20.
12. Savic I, Pauli S, Thorell JO et al. In vivo demonstration of altered benzodiazepine receptor density in patients with generalized-epilepsy. J Neurology, Neurosurgery and Psychiatry. 1994; 57:784-797.
13. Nyberg-Hansen R. Sites and mode of termination of reticulospinal fibers in the cat. An experimental study with silver impregnation methods. J Comparative Neurology. 1965; 124:74-100.
14. Vacek J. Sensory-motor approach to the stabilization system of the spine in patient with chronic back pain. International Musculoskeletal Medicine. 2012; 34:48-50.
15. Page P, Frank C, Lardner R. Assessment and Treatment of Muscle Imbalance: The Janda Approach. 2009. Human Kinetics.
16. Rojas Vegas S, Abel T, Lindschulten R et al. Impact of exercise on neuroplasticity-related proteins in spinal cord injured humans. Neuroscience. 2008; 20:40-50.
17. Garcia-Larrea L, Maarrawi J, Peyron R et al. On the relation between sensory deafferentation, pain and thalamic activity in Wallenberg's syndrome: a PET-scan study before and after motor cortex stimulation. Eur J Pain. 2006; 10:677-88.
18. Holt K, Beck RW, Sexton S. Reflex effects of a spinal adjustment on blood pressure. Proceeding of the Association of Chiropractic Colleges: Research agenda conference 2006. Washington, DC.
19. Carrick FR. Changes in brain function after manipulation of the cervical spine. J Manipulative Physiol Ther. 1994; 20:529-545.
20. Page SJ, Gater DR, Bach-Rita P. Reconsidering the motor recovery plateau in stroke rehabilitation. Arch Phys Med Rehab. 2004; 85:1377-1381.
21. Panjabi MM. A hypothesis of chronic back pain: ligament sub failure injuries lead to muscle control dysfunction. Eur Spine J. 2006; 15:668-676.
22. Karni A, Mayer G, Jezzard P et al. Functional MRI evidence for adult motor cortex plasticity during motor skill learning. Nature. 1995; 377:155-158.
23. Krakauer J. Motor learning: its relevance to stroke recovery and neurorehabilitation. Current Opinion in Neurology. 2006; 19:84-90.
24. Rome L. Neurovertebral influence upon the autonomic nervous system: some of the somato-autonomic evidence to date. Chiropr J Austr. 2009; 39:9-33.
25. Rome L. Neurovertebral influence upon the autonomic nervous system: some of the somato-autonomic evidence to date – part II: somatovisceral. Chiropr J Austr. 2010; 39:9-33.
26. Dishman DJ. Spinal reflex attenuation associated with spinal manipulation. Spine. 2000; 25:2519-2525.
27. Oppelt M, Juehring DD, Sorgenfry G et al. A case study utilizing spinal manipulation and dynamic neuromuscular

stabilization care to enhance function of a post cerebrovascular accident patient. J Bodyw Mov Ther. 2014; 18:17-22.
28. Juehring DD, Barber MR. A case study utilizing Vojta/ Dynamic Neuromuscular Stabilization therapy to control symptoms of a chronic migraine suffer. J Bodyw Mov Ther. 2011; 15:538-41.
29. Colloca CJ, Keller TS, Gunzburg R et al. Neurophysiological response to intraoperative lumbosacral spinal manipulation. J Manipulative Physiol Ther. 2000; 23:447-457.
30. Kolar P. Facilitation of agonist-antagonist activation by reflex stimulation methods. In: Liebeson, C (Ed.). Rehabilitation of the Spine: A Practitioner's Manual, second ed. Lippincott Williams & Williams, Philadelphia, 2007.

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Diagnosis of a 64-year-old patient presenting with suspected lumbar spinal stenosis: an evidence-based case report

Peter C. Emary, BSc, DC[1,2]

Objective: *To present an evidence-based case report on the diagnosis of a patient with suspected lumbar spinal stenosis (LSS).*

Case: *A 64-year-old man presented with signs and symptoms suggestive of LSS, but physical examination and diagnostic imaging findings were inconclusive. Other co-morbidities included diabetes, congestive heart failure, and left hip joint osteoarthritis.*

Outcome: *PubMed was searched for systematic reviews of diagnostic studies on LSS. Two recent articles were found and appraised with respect to their validity, importance, and applicability in diagnosing the current patient. Copies of his magnetic resonance imaging were also obtained and used in combination with the appraised literature, including diagnostic test specificities and likelihood ratios, to confirm an LSS diagnosis.*

Summary: *This case illustrates how research evidence*

Objectif : *Présenter une étude de cas fondée sur des éléments probants au sujet d'un patient souffrant possiblement d'une sténose du canal rachidien lombaire (SCRL).*

Cas : *Un homme de 64 ans s'est présenté avec des signes et des symptômes laissant croire à une SCRL, mais les résultats de l'examen clinique et de l'imagerie ne sont pas concluants. Le patient souffre de comorbidités, notamment du diabète, une insuffisance cardiaque congestive et de l'arthrose à la hanche gauche.*

Résultat : *On a réalisé une recherche dans PubMed pour trouver des évaluations systématiques d'études diagnostiques sur la SCRL. On a trouvé et évalué deux récents articles afin d'établir leur validité, importance et applicabilité pour diagnostiquer le trouble de santé du patient en question. On a aussi obtenu et utilisé des copies des IRM en combinaison avec les ouvrages évalués, notamment la spécificité d'un test diagnostique et les rapports de vraisemblance, pour confirmer la SCRL.*

Résumé : *Ce cas montre comment les résultats de recherche peuvent servir dans la pratique clinique, en*

[1] Master of Science (MSc) Candidate, MSc Advanced Professional Practice (Clinical Sciences), Anglo-European College of Chiropractic, 13-15 Parkwood Road, Bournemouth, Dorset, BH5 2DF, United Kingdom
[2] Private Practice, 201C Preston Parkway, Cambridge, ON, N3H 5E8, Canada
Phone: 519-653-2101. E-mail: pcemary@hotmail.com
Consent: The patient has provided written consent to having his personal health information, including radiographs, published.

can be used in clinical practice, particularly in the diagnosis of an individual patient.

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KEY WORDS: lumbar spinal stenosis, diagnosis, chiropractic, evidence based practice, case reports

particulier dans le diagnostic du trouble de santé d'un patient.

(JCCA 2015; 59(1):46-52)

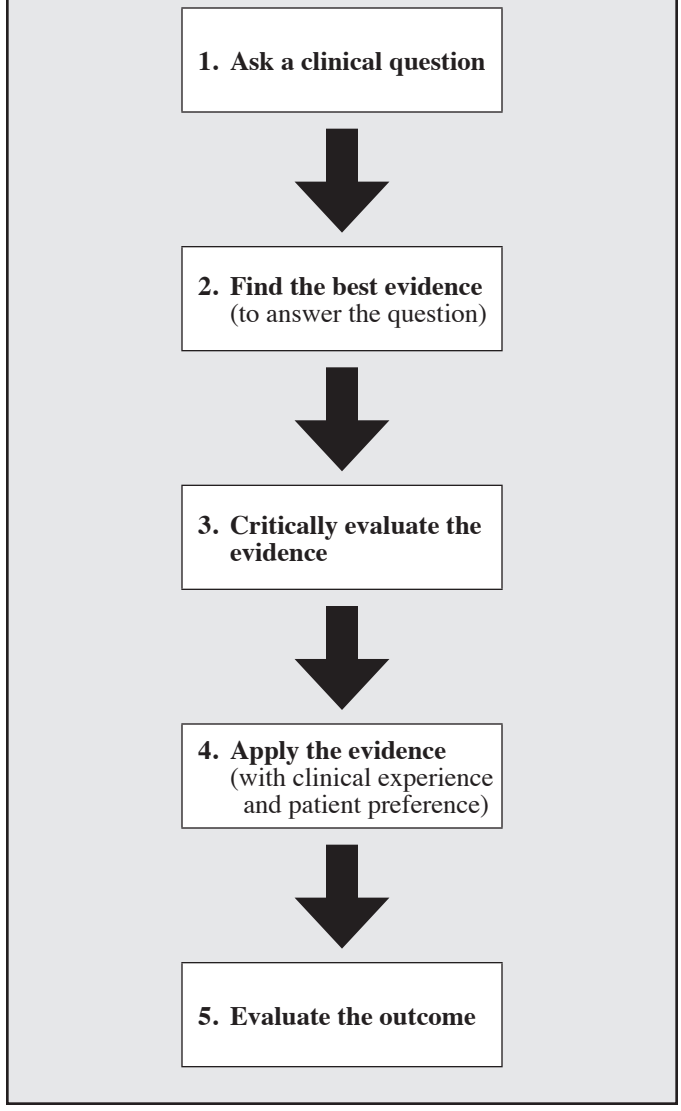
MOTS CLÉS : sténose du canal rachidien lombaire, diagnostic, chiropratique, pratique basée sur des données probantes, études de cas

Introduction

The North American Spine Society defines *lumbar spinal stenosis (LSS)* as "a clinical syndrome of buttock or lower extremity pain, which may occur with or without back pain, associated with diminished space available for the neural and vascular elements in the lumbar spine."[1] When symptomatic, its characteristic provocative and palliative features include exercise or positionally induced neurogenic claudication (i.e. radiating leg pain with walking or prolonged standing) that is typically relieved with forward flexion, sitting, and/or lying down. Yet despite these characteristic features, there is no generally accepted "gold standard" for the diagnosis of LSS.[2] Moreover, its radiographic findings often correlate poorly with patient symptoms.[3] As a consequence, a wide range of clinical, electrodiagnostic, and radiological tests are used.[4] Complicating matters are the various differential diagnoses for LSS such as vascular claudication, referred lumbar and/or radicular pain, compression fractures, and hip osteoarthritis, which commonly co-exist in older adults.[3] For the chiropractor, an accurate diagnosis is important because it determines how these patients will be managed.

Using the five steps of evidence-based medicine[5] (Figure 1), the following case report will demonstrate how current research literature informed the diagnosis of a patient presenting with suspected LSS.

Figure 1.
The five steps of evidence-based medicine (based on the model by Sackett et al.[5]).



Case Report

A 64-year-old obese man presented with a chief complaint of chronic left-sided low back and lower extremity pain. He described it as a constant ache down his leg to the mid-shin, accompanied by "pins and needles." He also complained of "numbness" along his right anterolateral thigh. The pain severity on the left was graded between a six out of 10 at best and nine out of 10 at worst. His overall Back Bournemouth Questionnaire[6] score totalled 58

out of 70, where zero equals no disability and 70 equals complete disability. Bending or twisting at the waist (e.g. shovelling or vacuuming), standing up from sitting, dancing, and prolonged standing were all described as provocative; topical analgesics, acupuncture, sitting, and riding an exercise bike were palliative. He originally injured his lower back eight years ago lifting heavy 10 kilogram metal frames at work. After re-injuring it again two years later, he was taken off work by his family physician, and had been on long-term disability ever since. A recent magnetic resonance imaging (MRI) study revealed multiple lower lumbar degenerative changes (as noted in the radiologist's report), including a mild disc bulge at L3-4 and bilateral foraminal stenosis at L3-4 and L4-5. The patient had been taking daily oral analgesic (Percocet) and anti-inflammatory (Celebrex) medications; but because of ongoing lower back and leg pain, his physician referred him for chiropractic treatment.

On examination, he had difficulty getting out of a chair (i.e. Minor's sign) and walked with an antalgic left-sided limp. His lumbar spine range of motion while seated was painful and limited by 50% in extension, 50% in left rotation, and 25% in right and left lateral flexion. Motion palpation revealed restricted sacroiliac joints, along with lumbar paraspinal and gluteal muscle spasm on the left. Several orthopaedic tests[7] including the Straight Leg Raise, Double Leg Raise, Yeoman's, and seated Kemp's provoked his left-sided low back pain and leg pain, while Nachlas' revealed tight quadriceps muscles, bilaterally. Lower limb neurologic exam including motor, reflex, sensory, Babinski, and vibratory testing was unremarkable, except for diminished Achilles tendon reflexes, graded as +1 on the right and 0 on the left.

Based on his age, history, and physical examination findings, the working diagnosis was LSS. Before a definitive diagnosis could be established, however, other co-morbidities with possible overlapping symptoms needed to be considered. For instance, his positive Straight Leg Raise and Double Leg Raise test results, along with reported MRI findings of L3-4 and L4-5 foraminal stenosis, suggested an underlying diagnosis of lumbar radiculopathy. His medical history included diagnoses of type II diabetes and congestive heart failure, making peripheral neuropathy and/or vascular claudication possible differentials. He also had a history of severe and painful left hip joint osteoarthritis, and was on a surgical wait list for total hip arthroplasty. The question for this patient, therefore (as far as his chiropractic management was concerned), was how to confirm a clinical diagnosis of LSS?

Clinical Question

To answer this, the following foreground question was posed: *Based on this patient's history and physical examination findings, how likely is it that he actually has (or does not have) LSS?*

Using the mnemonic, *PICO* (i.e. **P**atient/problem, **I**ntervention, **C**omparison, and **O**utcome[s] of interest),[5] this question can be broken down as follows:

P = A 64-year-old man with suspected LSS.
I = Positive/negative history and physical examination findings.
C = None.
O = Diagnostic likelihood of LSS.

Literature Search

The best evidence to answer a clinical question about diagnosis is a systematic review of diagnostic studies.[8] To begin, therefore, a search of the English language literature (from inception to January 27, 2014; limited to human studies) was conducted using the PubMed database. Because of interest in diagnostic studies, the subheading '*diagnosis*' was selected under the medical subject heading '*spinal stenosis*,' and this was combined using the Boolean operator '*AND*' with the text word '*lumbar*.' This search yielded 1,096 hits. After filtering the search to '*systematic reviews*' from the past '*10 years*,' 18 total citations were produced. Inclusion criteria consisted of systematic reviews containing current information on the accuracy of clinical diagnostic tests for LSS. Six of the retrieved studies were deemed potentially relevant based on their titles, and abstract review identified two as pertinent.[3,4] Of the articles excluded, two were reviews of radiographic LSS parameters only,[2,9] one was not a systematic review,[1] and the other had been updated since its original publication.[10] Therefore, two recent systematic reviews pertaining to the accuracy of clinical diagnostic tests for LSS were identified and retrieved. The entire literature search, including retrieval of manuscripts, took less than 30 minutes.

Critical Evaluation of the Evidence

In both systematic reviews retrieved,[3,4] the clinical tests/symptoms found to be most useful in the diagnosis of LSS included the following: absence of pain when seated, improvement when bending forward, bilateral buttock or leg pain, neurogenic claudication, a wide-based gait, abnormal Romberg test, and a score of seven or higher on a diagnostic support tool of history and physical examination findings. (The corresponding sensitivities, specificities, likelihood ratios [LRs], and 95% confidence intervals [CIs] of all these tests are shown in Table 1; the 'diagnostic support tool' for LSS[11] is shown in more detail in Table 2.) Before these results could be applied to the current patient, the two systematic reviews were first appraised using a template provided by Sackett *et al*.[5] Specifically, the papers were appraised with respect to their (i) validity, (ii) importance, and (iii) applicability to the diagnosis of the current patient.

(i) Are the results of these systematic reviews of diagnostic studies valid?

Both articles by Suri *et al*.[3] and de Schepper *et al*.[4] were qualitative systematic reviews of diagnostic studies. Both included a methods section that described finding and including all relevant studies, as well as an assessment of their individual validity. (Only diagnostic studies with clearly described clinical tests and reference standards were included.) Across these studies, a variety of diagnostic tests for LSS were evaluated; but between the two systematic reviews, there was consistency of results concerning the accuracy of these tests. Therefore based on the above criteria, the results of both systematic reviews were deemed valid.

(ii) Are the valid results of these systematic reviews important?

The results were deemed important for several reasons. First of all, the clinical findings listed in Table 1 were all found to be highly specific and some highly sensitive for LSS. Essentially, when a test or symptom has high specificity, a positive result will rule in the diagnosis.[5] Likewise, a negative result for a test with high sensitivity will rule out the diagnosis. Secondly (when positive), these clinical findings were all shown to increase the likelihood of LSS by at least 3-fold or greater. LRs ≥2.0 have been considered to produce meaningful changes in the prob-

Table 1.
Diagnostic accuracy of history and physical examination findings for LSS.

Clinical Finding	Sensitivity (95% CI)	Specificity (95% CI)	Positive LR (95% CI)	Negative LR (95% CI)
Absence of pain when seated[3]	0.47 (0.32-0.61)	0.94 (0.85-1.00)	7.4 (1.9-30)	0.57 (0.43-0.76)
Improvement when bending forward[3,4]	0.52 (0.45-0.58)	0.92 (0.88-0.95)	6.4 (4.1-9.9)	0.52 (0.46-0.60)
Bilateral buttock or leg pain[3,4]	0.51 (0.40-0.62)	0.92 (0.87-0.97)	6.3 (3.1-13)	0.54 (0.43-0.68)
Neurogenic claudication[3,4]	0.82 (0.77-0.87)	0.78 (0.73-0.83)	3.7 (2.9-4.8)	0.23 (0.17-0.31)
Wide-based gait[3,4]	0.42 (0.27-0.57)	0.97 (0.91-1.00)	13.0 (1.9-95)	0.60 (0.46-0.78)
Abnormal Romberg test[3,4]	0.40 (0.25-0.54)	0.91 (0.81-1.00)	4.2 (1.4-13)	0.67 (0.51-0.87)
Diagnostic support tool[4] (LSS score ≥7)	0.93 (0.89-0.96)	0.72 (0.66-0.78)	3.3 (2.7-4.0)	0.10 (0.06-0.16)

Table 2.
Prediction rule for identifying patients with LSS.[11]

Clinical Finding	Risk score assigned	Risk score for current patient
History		
Age (years)		
•60-70	1	1
•>70	2	0
Absence of diabetes	1	0
Neurogenic claudication	3	3
Exacerbation of symptoms when standing up	2	2
Symptom improvement when bending forward	3	3
Physical examination		
Symptoms induced by having patients bend forward	–1	0
Symptoms induced by having patients bend backward	1	1
Good peripheral artery circulation	3	Not performed
Abnormal Achilles tendon reflex	1	1
Straight leg raising positive for reproducing pain	–2	–2
Score interpretation		
Score range	–2 to 17	
Positive score	≥7	9

ability of a given diagnosis.[12] Thirdly, when the findings of 'neurogenic claudication' and the 'diagnostic support tool' are negative, the likelihood of LSS is reduced by 77% and 90% respectively. Finally because none of their CIs included the value of 1.0, these LRs were all shown to be statistically significant in the general population.[5]

(iii) Are the valid, important results of these systematic reviews applicable to our patient?

Each of the diagnostic tests listed in Table 1 were validated on LSS patients presenting to surgical and/or primary care medical clinics. Although LSS severity is expected to be high in these patient populations, the current patient also presented within a primary care setting,[13] and was similar in age and symptomatology to those in the aforementioned studies. Therefore, the results from the systematic reviews were deemed applicable to this patient.

Application of the Evidence

Although gait analysis and Romberg testing were not performed, the current patient did exhibit several of the clinical findings listed in Table 1. These included an absence of pain when seated (LR 7.4; 95% CI, 1.9-30), improvement when bending forward (LR 6.4; 95% CI, 4.1-9.9), and neurogenic claudication (LR 3.7; 95% CI, 2.9-4.8). All three of these findings are highly specific for LSS, thereby ruling in the diagnosis. Using the diagnostic support tool[11] shown in Table 2, the combined findings of age between 60-70, diabetes, neurogenic claudication, exacerbation of symptoms when standing up, symptom improvement when bending forward, provocation with bending backward, abnormal Achilles tendon reflexes, and a positive Straight Leg Raise, yielded a score of nine – a positive result (LR 3.3; 95% CI, 2.7-4.0). This finding also has good specificity for LSS. Furthermore, its LR can be interpreted and communicated to the patient as a 95% probability that he is between 2.7 and 4.0 times more likely to have LSS compared with a patient who tests negative with this diagnostic tool. The patient's other LRs were interpreted in a similar manner.

The post-test probability of actually having LSS was also determined using the patient's positive LRs (see Figure 2). Depending on the diagnostic criteria used, the prevalence of radiographic LSS in the general population of adults' aged 60-69 years ranges between 19.4% and 47.2%.[14] Assuming a primary care clinic prevalence (or

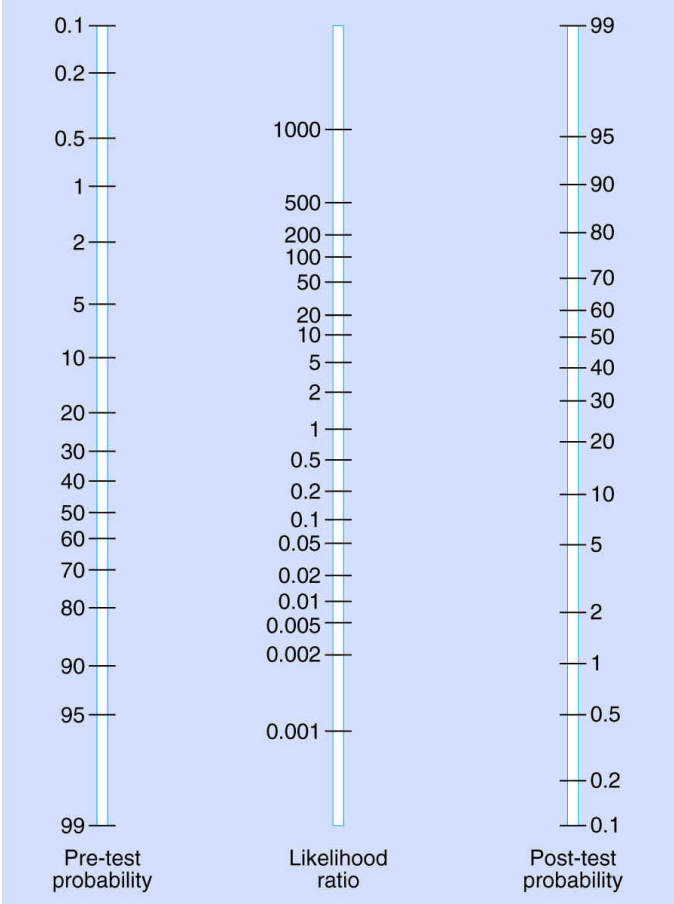


Figure 2.
Nomogram for determining the post-test probability of a target disorder.[5]
*To determine post-test probability, a line would be drawn between the patient's pre-test probability (**left**), through the LR of the diagnostic test (**centre**), and extended through to the post-test probability (**right**). Source: reprinted with permission from D.L. Sackett, S.E. Straus, W.S. Richardson, W. Rosenberg, and R.B. Haynes, Evidence-Based Medicine, 2nd ed., p. 79, M. Parkinson, © 2000 Churchill Livingstone.*

pre-test probability) of 19.4% and using only the finding of symptom improvement with sitting as an example, the probability of this patient's LSS diagnosis would increase to approximately 70%. However, if the clinic prevalence of LSS were 47.2%, his post-test probability would be 90%. In either case, LSS was likely. To confirm the pa-

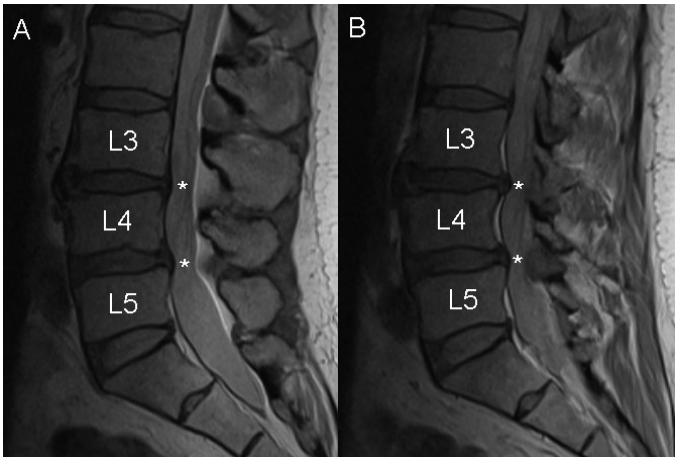


Figure 3.
*Proton density sagittal (**A**) and left para-sagittal (**B**) MR images of the patient's lumbar spine showing mild-to-moderate intervertebral disc dessication and protrusion into the thecal sac at L3-4 and L4-5 (**asterisks**).*

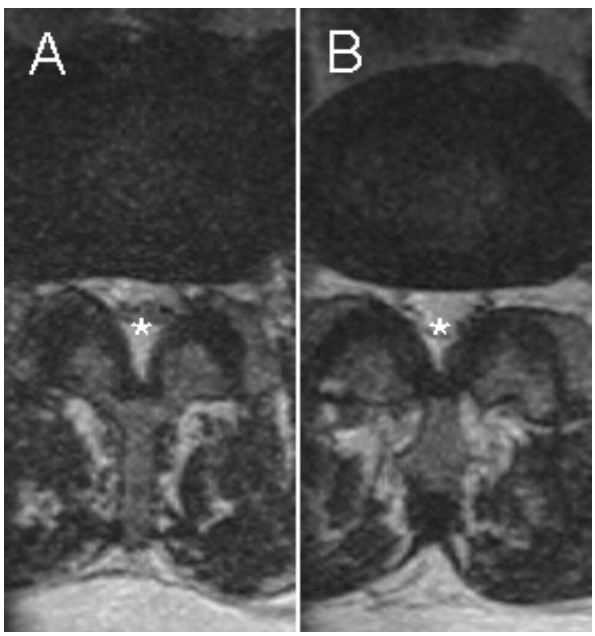


Figure 4.
*T2-weighted coronal spot view MR images of L3-4 (**A**) and L4-5 (**B**) showing disc protrusion, bilateral facet joint osteoarthritis, and ligamentum flavum hypertrophy, with resultant central canal (**asterisks**), lateral recess, and intervertebral foraminal stenosis (most severe at L3-4), bilaterally.*

tient's diagnosis, copies of his MR images were obtained, and these revealed further anatomical evidence of LSS (Figures 3 and 4).

Evaluation of the Outcome

Various conservative and surgical treatment options are available to patients with LSS.[1] However, because this case report was written within the context of an 'observation-only' clinical placement,[15] the author did not actually communicate a diagnosis (or plan of management) to the patient. (Instead, he was referred to one of the other chiropractors at the centre.) Nevertheless, this evidence-based case report illustrates how research literature can be retrieved relatively quickly, appraised, and used in the management of an individual patient.

Limitations

This case report has some limitations. Firstly, gait analysis and Romberg testing[7] were not performed as part of the neurological exam on this patient. Though specific and predictive of LSS, the clinical findings of a 'wide-based gait' and 'abnormal Romberg test' would not have altered the patient's final LSS diagnosis. Furthermore, in a recently published guideline[16] the North American Spine Society has concluded that there is insufficient evidence regarding the diagnostic accuracy of the Romberg test for LSS. Secondly, the literature search for this case report was limited to PubMed. By not searching other databases such as the Cochrane Library and/or the Cumulative Index to Nursing and Allied Health Literature, other pertinent systematic reviews of diagnostic studies may have been missed. Thirdly, other systematic review appraisal checklists such as the Amstar[17] were not employed in this case. Instead, the author relied on the template provided in the textbook by Sackett *et al*.[5]. Finally, the results of this case report may not be generalizable to other chiropractic patients or practices. For instance, this patient presented within a primary care, community health centre setting.[13] Integrated chiropractic services such as this have only been described within one other city in all of Canada.[18] What is more, many of the diagnostic tests highlighted in this case require further validation within non-specialized clinical settings,[3] once again limiting these results toward other chiropractic patients.

Summary

According to Sackett *et al*.,[5] "evidence-based medicine is the conscientious, explicit, and judicious use of the current best evidence in making decisions about the care of individual patients." In this case, a 64-year-old man presented with signs and symptoms suggestive of LSS, including several co-morbidities. However, by using the results of two recent systematic reviews – and in particular, their specificities and LRs – a more accurate diagnosis of LSS was reached.

References

1. Watters WC 3rd, Baisden J, Gilbert TJ, Kreiner S, Resnick DK, Bono CM, Ghiselli G, Heggeness MH, Mazanec DJ, O'Neill C, Reitman CA, Shaffer WO, Summers JT, Toton JF, North American Spine Society. Degenerative lumbar spinal stenosis: an evidence-based clinical guideline for the diagnosis and treatment of degenerative lumbar spinal stenosis. Spine J. 2008; 8(2):305-310.
2. Steurer J, Roner S, Gnannt R, Hodler J, LumbSten Research Collaboration. Quantitative radiologic criteria for the diagnosis of lumbar spinal stenosis: a systematic literature review. BMC Musculoskelet Disord. 2011; 12:175.
3. Suri P, Rainville J, Kalichman L, Katz JN. Does this older adult with lower extremity pain have the clinical syndrome of lumbar spinal stenosis? JAMA. 2010; 304(23):2628-2636.
4. de Schepper EIT, Overdevest GM, Suri P, Peul WC, Oei EHG, Koes BW, Bierma-Zeinstra SMA, Luijsterburg PAJ. Diagnosis of lumbar spinal stenosis: an updated systematic review of the accuracy of diagnostic tests. Spine (Phila Pa 1976). 2013; 38(8):E469-E481.
5. Sackett DL, Straus SE, Richardson WS, Rosenberg W, Haynes RB. Evidence-based Medicine: How to Practice and Teach EBM. 2nd ed. London: Churchill Livingstone; 2000.
6. Bolton JE, Breen AC. The Bournemouth Questionnaire: a short-form comprehensive outcome measure. I. Psychometric properties in back pain patients. J Manipulative Physiol Ther. 1999; 22(8):503-510.
7. Evans RC. Illustrated Essentials in Orthopedic Physical Assessment. St. Louis: Mosby; 1994.
8. Miller PJ, Jones-Harris AR. The evidence-based hierarchy: is it time for change? A suggested alternative. J Manipulative Physiol Ther. 2005; 28(6):453-457.
9. Andreisek G, Imhof M, Wertli M, Winklhofer S, Pfirrmann CW, Hodler J, Steurer J, Lumbar Spinal Stenosis Outcome Study Working Group Zurich. A systematic review of semiquantitative and qualitative radiologic criteria for the diagnosis of lumbar spinal stenosis. AJR Am J Roentgenol. 2013; 201(5):W735-W746.
10. de Graaf I, Prak A, Bierma-Zeinstra S, Thomas S, Peul W, Koes B. Diagnosis of lumbar spinal stenosis: a systematic review of the accuracy of diagnostic tests. Spine (Phila Pa 1976). 2006; 31(10):1168-1176.
11. Konno S, Hayashino Y, Fukuhara S, Kikuchi S, Kaneda K, Seichi A, Chiba K, Satomi K, Nagata K, Kawai S. Development of a clinical diagnosis support tool to identify patients with lumbar spinal stenosis. Eur Spine J. 2007; 16(11):1951-1957.
12. Pinksy LE, Wipf JE, Ramsey SD. Evidence-Based Clinical Practice: Concepts and Approaches. Boston: Butterworth-Heinemann; 2000.
13. Langs Community Health Centre: About Langs. Available from: http://www.langs.org/about-langs/ [Accessed 5 December 2013].
14. Kalichman L, Cole R, Kim DH, Li L, Suri P, Guermazi A, Hunter DJ. Spinal stenosis prevalence and association with symptoms: the Framingham Study. Spine J. 2009; 9(7):545-550.
15. MSc Advanced Professional Practice (Clinical Sciences). Anglo-European College of Chiropractic: Bournemouth. Available from: http://www.aecc.ac.uk/cpd/postgrad/app-clinical.aspx [Accessed 19 March 2014].
16. Kreiner DS, Shaffer WO, Baisden JL, Gilbert TJ, Summers JT, Toton JF, Hwang SW, Mendel RC, Reitman CA. An evidence-based clinical guideline for the diagnosis and treatment of degenerative lumbar spinal stenosis (update). Spine J. 2013; 13(7):734-743.
17. Shea BJ, Grimshaw JM, Wells GA, Boers M, Andersson N, Hamel C, Porter AC, Tugwell P, Moher D, Bouter LM. Development of AMSTAR: a measurement tool to assess the methodological quality of systematic reviews. BMC Med Res Methodol. 2007; 7:10.
18. Baskerville NB, Keenan D. How Chiropractors began working in a Community Health Centre in Ottawa. J Can Chiropr Assoc. 2005; 49(1):13-20.

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Is there a role for neck manipulation in elderly falls prevention? – An overview

Julie C. Kendall, BAppSc, MClinChiro[1]
Jan Hartvigsen, DC, PhD[2]
Simon D. French, BAppSc(Chiro), MPH, PhD[3]
Michael F. Azari, BAppSc(Chiro), BSc(Hons), PhD*[1,4]

Many risk factors exist for falls in the elderly. Dizziness is an important risk factor for such falls. Spinal pain has also been identified as a risk factor for these falls. In this overview of the literature, we examine studies, including trials, of neck manipulation for neck pain, unsteadiness and falls risk relevant to the elderly. We also examine two related, but not mutually exclusive, mechanisms through which a putative beneficial effect may be mediated. These are the effects of neck manipulation on neck pain and on non-specific dizziness. We focus on the available evidence primarily in terms of clinical data rather than laboratory-based measures of balance. We conclude that chiropractors may have a role in falls

Beaucoup de facteurs de risque de chute existent chez les personnes âgées. L'étourdissement fait partie des facteurs de risque importants. Les douleurs lombaires sont également considérées comme un facteur de risque de chute. Dans le présent aperçu d'ouvrage, on examine des études, y compris des essais, sur la manipulation cervicale pour traiter les douleurs cervicales, le manque d'assurance en position debout et les risques de chute chez les personnes âgées. On examine aussi deux mécanismes liés, mais non mutuellement exclusifs, à partir desquels il serait possible de véhiculer des améliorations estimées. Il s'agit des effets de la manipulation cervicale pour traiter les douleurs cervicales et les étourdissements non précisés. On se concentre sur les preuves disponibles principalement en matière de données cliniques, et non pas les mesures d'équilibre réalisées en laboratoire. Conclusion : les chiropraticiens ont vraisemblablement un rôle à jouer dans les stratégies de prévention des chutes chez les personnes âgées souffrant de douleurs cervicales

[1] Discipline of Chiropractic, School of Health Sciences, RMIT University, Melbourne, Australia
[2] Department of Sports Science and Clinical Biomechanics, University of Southern Denmark and Nordic Institute of Chiropractic and Clinical Biomechanics, Odense, Denmark
[3] School of Rehabilitation Therapy, Queens University, Kingston, Canada
[4] Health Innovations Research Institute, RMIT University, Melbourne, Australia
* Address correspondence to:
Dr. Michael Azari
RMIT University
PO Box 71 Bundoora VIC 3083 Australia
Tel: +61 3 9925 7744
Michael.Azari@rmit.edu.au
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prevention strategies in the subpopulation of the elderly that suffer from mechanical neck pain or dysfunction and non-specific dizziness. However, this role remains to be rigorously studied and properly defined.

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KEY WORDS: dizziness, elderly, risk factor, neck pain, manipulation, chiropractic

mécaniques ou d'un dysfonctionnement cervical, ainsi que d'étourdissements non précisés. Toutefois, ce rôle doit faire l'objet d'études rigoureuses et être défini adéquatement.

(JCCA 2015; 59(1):53-63)

MOTS CLÉS : étourdissement, personnes âgées, facteur de risque, douleur cervicale, manipulation, chiropratique

Introduction

Falls in the elderly can be due to many causes. Dizziness is an important risk factor for these falls. In this overview of the literature, we examine the relationship between non-specific dizziness, an important form of dizziness in the elderly[1], and neck pain and dysfunction. We further examine whether rigorous evaluation of neck manipulation is justified for the treatment of non-specific dizziness that is concomitant with chronic neck pain or dysfunction, with the aim of reducing the risk of falls in the elderly. This review does not focus on research data in relation to changes in laboratory-based measurements of balance such as postural sway and their changes with neck pain or neck manipulation.[2,3] Instead, we focus primarily on clinical research data. A non-systematic method using Pubmed searches was used to source the available literature on the subject. No language restrictions were applied. Care was taken to guard against inclusion or exclusion bias.

Falls in the elderly and dizziness

Many elderly patients with chronic neck pain and concomitant non-specific dizziness or unsteadiness consult chiropractors and other practitioners who perform spinal manipulative therapy (SMT). It has also been shown that at least the elderly patients who present to chiropractors in Auckland New Zealand and Melbourne Australia have risk factors for falls, including dizziness, that are comparable to the community dwelling elderly in general.[4] However, the possible therapeutic effect of the primary modality of chiropractic treatment, namely SMT, for non-specific dizziness and prevention of falls in the elderly is yet to be adequately investigated. It is well established that falls in the elderly constitute an important global health problem. Every year in the US[5] and Australia[6], approximately one in three elderly people fall, with 10-20% experiencing serious injury including fractures[7]. Similarly, a large population study has found the incidence of falls over a twelve month-period in Canadian elderly to be as high as 19.8%.[8] Falls in the elderly are associated with increased morbidity, disability, loss of independence and even death. Hence, they constitute a serious health problem with substantial human costs.[9,10] They account for 75% of all casualty visits in the elderly, and result in significant hospital stays.[9] The frequency of falls in the elderly increases sharply with age[11], making this health problem particularly significant in aging populations of the industrialised world. These falls also result in substantial economic costs to the individuals concerned and the health care system, with the estimated cost per fall between $2,000 to $42,000[12] and the total economic burden for falls $23.3 billion in the USA, with comparable substantial costs in the UK[13] and Canada[8]. In addition, falls in the elderly are an increasingly important global health problem. As the size of the elderly population grew in Australia, the annual direct health costs of fall-related injuries are estimated to almost triple in five decades from $498.2 million in 2001.[14]

Dizziness, which predisposes the elderly to falls[4,15-17], is also very common in the elderly population. For instance, a study from Scotland found the point prevalence for dizziness to be 30% in 893 elderly people[18], and the comparable proportion in Brazil has been reported as high as 45% in a cohort of 391 community dwelling elderly adults[19]. Moreover, the proportion of those above the age of 70 presenting with non-specific dizziness was recently reported as 63% in South Korea.[20] Moreover, dizziness becomes more prevalent with age.[21] There is a strong as-

sociation between dizziness and falls in the elderly.[15,22,23] A recent systematic review has also confirmed dizziness as a risk factor for falls in the elderly.[23] In short, falls, falls-related injuries, and dizziness are common and closely associated in the elderly.

The neck, postural balance and dizziness

Vestibular disorders are widely believed to constitute the most common cause of dizziness. However, there is evidence in the primary care setting that cardiovascular disease and related medications may be the most common cause in the elderly.[24] Consistent with this notion, vestibular disorders have been found to be much less prevalent in the general population than the symptoms of vertigo dizziness and unsteadiness.[16] It is generally accepted that the second most common cause of dizziness in the elderly (after benign positional paroxysmal vertigo) is what is termed "multisensory dizziness".[25,26] This condition is attributed to aging and deterioration of multiple sensory systems, namely the vestibular, optic and proprioceptive. The prevalence of dizziness in the elderly that can be attributed to pain and dysfunction of the cervical spine (cervicogenic dizziness) is not known. At least some patients diagnosed with multisensory dizziness may suffer from cervicogenic dizziness. For this reason, this review focuses on 'non-specific' rather than 'cervicogenic' dizziness.

It is established that somatic afferent information from the neck, particularly the upper cervical spine, converges with vestibular and visual inputs on central nervous system (CNS) nuclei involved in processing and integration of postural balance inputs. For instance, Hikosaka and Maeda demonstrated that somatic sensory information for upper cervical spine is relayed to motorneurons in the abducens nucleus modulating the vestibulo-ocular reflex that causes abduction of the eye to the contralateral side of head rotation to allow for fixed gaze.[27] Conversely, Corneil and colleagues showed that cervical spine muscles respond to stimulation of the superior colliculus[28], which is a primary CNS centre for processing of visual information. Furthermore, Shinoda and colleagues showed that stimulation of the semicircular canals in the inner ear is relayed to cervical motorneurons.[29] Finally, Peterson and co-workers demonstrated that the vestibulocollic and the cervicocollic reflexes interact and produce a summative effect on muscle activation in the neck.[30] Taken together these data convincingly demonstrate integration of vestibular, visual and proprioceptive (particularly from the neck) inputs to maintain postural balance. Given this integration, it is probable that abnormal cervical proprioceptive input to the CNS (as a result of injury, pain, or musculoskeletal dysfunction of the neck) may create a mismatch with the other inputs thereby causing dizziness.[31-33] This can be particularly so in the context of the elderly who suffer deterioration of multiple sensory systems.

In agreement with this premise, a recent study has found greater levels of sensorimotor dysfunction (particularly in terms of joint position error in the neck) in association with upper cervical pain than lower cervical pain[34] correlating with the higher density of proprioceptors in the upper cervical region. We also know that anaesthetising the deep structures of the neck, or unilateral sectioning of the cervical dorsal roots induce severe ataxia and disturbance of balance.[31,35] Equally importantly, stimulation of the abundant neck muscle spindle afferents[36], by the use of vibration, increases body sway[37,38], and influences the velocity and direction of gait and running[39,40]. Additionally, there is evidence that the elderly may be more reliant on proprioceptive input for maintenance of postural balance than younger people.[41] For instance, afferent input from the legs is important for postural control in healthy elderly people.[42] Furthermore, the elderly with polyneuropathy suffer from a higher risk of falls.[43] In addition, the inability to stand in tandem stance is associated with double the risk of falls in the elderly.[44] These data together demonstrate the importance of proprioception to postural balance, particularly in the elderly. They also support the notion that neck pain and/or disturbed proprioception may contribute, or act as a predisposing factor, to dizziness and falls.

Neck pain and dizziness

Neck pain is common in the general population. Cote and colleagues demonstrated in a large population study in 1997 that the point prevalence of neck pain in Saskatchewan adults was 22.2%.[45] This study also reported six month prevalence of: low intensity and low disability neck pain; high intensity low disability neck pain; and high intensity and moderately or severely disabling neck pain were found to be 39.7%, 10.1%, and 4.6% respectively.[45] In addition, neck pain is common in the elderly. Its prevalence has been estimated at 36.1% and 40.5% for men and women respectively in community dwelling elderly people in Australia.[46]

Musculoskeletal problems of the neck can cause disturbance of balance, which is termed "cervicogenic dizziness" "cervical dizziness" or "cervical vertigo".[47-50] Patients who have suffered whiplash neck injuries as a result of motor vehicle accidents often complain of dizziness and exhibit motor co-ordination deficits.[51,52] Persistent neck pain following whiplash injury has also been recently associated, in a small cohort, with impairments of a variety of dynamic and functional balance tasks such as a timed 10m walk.[53] These signs and symptoms are not surprising, due to the stretch and sheer forces involved in whiplash injury, which can damage vestibular and neck receptors. Furthermore, in cases of whiplash that warrant the diagnosis of mild traumatic brain injury (or concussion), it may be the damage to the brain itself that causes the common post-injury symptom of dizziness.[54] Nevertheless, it is important to note that dizziness balance deficits and joint position errors are also common in patients with non-traumatic neck pain.[33,51,55-62] It is likely then that pain originating from the neck may in itself be responsible for, or at least associated with, dizziness in these cases.

Not surprisingly, in a recent secondary analysis of a prospective cohort study of 516 randomly selected community dwelling elderly participants (aged 73-92 years old), risk factors associated with dizziness were correlated with risk of falls over a 12 month period. In this study, 42% of the participants reported dizziness in general.[63] Interestingly, self-reported neck or back pain was far more common in those reporting dizziness with a high degree of statistical significance. As expected, participants with dizziness, anxiety, depression and history of transient ischemic attacks were more likely to experience multiple falls (more than 2 falls). However, the strongest predictors of multiple falls were found to be neck and back pain as well as anxiety in this population.[63] Therefore, there may be a strong causal relationship between neck pain, dizziness, and multiple falls in at least a subpopulation of the elderly.

There are reports, in neck pain patients, of a correlation between cervical joint stiffness and hypertonicity of the upper cervical musculature on one hand, and the presence of dizziness on the other.[64,65] A recent study has found that in middle-aged chronic neck pain patients, the presence of vertigo is highly correlated with neck stiffness.[66] In addition, fatigue of the neck muscles disturbs standing balance.[32,60] Additionally, neck tenderness is associated with cervical vertigo in the elderly.[48] Taken together these data suggest that neck pain, and associated joint stiffness and muscular hypertonicity and tenderness, may cause postural imbalance possibly by altering the proprioceptive input to the CNS, or its processing by the CNS, leading to dizziness. This in turn, could predispose the individual, particularly the elderly, to falls.

Spinal manipulative therapy for mechanical neck pain

SMT, including joint manipulation and mobilisation, has been used clinically for neck pain by chiropractors and other health care practitioners for many years. Several studies have shown that SMT and spinal exercise regimes are significantly more effective than usual medical care in reducing neck pain.[67,68] A recent systematic review has found high quality evidence for greater short term pain relief with manual therapy and exercise over exercise alone.[69] The same review also found evidence, although of low quality, for clinically important long term improvement in pain and functional status with manual therapy and exercise compared to no treatment.[69] Similarly, Maiers and colleagues have recently reported short-term effectiveness of SMT in the elderly. Theirs was the first randomised controlled trial (RCT) of cervical SMT in community dwelling elderly comparing its effectiveness for chronic mechanical neck pain with home and supervised exercises using 241 participants.[70] They found that the SMT and home exercise group had a statistically significant reduction in pain at the conclusion of the treatment period compared to either the home exercise alone or home exercise and supervised exercise groups, even though this effect was not sustained at 40- or even 14-weeks follow-up.

In a 2010 Cochrane systematic review of 27 RCTs, Gross and colleagues found low quality evidence for the benefits of manipulation and mobilisation compared to control for short-term neck pain reduction.[71] In another 2010 systematic review of manual therapy for neck pain, D'Sylva and colleagues reported low quality evidence suggesting a clinically significant improvement in pain relief, improved function and global perceived effect when manipulation and mobilisation were combined with massage. Once again, poor methodological quality of the studies and inadequate control for bias prevented a more conclusive assessment of the potential of SMT to

treat neck pain. It is important to realise that compared to drug trials, rigorous RCTs of SMT are more challenging to design and the choice of control intervention is particularly problematic due to the hands-on nature of manual therapy. It is not possible to blind the clinician with respect to the treatment delivered, and it is difficult to blind the participant. Control interventions for SMT that have been used to date often are other physical treatment modalities, such as mobilisation, or exercise therapy. Given that mobilisation, exercise and similar "control" interventions are likely to exert a therapeutic effect on the neck, the search for a more inert control for these studies needs to continue. Using a truly inert control may demonstrate greater therapeutic effect for SMT in treating neck pain. Efforts are currently underway to develop such a control.[72,73] Nevertheless, SMT has shown at least short-term effectiveness in treating neck pain. It has also been found to be a relatively safe intervention[74,75] particularly in the elderly[76].

Spinal manipulative therapy for non-specific dizziness

Usual treatment of non-specific dizziness in the elderly comprises mainly of vestibular rehabilitation.[41,77,78] According to a recent Cochrane systematic review, there is moderate evidence to support the use of vestibular rehabilitation for vertigo and dizziness of vestibular origin, such as benign paroxysmal positional vertigo (BPPV) and Ménière's disease.[79] However, non-specific dizziness is a diagnosis by exclusion, and it has no established satisfactory treatment at present.[20] In the elderly, patients with non-specific dizziness are often diagnosed with multisensory dizziness or presbyastasis (age related decreased vestibular function) and are often inadequately treated. Nonetheless, their treatment often involves vestibular manoeuvres and vestibular rehabilitation exercises.[80] Vestibular rehabilitation has been found to provide some relief for non-specific dizziness, alone and in combination with other therapies. For instance, vestibular rehabilitation neck exercises alone have been shown to be moderately effective for non-specific dizziness in a retrospective study of 153 elderly cases.[20] Furthermore, Andersson's group conducted two small RCTs, with dizziness due to a range of aetiologies, of vestibular rehabilitation combined with cognitive behavioural therapy (CBT) compared to a waiting list control.[81,82] These studies showed significant improvement with dizziness using provocative head movements, but no improvement in depression, anxiety or stress. Although, results from these studies are limited by small sample sizes and variability in treatment protocols, they do show promise for the use of neck rehabilitation exercises in treatment of non-specific dizziness, particularly in the elderly population. However, while these studies have aimed their treatment at rehabilitation of the vestibular system, the role of proprioceptive input from the coupling movements of the neck joints during these movements cannot be discounted. Therefore, focused modulation or rehabilitation of proprioceptive input from the neck in non-specific dizziness, is an area deserving of concerted research attention.

There is, in fact, a growing body of preliminary evidence for physical and manual treatment of the neck for non-specific dizziness. A course of individualised physiotherapy including massage and cervical and thoracic joint mobilisation has been reported to improve dizziness in a cohort of 20 neck pain patients.[64] Matsui and co-workers have recently reported that a course of primarily conservative physical therapy, was able to substantially reduce dizziness in neck pain patients.[65] A recent feasibility study has also reported significant improvement in dizziness handicap inventory (DHI) scores with Osteopathic SMT in 16 adults with chronic vertigo.[83] Similarly, improvement in DHI scores has been reported in a small group of elders in a recent pragmatic single group pre-test/post-test chiropractic intervention study.[84] In addition, a pilot randomised study of chiropractic care, including SMT, by the same group has reported improvement in both pain and DHI scores in the elderly.[85] These observations are consistent with anecdotal evidence from practitioners of SMT that some patients presenting with neck pain also report an improvement with their sense of balance. There are a number of clinical studies including five RCTs on the efficacy of manual therapy for cervicogenic dizziness.[57,64,88,89,96,97] However, two systematic reviews have concluded that there is limited to moderate evidence for the use of manual therapies, particularly cervical manipulation and mobilisation, in the management of this condition.[86,87] These two systematic reviews analysed five RCTs and 14 non-randomised controlled trials or cohort studies.[57,64,88-106] The overall quality of these data was found to be moderate to low due to lack of control groups, and inadequate blinding and randomisation. In spite of these

limitations, symptomatic improvement in dizziness was consistently reported. Treatments included mobilisation and or manipulation alone[88-94] and in combination with other therapies such as acupuncture[95], massage[57,64,96-103], traction[99,104], ergonomic advice[57,64,105], anti-inflammatory medications[95,98,105], and home exercises[57,64,98,106]. These data show promising preliminary evidence that warrants rigorous research in this area.

Conclusion

At present there is no satisfactory treatment for non-specific or age related dizziness. However, there is preliminary evidence that physical treatment of the neck may improve balance in neck pain patients. Therefore, it is important to examine the possible therapeutic effect of chiropractic interventions (particularly SMT) directed at the neck in treatment of this condition and prevention of falls in this subpopulation of the elderly. The need for this research is particularly acute given the substantial health and economic costs of falls in the elderly and the aging of the population in the industrialised world. We know that postural stability and motor control relies on integration of proprioceptive vestibular and visual inputs by the CNS, inputs that are widely believed to deteriorate with age. Neck pain is widely believed to be capable of compromising mechanisms of postural balance by distorting the proprioceptive input from the neck to the CNS. It is possible that integration of incongruous inputs by CNS balance centres becomes more challenging as proprioceptive visual and vestibular sensory mechanisms age. Whilst neck manipulation, a commonly practiced treatment by chiropractors, has shown effectiveness for neck pain, it has not been adequately evaluated for non-specific dizziness. However, there is encouraging preliminary data that seems to support the use of neck manipulation in treating this condition. Given that both spinal pain and dizziness are risk factors for balance deficits and falls in the elderly, rigorously designed randomised controlled trials are needed in this area. These trials should focus on both efficacy and effectiveness of the various chiropractic neck manipulation and mobilisation techniques and strategies on: neck pain; non-specific dizziness; dizziness-related disability; and falls frequency in the community dwelling elderly population.

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References:

1. Dros J, Maarsingh OR, van der Windt DA, Oort FJ, ter Riet G, de Rooij SE, et al. Profiling dizziness in older primary care patients: an empirical study. PloS one. 2011;6(1):e16481. PubMed PMID: 21304984. Pubmed Central PMCID: PMC3031582. Epub 2011/02/10. eng.
2. Ruhe A, Fejer R, Walker B. Does postural sway change in association with manual therapeutic interventions? A review of the literature. Chiropractic & Manual Therapies. 2013;21(1):9. PubMed PMID: 23374610. Pubmed Central PMCID: PMC3575324. Epub 2013/02/05. eng.
3. Ruhe A, Fejer R, Walker B. On the relationship between pain intensity and postural sway in patients with non-specific neck pain. J Back Musculoskeletal Rehabilitation. 2013 Jan 1;26(4):401-9. PubMed PMID: 23948827. Epub 2013/08/21. eng.
4. Holt KR, Noone PL, Short K, Elley CR, Haavik H. Fall risk profile and quality-of-life status of older chiropractic patients. J Manip Physiol Ther. 2011 Feb;34(2):78-87. PubMed PMID: 21334539. Epub 2011/02/22. eng.
5. Alexander BH, Rivara FP, Wolf ME. The cost and frequency of hospitalization for fall-related injuries in older adults. Am J Public Health. 1992 Jul;82(7):1020-3. PubMed PMID: 1609903. Pubmed Central PMCID: 1694056. Epub 1992/07/01. eng.
6. Pointer S, Harrison J, Bradley C. National injury prevention plan. Priorities for 2004 and beyond: Discussion paper. Injury Research and Statistics Series. 2003 July;18.
7. Kruschinski C, Sheehy O, Hummers-Pradier E, Lelorier J. Fracture risk of patients suffering from dizziness: a retrospective cohort study. Euro J General Practice. 2010 Dec;16(4):229-35. PubMed PMID: 20849315. Epub 2010/09/21. eng.
8. Sibley KM, Voth J, Munce SE, Straus SE, Jaglal SB. Chronic disease and falls in community-dwelling Canadians over 65 years old: a population-based study exploring associations with number and pattern of chronic conditions. BMC Geriatrics. 2014;14:22. PubMed PMID: 24529293. Pubmed Central PMCID: PMC3928582. Epub 2014/02/18. eng.
9. Campbell AJ, Borrie MJ, Spears GF, Jackson SL, Brown JS, Fitzgerald JL. Circumstances and consequences of falls experienced by a community population 70 years and over during a prospective study. Age and Ageing. 1990 Mar;19(2):136-41. PubMed PMID: 2337010. Epub 1990/03/01. eng.
10. Cripps R, Carman J. Falls by the elderly in Australia:

Trends and data for 1998. Injury Research and Statistics Series. 2001;6.
11. Kirchhoff M, Melin A. Screening for fall risk in the elderly in the capital region of Copenhagen: the need for fall assessment exceeds the present capacity. Danish Medical Bulletin. 2011 Oct;58(10):A4324. PubMed PMID: 21975155. Epub 2011/10/07. eng.
12. Heinrich S, Rapp K, Rissmann U, Becker C, Konig HH. Cost of falls in old age: a systematic review. Osteoporosis International: a journal established as result of cooperation between the European Foundation for Osteoporosis and the National Osteoporosis Foundation of the USA. 2010 Jun;21(6):891-902. PubMed PMID: 19924496. Epub 2009/11/20. eng.
13. Davis JC, Robertson MC, Ashe MC, Liu-Ambrose T, Khan KM, Marra CA. International comparison of cost of falls in older adults living in the community: a systematic review. Osteoporosis International: a journal established as result of cooperation between the European Foundation for Osteoporosis and the National Osteoporosis Foundation of the USA. 2010 Aug;21(8):1295-306. PubMed PMID: 20195846. Epub 2010/03/03. eng.
14. Moller J. Projected costs of fall related injury to older persons due to demographic change in Australia. Report to Commonwealth Department of Health and Aging. 2003 July.
15. Tinetti ME, Williams CS, Gill TM. Health, functional, and psychological outcomes among older persons with chronic dizziness. J Am Geriatrics Society. 2000 Apr;48(4):417-21. PubMed PMID: 10798469. Epub 2000/05/08. eng.
16. Bisdorff A, Bosser G, Gueguen R, Perrin P. The epidemiology of vertigo, dizziness, and unsteadiness and its links to co-morbidities. Frontiers in Neurology. 2013;4:29. PubMed PMID: 23526567. Pubmed Central PMCID: PMC3605504. Epub 2013/03/26. eng.
17. Kerber KA, Enrietto JA, Jacobson KM, Baloh RW. Disequilibrium in older people: a prospective study. Neurology. 1998 Aug;51(2):574-80. PubMed PMID: 9710038. Epub 1998/08/26. eng.
18. Colledge NR, Wilson JA, Macintyre CC, MacLennan WJ. The prevalence and characteristics of dizziness in an elderly community. Age and Ageing. 1994 Mar;23(2):117-20. PubMed PMID: 8023718. Epub 1994/03/01. eng.
19. de Moraes SA, Soares WJ, Ferriolli E, Perracini MR. Prevalence and correlates of dizziness in community-dwelling older people: a cross sectional population based study. BMC Geriatrics. 2013;13:4. PubMed PMID: 23290128. Pubmed Central PMCID: PMC3545826. Epub 2013/01/08. eng.
20. Jung JY, Kim JS, Chung PS, Woo SH, Rhee CK. Effect of vestibular rehabilitation on dizziness in the elderly. Am J Otolaryngology. 2009 Sep-Oct;30(5):295-9. PubMed PMID: 19720245. Epub 2009/09/02. eng.
21. Jonsson R, Sixt E, Landahl S, Rosenhall U. Prevalence of dizziness and vertigo in an urban elderly population. J Vestibular Res: equilibrium & orientation. 2004;14(1):47-52. PubMed PMID: 15156096. Epub 2004/05/25. eng.
22. Perez-Jara J, Olmos P, Abad MA, Heslop P, Walker D, Reyes-Ortiz CA. Differences in fear of falling in the elderly with or without dizzines. Maturitas. 2012 Nov;73(3):261-4. PubMed PMID: 22853871. Epub 2012/08/03. eng.
23. Deandrea S, Lucenteforte E, Bravi F, Foschi R, La Vecchia C, Negri E. Risk factors for falls in community-dwelling older people: a systematic review and meta-analysis. Epidemiology (Cambridge, Mass). 2010 Sep;21(5):658-68. PubMed PMID: 20585256. Epub 2010/06/30. eng.
24. Maarsingh OR, Dros J, Schellevis FG, van Weert HC, van der Windt DA, ter Riet G, et al. Causes of persistent dizziness in elderly patients in primary care. Annals Family Med. 2010 May-Jun;8(3):196-205. PubMed PMID: 20458102. Pubmed Central PMCID: PMC2866716. Epub 2010/05/12. eng.
25. Kao AC, Nanda A, Williams CS, Tinetti ME. Validation of dizziness as a possible geriatric syndrome. J Am Geriatrics Society. 2001 Jan;49(1):72-5. PubMed PMID: 11207845. Epub 2001/02/24. eng.
26. Wetmore SJ, Eibling DE, Goebel JA, Gottshall KR, Hoffer ME, Magnusson M, et al. Challenges and opportunities in managing the dizzy older adult. Otolaryngology – Head and Neck Surgery: official journal of American Academy of Otolaryngology-Head and Neck Surgery. 2011 May;144(5):651-6. PubMed PMID: 21493351. Epub 2011/04/16. eng.
27. Hikosaka O, Maeda M. Cervical effects on abducens motoneurons and their interaction with vestibulo-ocular reflex. Experimental Brain Research Experimentelle Hirnforschung Experimentation Cerebrale. 1973 Dec 20;18(5):512-30. PubMed PMID: 4794882. Epub 1973/12/20. eng.
28. Corneil BD, Olivier E, Munoz DP. Neck muscle responses to stimulation of monkey superior colliculus. I. Topography and manipulation of stimulation parameters. J Neurophysiology. 2002 Oct;88(4):1980-99. PubMed PMID: 12364523. Epub 2002/10/05. eng.
29. Shinoda Y, Sugiuchi Y, Futami T, Ando N, Kawasaki T, Yagi J. Synaptic organization of the vestibulo-collic pathways from six semicircular canals to motoneurons of different neck muscles. Progress Brain Res. 1993;97:201-9. PubMed PMID: 8234746. Epub 1993/01/01. eng.
30. Peterson BW, Bilotto G, Goldberg J, Wilson VJ. Dynamics of vestibulo-ocular, vestibulocollic, and cervicocollic reflexes. Annals of the New York Academy

of Sciences. 1981;374:395-402. PubMed PMID: 6951444. Epub 1981/01/01. eng.
31. de Jong PT, de Jong JM, Cohen B, Jongkees LB. Ataxia and nystagmus induced by injection of local anesthetics in the Neck. Annals Neurology. 1977 Mar;1(3):240-6. PubMed PMID: 407834. Epub 1977/03/01. eng.
32. Gosselin G, Rassoulian H, Brown I. Effects of neck extensor muscles fatigue on balance. Clinical Biomechanics (Bristol, Avon). 2004 Jun;19(5):473-9. PubMed PMID: 15182982. Epub 2004/06/09. eng.
33. Karlberg M, Johansson R, Magnusson M, Fransson PA. Dizziness of suspected cervical origin distinguished by posturographic assessment of human postural dynamics. J Vestibular Res: equilibrium & orientation. 1996 Jan-Feb;6(1):37-47. PubMed PMID: 8719508. Epub 1996/01/01. eng.
34. Treleaven J, Clamaron-Cheers C, Jull G. Does the region of pain influence the presence of sensorimotor disturbances in neck pain disorders? Manual Therapy. 2011 Dec;16(6):636-40. PubMed PMID: 21890397. Epub 2011/09/06. eng.
35. Ishikawa K, Matsuzaki Z, Yokomizo M, Terada N, Miyazaki S, Togawa K. Effect of unilateral section of cervical afferent nerve upon optokinetic response and vestibular nystagmus induced by sinusoidal rotation in guinea pigs. Acta oto-laryngologica Supplementum. 1998;537:6-10. PubMed PMID: 9870641. Epub 1998/12/31. eng.
36. Boyd-Clark LC, Briggs CA, Galea MP. Muscle spindle distribution, morphology, and density in longus colli and multifidus muscles of the cervical spine. Spine. 2002 Apr 1;27(7):694-701. PubMed PMID: 11923661. Epub 2002/03/30. eng.
37. Kavounoudias A, Gilhodes JC, Roll R, Roll JP. From balance regulation to body orientation: two goals for muscle proprioceptive information processing? Experimental Brain Res Experimentelle Hirnforschung Experimentation Cerebrale. 1999 Jan;124(1):80-8. PubMed PMID: 9928792. Epub 1999/02/03. eng.
38. Pyykko I, Aalto H, Seidel H, Starck J. Hierarchy of different muscles in postural control. Acta oto-laryngologica Supplementum. 1989;468:175-80. PubMed PMID: 2635499. Epub 1989/01/01. eng.
39. Bove M, Courtine G, Schieppati M. Neck muscle vibration and spatial orientation during stepping in place in humans. J Neurophysiology. 2002 Nov;88(5):2232-41. PubMed PMID: 12424265. Epub 2002/11/09. eng.
40. Courtine G, Papaxanthis C, Laroche D, Pozzo T. Gait-dependent integration of neck muscle afferent input. Neuroreport. 2003 Dec 19;14(18):2365-8. PubMed PMID: 14663192. Epub 2003/12/10. eng.
41. Ricci NA, de Faria Figueiredo Goncalves D, Coimbra AM, Coimbra IB. Sensory interaction on static balance: a comparison concerning the history of falls of community-dwelling elderly. Geriatrics & Gerontology International. 2009 Jun;9(2):165-71. PubMed PMID: 19740360. Epub 2009/09/11. eng.
42. Kristinsdottir EK, Fransson PA, Magnusson M. Changes in postural control in healthy elderly subjects are related to vibration sensation, vision and vestibular asymmetry. Acta oto-laryngologica. 2001 Sep;121(6):700-6. PubMed PMID: 11678169. Epub 2001/10/27. eng.
43. Richardson JK. Factors associated with falls in older patients with diffuse polyneuropathy. J Am Geriatrics Society. 2002 Nov;50(11):1767-73. PubMed PMID: 12410893. Epub 2002/11/02. eng.
44. Hansson EE, Mansson NO, Ringsberg KA, Hakansson A. Falls among dizzy patients in primary healthcare: an intervention study with control group. International J Rehabil Res Internationale Zeitschrift fur Rehabilitationsforschung Revue internationale de recherches de readaptation. 2008 Mar;31(1):51-7. PubMed PMID: 18277204. Epub 2008/02/16. eng.
45. Cote P, Cassidy JD, Carroll L. The Saskatchewan Health and Back Pain Survey. The prevalence of neck pain and related disability in Saskatchewan adults. Spine. 1998 Aug 1;23(15):1689-98. PubMed PMID: 9704377. Epub 1998/08/15. eng.
46. March LM, Brnabic AJ, Skinner JC, Schwarz JM, Finnegan T, Druce J, et al. Musculoskeletal disability among elderly people in the community. The Medical J Austr. 1998 May 4;168(9):439-42. PubMed PMID: 9612455. Epub 1998/06/05. eng.
47. Yacovino DA. Cervical vertigo: myths, facts, and scientific evidence. Neurologia (Barcelona, Spain). 2012 Sep 13. PubMed PMID: 22981375. Epub 2012/09/18. Vertigo cervical: mitos, realidades y evidencia cientifica. Eng
48. Morinaka S. Musculoskeletal diseases as a causal factor of cervical vertigo. Auris, Nasus, Larynx. 2009 Dec;36(6):649-54. PubMed PMID: 19493640. Epub 2009/06/06. eng.
49. Michels T, Lehmann N, Moebus S. Cervical vertigo – cervical pain: an alternative and efficient treatment. J Alternative and Complementary Med (New York, NY). 2007 Jun;13(5):513-8. PubMed PMID: 17604554. Epub 2007/07/03. eng.
50. Kristjansson E, Treleaven J. Sensorimotor function and dizziness in neck pain: implications for assessment and management. J Orthopaedic Sports Physical Therapy. 2009 May;39(5):364-77. PubMed PMID: 19411769. Epub 2009/05/05. eng.
51. Sjolander P, Michaelson P, Jaric S, Djupsjobacka M. Sensorimotor disturbances in chronic neck pain – range of motion, peak velocity, smoothness of movement, and repositioning acuity. Manual Therapy. 2008 May;13(2):122-31. PubMed PMID: 17197230. Epub 2007/01/02. eng.

52. Hinoki M. Vertigo due to whiplash injury: a neurotological approach. Acta oto-laryngologica Supplementum. 1984;419:9-29. PubMed PMID: 6599233. Epub 1984/01/01. eng.
53. Stokell R, Yu A, Williams K, Treleaven J. Dynamic and functional balance tasks in subjects with persistent whiplash: a pilot trial. Manual Therapy. 2011 Aug;16(4):394-8. PubMed PMID: 21367648. Epub 2011/03/04. eng.
54. Hartvigsen J, Boyle E, Cassidy JD, Carroll LJ. Mild traumatic brain injury after motor vehicle collisions: what are the symptoms and who treats them? A population-based 1-year inception cohort study. Arch Phys Med Rehabi. 2014 Mar;95(3 Suppl):S286-94. PubMed PMID: 24581914. Epub 2014/03/04. eng.
55. Michaelson P, Michaelson M, Jaric S, Latash ML, Sjolander P, Djupsjobacka M. Vertical posture and head stability in patients with chronic neck pain. J Rehabil Med: official journal of the UEMS European Board of Physical and Rehabilitation Medicine. 2003 Sep;35(5):229-35. PubMed PMID: 14582555. Epub 2003/10/30. eng.
56. Karlberg M, Persson L, Magnusson M. Impaired postural control in patients with cervico-brachial pain. Acta oto-laryngologica Supplementum. 1995;520 Pt 2:440-2. PubMed PMID: 8749184. Epub 1995/01/01. eng.
57. Karlberg M, Magnusson M, Malmstrom EM, Melander A, Moritz U. Postural and symptomatic improvement after physiotherapy in patients with dizziness of suspected cervical origin. Arch Phys Med Rehabil. 1996 Sep;77(9):874-82. PubMed PMID: 8822677. Epub 1996/09/01. eng.
58. Koskimies K, Sutinen P, Aalto H, Starck J, Toppila E, Hirvonen T, et al. Postural stability, neck proprioception and tension neck. Acta oto-laryngologica Supplementum. 1997;529:95-7. PubMed PMID: 9288281. Epub 1997/01/01. eng.
59. McPartland JM, Brodeur RR, Hallgren RC. Chronic neck pain, standing balance, and suboccipital muscle atrophy – a pilot study. J Manip Physiol Thera. 1997 Jan;20(1):24-9. PubMed PMID: 9004119. Epub 1997/01/01. eng.
60. Schieppati M, Nardone A, Schmid M. Neck muscle fatigue affects postural control in man. Neuroscience. 2003;121(2):277-85. PubMed PMID: 14521987. Epub 2003/10/03. eng.
61. Sjostrom H, Allum JH, Carpenter MG, Adkin AL, Honegger F, Ettlin T. Trunk sway measures of postural stability during clinical balance tests in patients with chronic whiplash injury symptoms. Spine. 2003 Aug 1;28(15):1725-34. PubMed PMID: 12897500. Epub 2003/08/05. eng.
62. Field S, Treleaven J, Jull G. Standing balance: a comparison between idiopathic and whiplash-induced neck pain. Manual Therapy. 2008 Jun;13(3):183-91. PubMed PMID: 17306599. Epub 2007/02/20. eng.
63. Menant JC, Wong A, Sturnieks DL, Close JC, Delbaere K, Sachdev PS, et al. Pain and anxiety mediate the relationship between dizziness and falls in older people. J Am Geriatrics Society. 2013 Mar;61(3):423-8. PubMed PMID: 23351026. Epub 2013/01/29. eng.
64. Malmstrom EM, Karlberg M, Melander A, Magnusson M, Moritz U. Cervicogenic dizziness – musculoskeletal findings before and after treatment and long-term outcome. Disability and Rehabilitation. 2007 Aug 15;29(15):1193-205. PubMed PMID: 17653993. Epub 2007/07/27. eng.
65. Matsui T, Ii K, Hojo S, Sano K. Cervical neuro-muscular syndrome: discovery of a new disease group caused by abnormalities in the cervical muscles. Neurologia medico-chirurgica. 2012;52(2):75-80. PubMed PMID: 22362287. Epub 2012/03/01. eng.
66. Yahia A, Ghroubi S, Jribi S, Malla J, Baklouti S, Ghorbel A, et al. Chronic neck pain and vertigo: Is a true balance disorder present? Annals Phys Rehabil Med. 2009 Sep-Oct;52(7-8):556-67. PubMed PMID: 19747892. Epub 2009/09/15. eng.
67. Bronfort G, Evans R, Nelson B, Aker PD, Goldsmith CH, Vernon H. A randomized clinical trial of exercise and spinal manipulation for patients with chronic neck pain. Spine. 2001 Apr 1;26(7):788-97; discussion 98-9. PubMed PMID: 11295901. Epub 2001/04/11. eng.
68. Harkness EF, Macfarlane GJ, Silman AJ, McBeth J. Is musculoskeletal pain more common now than 40 years ago?: Two population-based cross-sectional studies. Rheumatology (Oxford, England). 2005 Jul;44(7):890-5. PubMed PMID: 15784630. Epub 2005/03/24. eng.
69. Miller J, Gross A, D'Sylva J, Burnie SJ, Goldsmith CH, Graham N, et al. Manual therapy and exercise for neck pain: A systematic review. Manual Therapy. 2010 Jun 1. PubMed PMID: 20627797. Epub 2010/07/16. Eng.
70. Maiers M, Bronfort G, Evans R, Hartvigsen J, Svendsen K, Bracha Y, et al. Spinal manipulative therapy and exercise for seniors with chronic neck pain. The Spine Journal: official journal of the North American Spine Society. 2013 Nov 10. PubMed PMID: 24225010. Epub 2013/11/15. Eng.
71. Gross A, Miller J, D'Sylva J, Burnie SJ, Goldsmith CH, Graham N, et al. Manipulation or mobilisation for neck pain: a Cochrane Review. Manual Therapy. 2010 Aug;15(4):315-33. PubMed PMID: 20510644. Epub 2010/06/01. eng.
72. Walker BF, Losco B, Clarke BR, Hebert J, French S, Stomski NJ. Outcomes of usual chiropractic, harm & efficacy, the ouch study: study protocol for a randomized controlled trial. Trials. 2011;12:235. PubMed PMID: 22040597. Pubmed Central PMCID: 3224760. Epub 2011/11/02. eng.

73. Vernon H, Triano JT, Soave D, Dinulos M, Ross K, Tran S. Retention of blinding at follow-up in a randomized clinical study using a sham-control cervical manipulation procedure for neck pain: secondary analyses from a randomized clinical study. J Manip Physiol Thera. 2013 Oct;36(8):522-6. PubMed PMID: 24011656. Epub 2013/09/10. eng.
74. Gouveia LO, Castanho P, Ferreira JJ. Safety of chiropractic interventions: a systematic review. Spine. 2009 May 15;34(11):E405-13. PubMed PMID: 19444054. Epub 2009/05/16. eng.
75. Thiel HW, Bolton JE, Docherty S, Portlock JC. Safety of chiropractic manipulation of the cervical spine: a prospective national survey. Spine. 2007 Oct 1;32(21):2375-8; discussion 9. PubMed PMID: 17906581. Epub 2007/10/02. eng.
76. Rothwell DM, Bondy SJ, Williams JI. Chiropractic manipulation and stroke: a population-based case-control study. Stroke; a journal of cerebral circulation. 2001 May;32(5):1054-60. PubMed PMID: 11340209. Epub 2001/09/06. eng.
77. Belal A, Jr., Glorig A. Dysequilibrium of ageing (presbyastasis). J Laryngology and Otology. 1986 Sep;100(9):1037-41. PubMed PMID: 3760685. Epub 1986/09/01. eng.
78. Cohen HS, Kimball KT. Increased independence and decreased vertigo after vestibular rehabilitation. Otolaryngology – Head and Neck Surgery: official journal of American Academy of Otolaryngology-Head and Neck Surgery. 2003 Jan;128(1):60-70. PubMed PMID: 12574761. Epub 2003/02/08. eng.
79. Hillier SL, McDonnell M. Vestibular rehabilitation for unilateral peripheral vestibular dysfunction. Cochrane Database of Systematic Reviews (Online). 2011 (2):CD005397. PubMed PMID: 21328277. Epub 2011/02/18. eng.
80. Yardley L, Burgneay J, Andersson G, Owen N, Nazareth I, Luxon L. Feasibility and effectiveness of providing vestibular rehabilitation for dizzy patients in the community. Clinical Otolaryngology and Allied Sciences. 1998 Oct;23(5):442-8. PubMed PMID: 9800081. Epub 1998/11/04. eng.
81. Andersson G, Asmundson GJ, Denev J, Nilsson J, Larsen HC. A controlled trial of cognitive-behavior therapy combined with vestibular rehabilitation in the treatment of dizziness. Behaviour Research and Therapy. 2006 Sep;44(9):1265-73. PubMed PMID: 16290817. Epub 2005/11/18. eng.
82. Johansson M, Akerlund D, Larsen HC, Andersson G. Randomized controlled trial of vestibular rehabilitation combined with cognitive-behavioral therapy for dizziness in older people. Otolaryngology – Head and Neck Surgery: official journal of American Academy of Otolaryngology-Head and Neck Surgery. 2001 Sep;125(3):151-6. PubMed PMID: 11555746. Epub 2001/09/14. eng.
83. Fraix M. Osteopathic manipulative treatment and vertigo: a pilot study. PM & R: the journal of injury, function, and rehabilitation. 2010 Jul;2(7):612-8. PubMed PMID: 20659716. Epub 2010/07/28. eng.
84. Hawk C, Cambron J. Chiropractic care for older adults: effects on balance, dizziness, and chronic pain. J Manip Physiol Thera. 2009 Jul-Aug;32(6):431-7. PubMed PMID: 19712785. Epub 2009/08/29. eng.
85. Hawk C, Cambron JA, Pfefer MT. Pilot study of the effect of a limited and extended course of chiropractic care on balance, chronic pain, and dizziness in older adults. J Manip Physiol Thera. 2009 Jul-Aug;32(6):438-47. PubMed PMID: 19712786. Epub 2009/08/29. eng.
86. Reid SA, Rivett DA. Manual therapy treatment of cervicogenic dizziness: a systematic review. Manual Therapy. 2005 Feb;10(1):4-13. PubMed PMID: 15681263. Epub 2005/02/01. eng.
87. Lystad RP, Bell G, Bonnevie-Svendsen M, Carter CV. Manual therapy with and without vestibular rehabilitation for cervicogenic dizziness: a systematic review. Chiropractic & Manual Therapies. 2011;19(1):21. PubMed PMID: 21923933. Pubmed Central PMCID: PMC3182131. Epub 2011/09/20. eng.
88. Reid SA, Rivett DA, Katekar MG, Callister R. Sustained natural apophyseal glides (SNAGs) are an effective treatment for cervicogenic dizziness. Manual Therapy. 2008 Aug;13(4):357-66. PubMed PMID: 17951095. Epub 2007/10/24. eng.
89. Kang F, Wang QC, Ye YG. [A randomized controlled trial of rotatory reduction manipulation and acupoint massage in the treatment of younger cervical vertigo]. Zhongguo gu shang = China J Orthopaedics and Traumatology. 2008 Apr;21(4):270-2. PubMed PMID: 19102186. Epub 2008/12/24. chi.
90. Konrad K, Gerencser F. Manuelle Therapie Bei Schwindelpatienten. Manuelle Medizin. 1990;28(4):62-4.
91. Mahlstedt K, Westhofen M, Konig K. [Therapy of functional disorders of the craniovertebral joints in vestibular diseases]. Laryngo-rhino-otologie. 1992 May;71(5):246-50. PubMed PMID: 1616544. Epub 1992/05/01. Zur Therapie funktioneller Kopfgelenksstorungen bei Vestibularisaffektionen. ger.
92. Chen L, Zhan HS. [An transcranial Doppler ultrasonography and X-ray study of cervical vertigo patients treated by manipulation in supine position]. Zhong xi yi jie he xue bao = J Chinese Integrative Medicine. 2003 Nov;1(4):262-4. PubMed PMID: 15339526. Epub 2004/09/02. chi.
93. Zhou W, Jiang W, Li X, Zhang Y, Zhang J, Wu Z. Clinical study on manipulative treatment of derangement of the atlantoaxial joint. Journal Traditional Chinese

Medicine = Chung i tsa chih ying wen pan / sponsored by All-China Association of Traditional Chinese Medicine, Academy of Traditional Chinese Medicine. 1999 Dec;19(4):273-8. PubMed PMID: 10921131. Epub 2000/08/02. eng.
94. Galm R, Rittmeister M, Schmitt E. Vertigo in patients with cervical spine dysfunction. European Spine J: official publication of the European Spine Society, the European Spinal Deformity Society, and the European Section of the Cervical Spine Research Society. 1998;7(1):55-8. PubMed PMID: 9548360. Pubmed Central PMCID: PMC3615355. Epub 1998/04/21. eng.
95. Heikkila H, Johansson M, Wenngren BI. Effects of acupuncture, cervical manipulation and NSAID therapy on dizziness and impaired head repositioning of suspected cervical origin: a pilot study. Manual Therapy. 2000 Aug;5(3):151-7. PubMed PMID: 11034885. Epub 2000/10/18. eng.
96. Fang J. [Observation of curative effect on fixed-point spin reduction of spinal manipulation therapy for cervical vertigo]. Zhongguo gu shang = China J Orthopaedics and Traumatology. 2010 Feb;23(2):99-101. PubMed PMID: 20345030. Epub 2010/03/30. chi.
97. Du HG, Wei H, Huang MZ, Jiang Z, Ye SL, Song HQ, et al. [Randomized controlled trial on manipulation for the treatment of cervical vertigo of high flow velocity type]. Zhongguo gu shang = China J Orthopaedics and Traumatology. 2010 Mar;23(3):212-5. PubMed PMID: 20415082. Epub 2010/04/27. chi.
98. Bracher ES, Almeida CI, Almeida RR, Duprat AC, Bracher CB. A combined approach for the treatment of cervical vertigo. J Manipul Physiol Thera. 2000 Feb;23(2):96-100. PubMed PMID: 10714534. Epub 2000/03/14. eng.
99. Hülse M, Hölzl M. Vestibulospinale reaktionen bei der zervikogenen gleichgewichtsstorung: Die zervikogene unsicherheit. Hno. 2000;48(4):295-301.
100. Wu JR, Fang M, Hu J, Shen GQ, Jiang SY. [Effects of manipulation on head repositioning skill in patients with cervical vertigo]. Zhong xi yi jie he xue bao = J Chinese Integrative Medicine. 2006 Jan;4(1):76-8. PubMed PMID: 16409977. Epub 2006/01/18. chi.
101. Wu JR, Fang M, Hu J, Shen GQ, Jiang SY. Action of tuina on retro-positioning of skull spatial offset in patients with cervical vertigo. J Acupuncture and Tuina Science. 2008;6(2):83-6.
102. Strunk RG, Hawk C. Effects of chiropractic care on dizziness, neck pain, and balance: a single-group, preexperimental, feasibility study. J Chiropr Med. 2009 Dec;8(4):156-64. PubMed PMID: 19948306. Pubmed Central PMCID: PMC2786230. Epub 2009/12/02. eng.
103. Biesinger E. C2 and C3 cervical nerve root syndrome: The influence of cervical spine dysfunction on ENT symptoms. [German] Das C2/C3-Syndrom: Der Einfluss zervikaler Afferenzen auf HNO-arztliche Krankheitsbilder. Manuelle Medizin. 1997;35(1):12-9. PubMed PMID: 1997093838. German.
104. Uhlemann C, Gramowski KH, Endres U, Callies R. Manual diagnosis and therapy in cervical giddiness. Manuelle Diagnostik und Therapie Beim Halsbedingten Schwindel. 1993 //;31(4):77-81.
105. Wing LW, Hargrave Wilson W. Cervical vertigo. Australian and New Zealand J Surg. 1974;44(3):275-7. PubMed PMID: 0975134195. English.
106. Reid SA, Rivett DA, Katekar MG, Callister R. Comparison of mulligan sustained natural apophyseal glides and maitland mobilizations for treatment of cervicogenic dizziness: a randomized controlled trial. Physical Therapy. 2014 Apr;94(4):466-76. PubMed PMID: 24336477. Epub 2013/12/18. eng.

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A variant extensor indicis muscle and the branching pattern of the deep radial nerve could explain hand functionality and clinical symptoms in the living patient

Myroslava Kumka, MD, PhD*

The purpose of this study is to document the topographic anatomy of an extensor indicis (EI) muscle with a double tendon and the associated distribution of the deep branch of the radial nerve (DBRN). Both EI tendons were positioned deep to the tendons of the extensor digitorum as they traversed the dorsal osseofibrous tunnel. They then joined the medial slips of the extensor expansion of the second and third digits. In all other dissected forearms, a tendon of the EI muscle joined the medial slip of the extensor expansion to the index finger. The DBRN provided short branches to the superficial extensor muscles, long branches to the abductor pollicis longus and extensor pollicis brevis muscles, and terminated as the posterior interosseous nerve. Descending deep to the extensor pollicis longus muscle, the posterior interosseous nerve sent branches to the extensor pollicis brevis and EI muscles. Understanding of the topographic anatomy of an EI with a double tendon, and the associated distribution of the DBRN,

L'objectif de cette étude est de documenter l'anatomie topographique du musculus extensor indicis (MEI), ou muscle extenseur de l'index, avec un tendon double et la distribution de la grosse branche du nerf radial (DGBNR) qui lui est associée. Les deux tendons du MEI sont positionnés profondément dans les tendons du extensor digitorum alors qu'ils traversent le tunnel osso-fibreux dorsal. Ils joignent ensuite les portions internes de l'expansion de l'extenseur au niveau du deuxième et troisième doigt. Dans tous les autres avant-bras disséqués, un tendon du MEI joint la portion interne de l'expansion de l'extenseur à l'index. La DGBNR fournit de petites branches aux muscles d'extenseur superficiels, de grosses branches aux muscles du long abducteur du pouce et du court extenseur du pouce, pour finir en tant que nerf interosseux postérieur. Le nerf interosseux postérieur descend profondément dans le muscle du long extenseur du pouce et envoie des branches au court extenseur du pouce et au MEI. Une bonne compréhension de l'anatomie topographique d'un MEI avec double tendon, et la DGBNR associée,

* Department of Anatomy, Canadian Memorial Chiropractic College, Ontario, Canada.
Correspondence should be addressed to:
Myroslava Kumka, MD, PhD,
Canadian Memorial Chiropractic College, Department of Anatomy,
6100 Leslie Street, Toronto, ON M2H 3J1, Canada
Tel: (416) 482-2340 ext:175
Email: mkumka@cmcc.ca

may contribute to accurate diagnosis and treatment of hand lesions.

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KEY WORDS: extensor indicis, neuropathy, radial nerve, variation, wrist pain

peut favoriser des diagnostics précis et améliorer le traitement de lésions aux mains.

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MOTS CLÉS : musculus extensor indicis, neuropathie, nerf radial, variation, douleur au poignet

Introduction

Anatomical variations of the extensor muscles of the hand, including the extensor indicis (EI), are common, and no standard pattern has been described.[1-13] These variations are often a matter of concern to clinicians as they may lead to a misdiagnosis. For example, a variant EI muscle may give rise to dorsal wrist pain and hence may be diagnosed incorrectly as a ganglion, soft tissue tumor, synovial cyst or tenovaginitis.[14-18] For surgeons, the variations of EI tendons are of particular clinical importance, since this is the muscle that is commonly used to perform graft and tendon transfer operations and should be considered during extensor tendon exploration for trauma.[5,7,19] Since hand surgery relies mainly on applied anatomy, it is essential to revisit not only the anatomy of the presented variance of the EI muscle but also the distribution of the deep branch of the radial nerve (DBRN) as it may help to explain clinical symptoms developed by the partial or complete entrapment of the main trunk of this nerve or its branches.[20-24] To our knowledge, no studies have investigated the correlation between the variant EI and the branching pattern of the radial nerve. Even though the distribution of the DBRN is complicated and inconsistent,[23] enhancing our understanding of the relationship between the radial nerve and the variant EI may contribute to accurate diagnosis, effective surgical procedures, and the modeling of neurovascular compartments.[25] Familiarity with the variations of extensor muscles and their innervation are crucial for assessment and treatment of pathologic conditions with magnetic resonance imaging, diagnostic sonography, and magnetic resonance neurography.[26-30]

The rationale of the presented study is to document the existence and topographic anatomy of the EI muscle with a double tendon and the associated distribution of the DBRN. This is done to enhance clinicians' awareness, so as to provide adequate assessment and treatment of hand lesions. It may also be useful for modeling of neurovascular and muscular compartments of the forearm and hand.

The following questions are posed: i) Could the presented variant in the morphology of the EI muscle explain hand functionality and clinical symptoms in the living patient?, ii) What branching pattern of the radial nerve should be expected in the case of an EI with a double tendon?, iii) What are the clinical implications of the branching pattern of the DBRN?

We present the topographic anatomy of the EI muscle with a double tendon and discuss its functional and clinical implications. The distribution of the DBRN within forearms possessing single or double tendons of the EI muscle is investigated and its clinical significance is discussed.

Material and methods

This study utilized 16 embalmed cadavers (32 upper limbs) of both genders and of different ages using anatomical macro-and microdissections. An 89-year-old male had a double tendon of the extensor indicis muscle bilaterally. This variant muscle was dissected carefully to expose its origin, course and insertion. All of the forearms were dissected to examine the distribution of the radial nerve branches within the posterior forearm to determine whether the branching pattern of the radial nerve varies significantly within the forearms possessing single or double tendons of the EI muscle. The specimens were documented and photographed.

Results

In an 89-year–old male cadaver, the EI muscle with a double tendon to the second and third digits was found bilaterally within the deep layer of the extensor muscle compartment of the forearm (Figure 1A). This variant muscle originated from the posterior surface of the distal third of

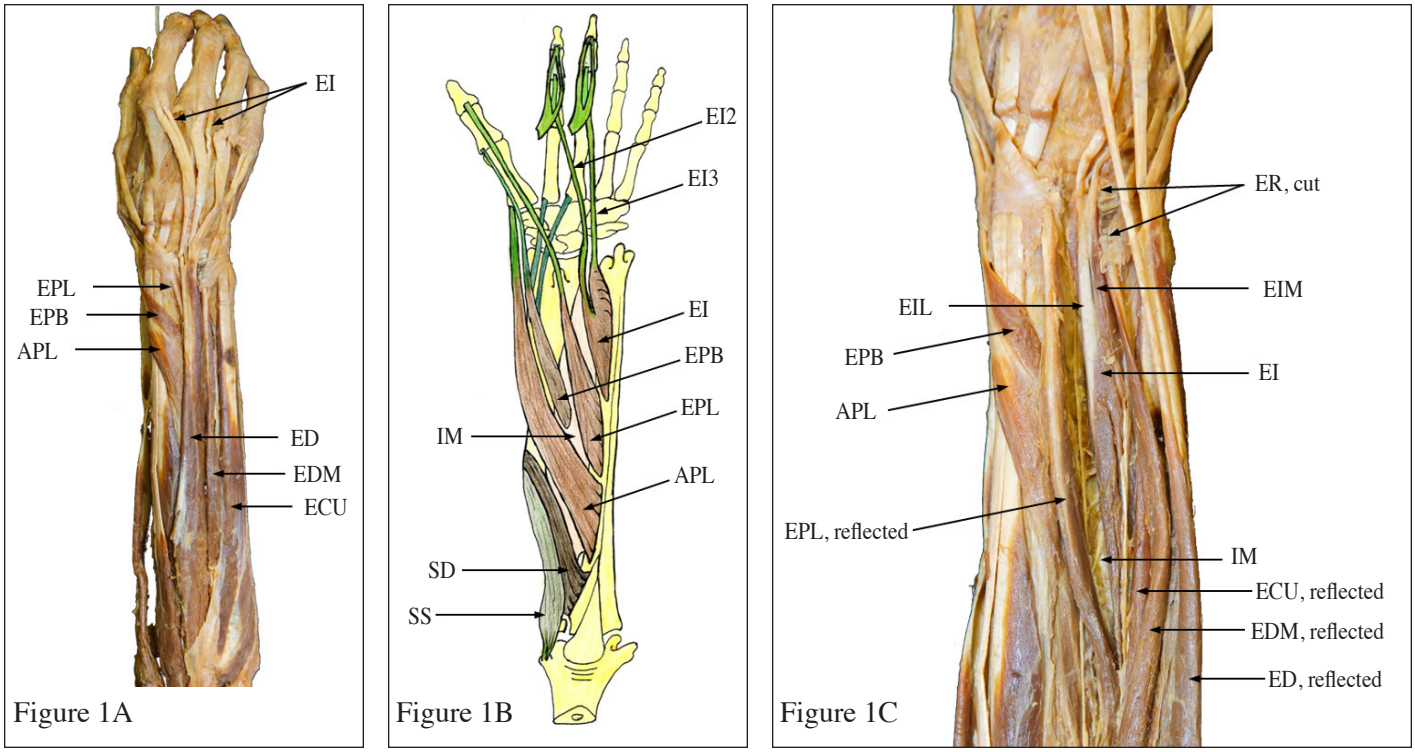

Figure 1

Topography of the extensor indicis (EI) muscle with a double tendon within the muscular layers of the extensor compartment of the forearm.

A – The superficial muscular layer includes ECU, extensor carpi ulnaris; EDM, extensor digiti minimi; ED, extensor digitorum. The deep layer includes EPL, extensor pollicis longus; EPB, extensor pollicis brevis; APL, abductor pollicis longus; EI with a double tendon.

B – Schematic representation illustrates the deep muscular layer. EI2, extensor indicis to the second digit; EI3, extensor indicis to the third digit; SS, supinator superficial part; SD, supinator deep part; APL; EPL; EPB; IM, interosseous membrane.

C – The extensor indicis lateral part (EIL) of the muscle belly becomes tendinous proximal to the extensor retinaculum (ER). The extensor indicis medial part (EIM) is longer with the musculotendinous junction descending deep to the superior margin of the ER. Both tendons of the EI are positioned medial and deep to the tendons of the ED, as they traverse the osseofibrous tunnel under the ER.

the shaft of the ulna, the adjacent interosseous membrane, and from the internal surface of the antebrachial fascia occupying the interface of the superficial-deep forearm extensors (Figure 1B). The lateral part of the muscle belly was shorter and became tendinous proximal to the extensor retinaculum. However, the medial part was longer, with the musculotendinous junction descending deep to the superior margin of the extensor retinaculum. Both tendons of the EI were positioned medial and deep to the tendons of the extensor digitorum (ED) muscle as they traversed the fourth osseofibrous tunnel under the extensor retinaculum (Figure 1C). On the dorsum of the hand, opposite the heads of the second and third metacarpal bones, the two tendons of the EI joined the ulnar sides of the tendons of the ED, enhancing the medial slips of the extensor expansion (EE) for the second and third digits

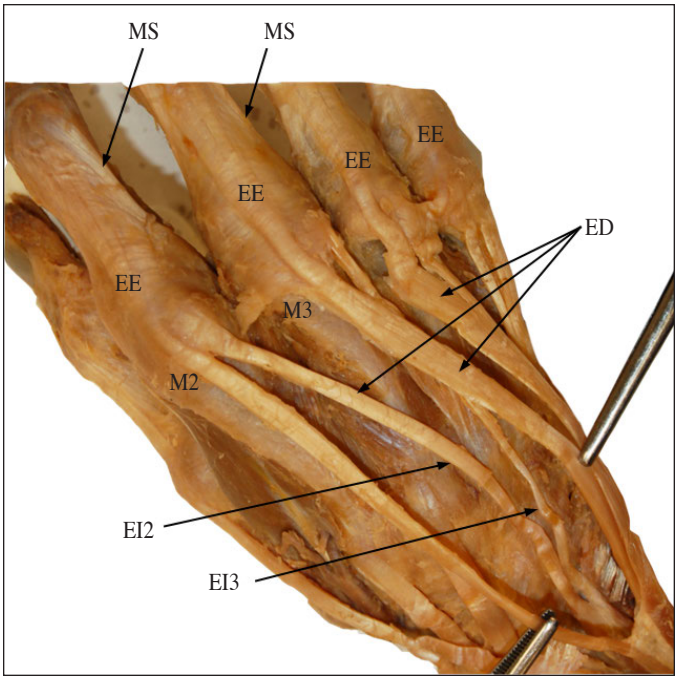

Figure 2
The distal attachments of the EI double tendon. On the dorsum of the hand, opposite the heads of the second metacarpal (M2) and the third metacarpal (M3) bones, the tendons of the EI to the second (EI2), and third (EI3) digits join the ulnar sides of the ED, enhancing the medial slips (MS) of the extensor expansion (EE) of the second and third digits.

(Figure 2). In all other dissected forearms, a tendon of EI muscle joined the EE to the index finger.

In consideration of the clinical importance of the distribution of the DBRN, we dissected 32 upper limbs to determine whether the branching pattern of the DBRN varied between the forearms possessing single versus double tendons of EI. The radial nerve arose from the posterior cord of the brachial plexus and incorporated the anterior primary rami of C5-T1 spinal nerves. From the axillary fossa, it wound around the posterior aspect of the humerus and pierced the lateral intermuscular septum to enter the anterior compartment of the arm dividing into superficial and deep terminal branches. In the literature, the DBRN is frequently called the posterior interosseous nerve (PIN).[31] Following the international anatomical terminology,[32] we classify the PIN as the terminal long branch of the DBRN.

The DBRN traversed the supinator muscle between the superficial and deep layers, emerging in the posterior compartment deep to the superficial layer of the extensor muscles, immediately providing the short branches to the ED, extensor digiti minimi, and extensor carpi ulnaris muscles. Descending superficial to the abductor pollicis longus (APL), the DBRN provided the long branches to the APL and the extensor pollicis brevis (EPB), and terminated as the PIN (Figures 3A and 3B). The PIN descended deep to the extensor pollicis longus (EPL) muscle on the posterior surface of the interosseous membrane sending two to three short nerve branches to supply the EPB and EI muscles (Figure 3C). Next, the PIN continued within the fourth dorsal osseofibrous tunnel between the tendon of the EPL and the lateral part of the EI terminating within the dorsal fascia and the dorsal surface of the fibrous capsules of the radiocarpal and intercarpal joints.

Discussion

It is important understand the anatomy of the variant EI muscle with a double tendon as this variant may explain clinical symptoms of certain hand lesions and should be considered in the differential diagnosis of a swelling, ganglion or other soft tissue tumors on the dorsum of the hand.[1,5,9,16]

Knowledge of the variant EI muscle with a double tendon is required for precise extensor muscle identification and confirmation of a preoperative diagnosis using dynamic sonography or magnetic resonance imaging, as well as for the planning of the best possible surgical treatments in this region.[28-38]

Schmidt et al.[39] argued that hand surgery informed by applied anatomy, and collaboration between anatomists and clinicians are essential to upgrading surgical technique and optimizing patient care.

The present study is congruent with the observations of Ritter et al.[40] and Doyle[41] with regard to the proximal attachments of the presented variant EI muscle to the posterior surface of the_shaft of the ulna, the adjacent interosseous membrane, and the connective tissue septum between the EI and the EPL muscles. Additionally, in the present case, the variant EI muscle attached to the internal surface of the antebrachial fascia within the superficial–deep extensor fascial plane. We hypothesize that

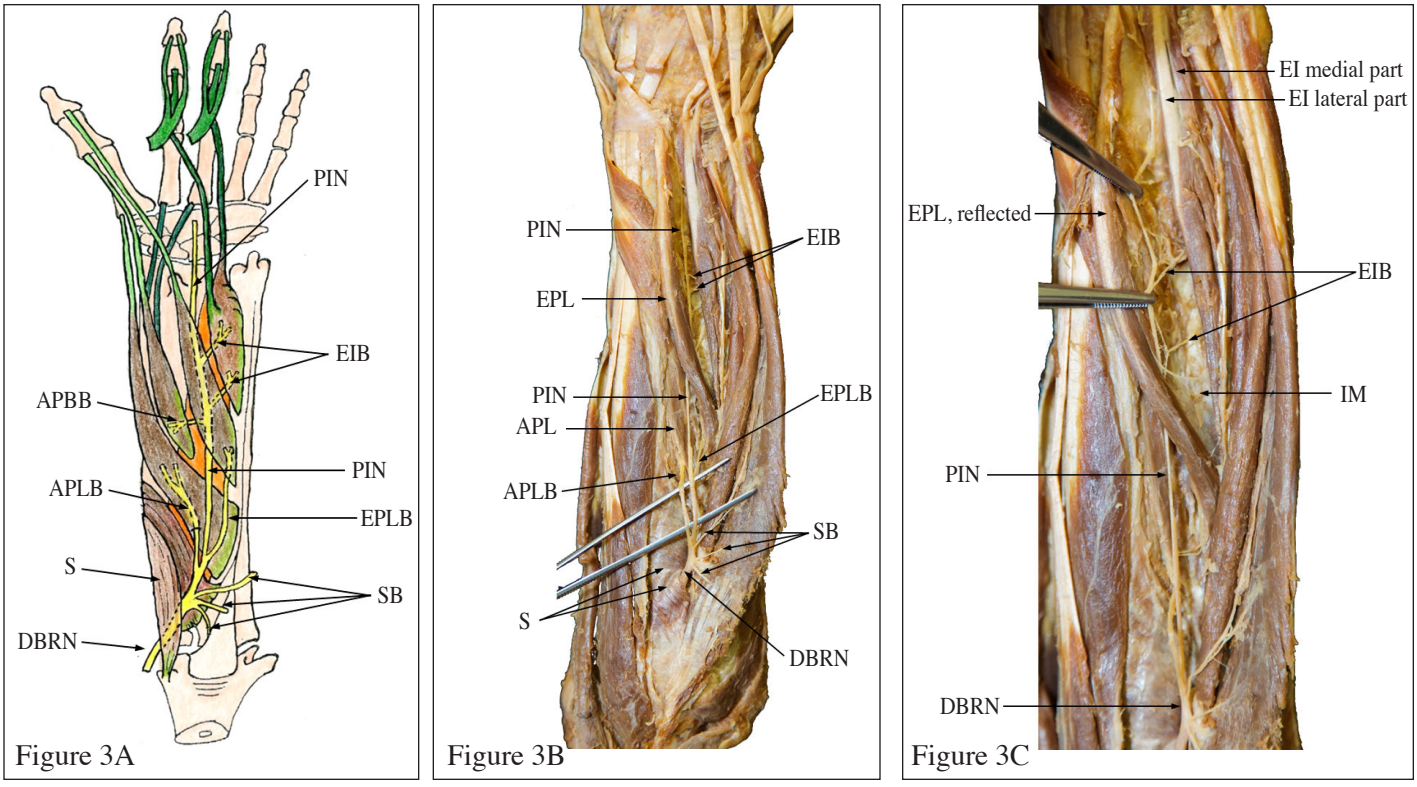

Figure 3

The branching pattern of the deep branch of the radial nerve (DBRN) in the forearm with the variant EI muscle.
A – Schematic representation illustrates the DBRN which traverses the supinator (S) muscle, and immediately provides the short branches (SB). The long branches include the abductor pollicis longus branch (APLB), the extensor pollicis longus branch (EPLB), and the posterior interosseous nerve (PIN). The PIN provides the abductor pollicis brevis branch (APBB), and the extensor indicis branches (EIB).
B – The DBRN descends superficial to the APL muscle providing the APLB and EPLB, and then as the PIN, descends deep to the EPL muscle.
C – The PIN descends deep to the EPL muscle on the posterior surface of the IM. Being located between the EPL and the lateral part of the EI muscle, the PIN provides the two to three EIB.

an overstretched EI muscle might lead to inflammation within this fascia and an increase in intracompartmental pressure. Moreover, the surgeon should take this attachment into consideration when performing any surgical procedure within the extensor forearm region.

Caudwell et al.[42] described the musculotendinous junction of the EI within the confines of the fourth dorsal osseofibrous tunnel in 75% of the specimens which they examined. Ritter and Inglis,[40] confirming the frequency of the musculotendinosus portion of EI within the fourth dorsal tunnel, remarked that the contents of the tunnel are extremely tightly confined when the hand and fingers are flexed. That is why an increase in the size of any of the components of the tunnel, including a hypertrophied EI, might produce pain and disability. Similarly, we believe that the presence of two tendons of the EI, especially the musculotendinous junction of its medial part, would increase the volume of the contents of the fourth dorsal osseofibrous tunnel and might cause pain and other clinical symptoms. We observed that in the tunnel itself,

the two tendons of the EI muscle are positioned medial and deep to the tendons of the ED muscle. Knowledge of this relationship is essential in tendon identification when harvesting for tendon transfer during such procedures as opponensplasty.[43]

Review of the literature indicates that the EI muscle confers independence to the index finger. Acting alone or together with the ED, it extends the index finger at the metacarpophalangeal and proximal interphalangeal joints and assists with the extension of the hand at the wrist. As a result, the EI muscle enhances tight grip.[43, 44] For this reason, we assume that the double tendon of the EI muscle, joining the EE, may increase the independence not only of the index finger but also of the middle finger, and may further enhance tight grip.

The EE is formed by the four tendinous extensions of the ED muscle on the dorsum of the proximal phalanx of each digit. Each extension is made by three slips, one axial and two collateral slips. The lateral collateral slips are thickened by the tendons of lumbrical and interosseous muscles and the medial collateral slips by tendons of the interosseous alone.[1,31,40,43,45]

In the presented case, the two tendons of the EI muscle joined the medial collateral slips of the EE to the middle and index fingers, as reported previously in the literature.[40,43,45] We hypothesize that by equalizing the thickness of the medial slips of the EE to the index and middle digits, the EI double tendon assists with the balancing and dissipation of mechanical stresses during the coordinated extension of these digits.

Understanding the anatomical branching pattern of the radial nerve within the posterior forearm is an essential step in recognizing the clinical symptoms of peripheral neuropathy.[22,43,46] It is also important in identifying the level of nerve injury,[47] for use during surgical repair using nerve grafts,[48-51] performing nerve blocks,[52] neurography,[30] and for the modeling of neuromuscular compartments.[25] Since total or partial lesions are often encountered in clinical settings, we sought to determine the branching pattern of the radial nerve in a case of an EI muscle with a double tendon.

According to our observation, the DBRN after emerging from the supinator gives off short branches to the superficial extensor muscles. This observation is in agreement with the majority of investigations, except that the nerve penetrating the supinator is often referred to as the PIN.[22,23,25,31,38,48,49,52-55] Following the international anatomical terminology,[32] we classify the PIN as the terminal long branch of the DBRN.

In our study, the innervation of the deep extensor muscles is supplied by the long branches of the DBRN, including the nerve to the APL, nerve to the EPB, and the PIN. The PIN, descending deep to the EPL, provides two to three short branches to supply the EPB and EI muscles. In the literature, all long branches are described as branches of the PIN with morphometric and schematic variances.[23,38,49]

Thus, we conclude that there are no differences in the branching pattern of the DBRN within forearms possessing either single or double tendons of EI. In both cases, having longer branches to APL and EPL allow for the possibility of isolated neuropathy of these branches. The innervation of the EI and EPB muscles by the short branches from the PIN most likely would be disrupted by the neuropathy of either the main trunk of DBRN or PIN as it descends deep to the EPL muscle.

Conclusions

1. A double tendon of the EI increases the volume of the contents of the fourth dorsal osseofibrous tunnel, which may result in clinical symptoms.
2. By equalizing the thickness of the medial slips of the extensor expansion, the two tendons of the EI may assist with the balancing of the mechanical stresses within the extensor expansion, contributing to the coordination of the extension of the second and third digits.
3. The branching pattern of the DBRN may result in a predisposition towards isolated neuropathy of the long branches or the posterior interosseous nerve, resulting in specific clinical symptoms.

References

1. Jones BV, Ipswich RN. An anomalous extensor indicis muscle. J Bone Joint Surg Br. 1959;41-B(4):763-765.
2. Schenck RR. Variations of the extensor tendons of the fingers. Surgical significance. J Bone Joint Surg Am. 1964;46:103-110.
3. Bergman RA, Thompson SA, Afifi AK. Catalog of Human Variation. Baltimore-Munich: Urban & Schwazzenberg, 1984:40-155.
4. Von Schroeder HP, Botte MJ. The extensor medii proprius and anomalous extensor tendons to the long finger. J Hand Surg Am. 1991;16(6):1141-1145.

5. GodwinY, Ellis H. Distribution of the extensor tendons on the dorsum of the hand. Clin Anat. 1992;5:394-403.
6. el-Badawi M, Butt M, al-Zuhair A, et al. Extensor tendons of the fingers: arrangement and variations. Clin Anat. 1995;8(6):391-398.
7. Von Schroeder HP, Botte MJ. Anatomy of the extensor tendons of the fingers: variations and multiplicity. J Hand Surg Am. 1995;20:27-34.
8. Yoshida Y. Anatomical studies on the extensor pollicis et indicis accessorius muscles and the extensor indicis radialis muscle in Japanese. Okajimas Folia Jpn. 1995;71(6):355-363.
9. Tan ST, Smith PJ. Anomalous extensor muscles of the hand: a review. J Hand Surg. 1999;24-A(3):449-455.
10. Shiraishi N, Matsumura G. Anatomical variations of the extensor pollicis brevis tendon and adductor pollicis longus tendon-relation to tenosynovectomy. Okajimas Folia Anat Jpn. 2005;82:25-29.
11. Ranade AV, Rai R, Prabhu LV, et al. Incidence of extensor digitorum brevis manus muscle. Hand (NY). 2008;3(4):320-323.
12. Zilber S, Oberlin C. Anatomical variations of the extensor tendons to the fingers over the dorsum of the hand: a study of 50 hands and a review of the literature. Plast Reconstr Surg. 2004;113(1):214-221.
13. Li J, Ren ZF. Bilateral extensor indicis brevis: a rare muscular variant. Case report. Rom J Morphol Embryol. 2012;53(1):185-187.
14. Vazquez JM, Linscheid RL. Anomalous extensor muscles simulating dorsal wrist ganglion. Clin Orthop Relat Res. 1972;86:84-86.
15. Reeder CA, Pandeya NK. Extensor indicis proprius syndrome secondary to an anomalous extensor indicis proprius muscle belly. J Am Osteopath Assoc. 1991;91(3):251-253.
16. Baker J, Gonzalez MH. Snapping wrist due to an anomalous extensor indicis proprius: a case report. Hand (NY). 2008;3(4):363-365.
17. Bolla SR, Vollala VR, Bovindala B, et al. Extensor digitorum brevis manus: Its clinical significance and morphology. International J Anatomical Variations. 2008;1:32-34.
18. Hanz KR, Saint-Cyr M, Semmler MJ, et al. Extensor tendon injuries: acute management and secondary reconstruction. Plast Reconstr Surg. 2008;121(3):109e-120e.
19. Celik S, Bilge O, Pinar Y, et al. The anatomical variations of the extensor tendons to the dorsum of the hand. Clin Anat. 2008;21(7):652-659.
20. Otenasek FG. Progressive paralysis of the nervous interosseus dorsalis. Pathological findings in one case. Bull Johns Hopkins Hosp. 1947;81:163-167.
21. Spinner M. Injuries to the Major Branches of peripheral Nerves of the Forearm. 2nd ed. Philadelphia: W.B. Saunders, 1978:80-157.
22. Hirayama T, Takemitsu Y. Isolated paralysis of the descending branch of the posterior interosseous nerve. Report of a case. J Bone Joint Surg Am. 1988;70(9):1402-1403.
23. Ay S, Apaydin N, Acar H, et al. Anatomic pattern of the terminal branches of posterior interosseous nerve. Clin Anat. 2005;18(4):290-295.
24. Alport AR, Sander HW. Clinical approach to peripheral neuropathy: anatomical localization and diagnostic testing. Continuum (Minneap Minn). 2012;18(1):13-38.
25. Ravichandiran M, Ravichandiran N, Ravichandiran K, et al. Neuromuscular partitioning in the extensor carpi radialis longus and brevis based on intramuscular nerve distribution patterns: A three-dimensional modeling study. Clin Anat. 2012;25(3):366-372.
26. Scott JR, Gobby M, Taggart I. Magnetic resonance imaging of acute tendon injury in the finger. J Hand Surg Br. 1995;20:286-288.
27. Hauger O, Chung CB, Lektrakul N, et al. Pulley system in the fingers: normal anatomy and simulated lesions in cadavers at MR imaging, CT, and US with and without contrast material distention of the tendon sheath. Radiology. 2000;217:201-212.
28. Swen WA, Jacobs JW, Huback PC, et al. Comparison of sonography and magnetic resonance imaging for the diagnosis of partial tears of finger extensor tendons in rheumatoid arthritis. Rheumatology (Oxford). 2000;39(1):55-62.
29. Clavero JA, GolanoP, Farinas O, et al. Extensor mechanism of the fingers: MR Imaging-anatomic correlation. Radio Graphics. 2003;23:593-611.
30. Cudlip SA, Howe FA, Phil D, et al. Magnetic resonance neurography studies of the median nerve before and after carpal tunnel decompression. J Neurosurg. 2002;96:1046-1051.
31. Moore KL, Dalley FD, Agur AMR. Clinically oriented anatomy. 7th ed. Philadelphia: Wolters Kluwer/Lippincott Williams &Wilkins, 2014:750-757; 761-764.
32. Terminologia Anatomica: International anatomical terminology. Federative Committee of Anatomical Terminology (FCAT). Stuttgart: Thieme, 1998:1-292.
33. Soni P, Stern CA, Foreman KB, et al. Advances in extensor tendon diagnosis and therapy. Plast Reconstr Surg. 2009;123(2):52e-57e.
34. Wu TS, Rosenberg M, VanDillen C, et al. Bedside ultrasound evaluation of tendon injuries. Ann Emerg Med. 2009:54(3):S67-S68.
35. Martinoli C. Musculoskeletal ultrasound: technical guidelines. Insights Imaging. 2010;1:99-144.
36. Li J, Ren ZF. Bilateral extensor indicis brevis: a rare muscular variant. Rom J Morphol Embryol. 2012;53(1):185-187.
37. Egloff DV, Verdan C. Pollicization of the index finger for reconstruction of the congenitally hypoplastic or absent thumb. J Hand Surg Am. 1983;8(6):839-848.

38. Elgafy H, Ebraheim NA, Rezcallah AT, et al. Posterior interosseous nerve terminal branches. Clin Orthop Relat Res. 2000;376:242-251.
39. Schmidt HM, Lanz U. Surgical Anatomy of the Hand. 1st ed. New York, NY: Thieme; 2004:1-267.
40. Ritter MA, Inglis AE. The Extensor Indicis Proprius Syndrome. J Bone Joint Surg Am. 1969;51-A(8):1645-1648.
41. Doyle JR. Extensor tendons-acute injuries. In: Green DP, ed. Operative hand surgery. 3rd ed. New York, NY: Churchill Livingstone, 1993:1925-1954.
42. Caudwell EW, Anson BJ, Wright RR. The extensor indicis proprius muscle: a study of 263 consecutive specimens. Q Bull Northwest Univ Med Sch. 1943;17:267-279.
43. Doyle J, Botte MJ. Surgical anatomy of the hand and upper extremity. Philadelphia: Lippincott Williams&Wilkins, 2003:134-135,141-142,221.
44. Williams PL, ed. Gray's Anatomy. The Anatomical Basis of Medicine and Surgery. 38th ed. Edinburgh: Churchill Livingstone, 1995:852-858.
45. Gonzalez MH, Weinzweig N, Kay T, et al. Anatomy of the extensor tendons to the index finger. J Hand Surg Am. 1996;21(6):988-991.
46. Alport AR, Sander HW. Clinical approach to peripheral neuropathy: anatomical localization and diagnostic testing. Continuum (Minneap Minn). 2012;18(1):13-38.
47. LI Hai, CAI Qi-xun, SHEN Pin-quan, et al. Posterior interosseous nerve entrapment after Monteggia fracrture-dislocation in children. Chinese J Traumatology. 2013;16(3):131-135.
48. Waters PM, Schwartz JT. Posterior interosseus nerve: an anatomic study of potential nerve grafts. J Hand Surg Am. 1993;18(4):743-745.
49. Missankov AA, Sehgal AK, Mennen U. Variations of the posterior interosseous nerve. J Hand Surg Br. 2000;25(3):281-282.
50. Leechavengvongs S, Witoonchart K, Uerpairojkit C. Penetrating injury to the terminal branches of the posterior interosseous nerve with nerve grafting. J Hand Surg Br. 2001;26(6):593-595.
51. Lawton JN, Cameron-Donaldson M, Blazar PE, et al. The anatomic considerations regarding the posterior interosseous nerve at the elbow. J Shoulder Elbow Surg. 2007;16(4):502-507.
52. Abrams RA, Ziets RJ, Lieber RL, et al. Anatomy of the radial nerve motor branches in the forearm. J Hand Surg. 1997;22(2):232-237.
53. Dellon AL, Seif SS. Anatomic dissections relating the posterior interosseous nerve to the carpus, and the etiology of dorsal wrist ganglion pain. J Hand Surg Am. 1978;3(4);326-332.
54. Carr D, Davis P. Distal posterior interossseous nerve syndrome. J Hand Surg. 1985;10(6):873-878.
55. Branovacki G, Hanson M, Crash R, et al. The innervation pattern of the radial nerve at the elbow and in the forearm. J Hand Surg. 1998;23(B):167-169.

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Ischemic compression and joint mobilisation for the treatment of nonspecific myofascial foot pain: findings from two quasi-experimental before-and-after studies

Guy Hains, DC[a]
Pierre B. Boucher, DC, PhD[b]
Anne-Marie Lamy, DC[a]

Objective: *The aim of this study was to evaluate the efficacy of myofascial therapy involving ischemic compression on trigger points in combination with mobilization therapy on patients with chronic nonspecific foot pain.*

Study design: *Two quasi-experimental before-and-after studies involving two different baseline states.*

Method*: Foot pain patients at a private clinic were divided into two separate cohorts: A, custom orthotic users; and B, non-users. In Study A, 31 users received 15 experimental treatments consisting of ischemic compressions on trigger points and mobilization of articulations through the foot immediately after study enrollment. In study B, ten non-users were prescribed a soft prefabricated insole and were monitored for five weeks before subsequently receiving 15 experimental treatments after the initial five-week delay.*

Outcome measures: *The Foot Function Index (FFI) and patients' perceived improvement score (PIS) on a scale from 0% to 100%.*

Results: *The Study A group (n=31) maintained a significant reduction in the FFI at all three follow-*

Objectif : *L'objectif de la présente étude est d'évaluer l'efficacité de la thérapie myofasciale impliquant une compression ischémique sur des points gâchettes combinée à une thérapie de mobilisation chez les patients souffrant de douleurs chroniques non spécifiques au pied.*

Plan d'étude : *Deux études quasi expérimentales avant/après sur deux états de référence différents.*

Méthodologie : *On a formé deux groupes avec les patients souffrant de douleurs au pied d'une clinique privée. A : les utilisateurs d'orthèses adaptées. B : ceux qui n'en utilisent pas. Dans l'étude A, 31 utilisateurs ont reçu 15 traitements expérimentaux impliquant une compression ischémique sur des points gâchettes et une mobilisation des articulations du pied immédiatement après l'inscription à l'étude. Dans l'étude B, 10 non-utilisateurs ont reçu une prescription de semelle souple préfabriquée et ont fait l'objet d'un suivi de 5 semaines. Après les 5 semaines de suivi, ils ont reçu 15 traitements expérimentaux.*

Mesures des résultats : *L'index de fonction du pied (IFP) et l'amélioration perçue par le patient (APP) sur une échelle de 0 % à 100 %.*

Résultats : *Le groupe de l'étude A (n=31) a montré une diminution importante de l'IFP aux trois évaluations*

[a] Private Practice
[b] Professor, Département de chiropratique, Université du Québec à Trois-Rivières, Québec, Canada. Pierre.B.Boucher@uqtr.ca

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up evaluations. Mean improvement from baseline in FFI was 47%, 49% and 56% at 0, 1 and 6 months, respectively, post-treatment. Mean PIS was 58%, 57%, and 58%, again at 0, 1 and 6 months post-treatment. For the Study B group, mean improvement in FFI was only 19% after the monitoring period, and 64% after the experimental treatment period. Mean PIS was 31% after monitoring, and 78% after experimental treatment. In repeated measures analyses, experimental treatment was associated with a significant main effect in both of these before-and after studies (all P values<0.01).

Conclusion: *Combined treatment involving ischemic compression and joint mobilization for chronic foot pain is associated with significant improvements in functional and self-perceived improvement immediately and at up to six-months post-treatment. Further validation of this treatment approach within a randomized controlled trial is needed.*

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KEY WORDS: foot pain, myofascial trigger points, ischemic compression, joint mobilization, chiropractic

de suivi. L'amélioration moyenne selon la référence de l'IFP était de 47 %, 49 % et 56 % après 0, 1 et 6 mois respectivement. La moyenne d'APP était de 58 %, 57 % et de nouveau 58 % 0, 1 et 6 mois après le traitement. Le groupe de l'étude B a montré une amélioration moyenne de l'IFP de seulement 19 % après la période de suivi, et de 64 % après la période de traitements expérimentaux. La moyenne d'APP était de 31 % après le suivi et de 78 % après les traitements expérimentaux. Dans les analyses de mesures répétées, on associe le traitement expérimental à un effet principal important dans les deux études avant/après (toutes les valeurs P<0,01).

Conclusion : *Un traitement combiné impliquant une compression ischémique sur des points gâchettes et une mobilisation des articulations pour soulager des douleurs chroniques au pied est associé à des améliorations fonctionnelles importantes et une amélioration cernée par le patient sur une période allant d'immédiatement après le traitement jusqu'à six mois plus tard. Il est nécessaire d'effectuer un essai contrôlé aléatoire pour valider ce traitement.*

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MOTS CLÉS : douleur au pied, myofascial, points gâchettes, compression ischémique, mobilisation des articulations, chiropratique

Introduction

Foot pain is very common. In one population-based study of 4,060 subjects, 17.4% had foot pain, aching or stiffness in either foot.[1] Plantar fasciitis affects about 10% of the population at some time during their lives.[2] Regarding hallux valgus, one systematic review and meta-analysis of 76 pooled surveys (496,957 participants) estimated that the prevalence was 23% in adults aged 16-65 years and 35.7% in elderly people aged 65 years or older. It was 30% in females compared to 13% in males.[3] Foot problems are commonly encountered in chiropractic practice.[4,5] It is often assumed that biomechanics is the cause of almost any condition seen in the foot;[6] this is seemingly proved by the biomechanical abnormalities present in the pathological foot.[6] But while biomechanical abnormalities are common, it should not be assumed that they are necessarily causatively associated with foot symptoms. In this regard, it is important to rule out other medical or specific causes of foot pain before assuming that a biomechanical fault is at play.[6]

Specific foot pain conditions

For background purposes, we summarize a number of common specific conditions that should be ruled out prior to applying a working diagnosis of nonspecific biomechanical foot pain.

Plantar fasciitis

The plantar fascia is a thick band of longitudinally arranged fibers which run from the tuberosity of the calcaneus. The thick, central portion divides distally into five slips (one for each digit), and inserts into the sides of the

sheath of the flexor tendon of each toe.[7] The cause of plantar fasciitis is a repetitive microtrauma overload injury.[8] The point of injury is usually located at the attachment of the plantar fascia on the inferior aspect of the calcaneus. On digital palpation a point of hyperirritability or severe point tenderness is found at the insertion of the plantar fascia into the calcaneus. The most common symptom is "first-step pain," immediately after prolonged non-weight bearing, which quickly diminishes after the next few steps. In proximal plantar fasciitis, over-pronation is commonly associated, as are simple training errors.[8] Less commonly, tenderness over the distal and mid portion of the plantar fascia indicates the presence of distal plantar fasciitis.[8,11] Plantar spurs occur in 20% of normal patients without plantar fasciitis and in about 50% of patients with plantar fasciitis, so their presence is suggestive, but by no means diagnostic.[9,11]

The natural history of plantar fasciitis is unclear.[11] Bone scans are indicated only in problematic cases.[9] Commonly administered treatments include nonsteroidal anti-inflammatory drugs (NSAIDs), heel pads or cups, orthotics, steroid injections, ultrasound, deep friction massage, shock wave therapy, and active release.[2,4,7,12] Surgery is thought to be indicated after nine months of failed conservative treatment.[5,8,9,10] Although there is consensus that conservative treatments are effective most of the time, there is no agreement as to which specific modality is most effective.[11]

Hallux valgus, hallux rigidus, turf toe, and bunion

There are many interrelated painful conditions of the great toe. Some are considered to be complications of acute or chronic sprain/strain of the first metatarsophalangeal joint. This category accounts for the most common athletic disorders of the foot.[13] Each of these diagnoses is often based simply on the elicitation of exquisite tenderness during passive extension of the great toe, with or without simultaneous observation of a bunion.[14] The exact cause of hallux valgus is unknown.[15] The literature suggests that advanced imaging studies and lab studies are uninformative for hallux problems in general.[15] Commonly administered treatments for hallux problems include manipulation, transverse friction therapy, gradual axial elongation and progressive mobilization of the first metatarsophalangeal joint.[13,14,16,17] There is some evidence that mobilization of the first metatarsophalangeal joint is of value in the treatment of hallux rigidus.[13] Yet, in one Cochrane systematic review, there was no evidence of a difference in effectiveness between various conservative treatments and no treatment for hallux problems. Furthermore, that same review found no evidence from randomized controlled trials of any benefit (e.g., diminution of joint deformity or pain) from the use of orthotics or splints.[14] For hallux valgus in particular, treatment has often been aimed at reducing deformity, and improving pain, function, and patient satisfaction.[15,16] However, the genuine effectiveness of non-surgical interventions for hallux valgus remain questionable.

Metatarsalgia

The main symptom is a burning sensation located at the ball of the foot. When speaking of metatarsalgia, it is customary to exclude conditions affecting the first metatarsophalangeal joint, as those conditions tend to fall under the umbrella of hallux valgus, hallux rigidus and other related conditions of the big toe.[18]

Morton's neuroma

Morton's neuroma is a misnomer as the underlying pathology does not involve a proliferative state of the nerve tissue.[19] For this reason, the term "Morton's metatarsalgia" is preferred by some authors. In any event, this condition is a paroxysmal neuralgia affecting the web spaces in the toes, typically the third. The pain may be sharp and lancinating when walking. The diagnosis is typically based on a history of symptoms such as pain, numbness or pins and needles in the ball of the foot and/or toes, particularly during walking. On examination digital pressure applied to the third and/or fourth intermetatarsal area provokes pain. Neuromas are perhaps the most misdiagnosed of all podiatric complaints and, in reality, are not nearly as prevalent as once believed. Instead, arthritis and other conditions causing inflammation of the metatarsophalangeal joints, and pressure on the interdigital nerves can mimic a true neuroma.[6]

Many patients undergo surgery for this condition, yet there is little evidence on the effectiveness of surgical as well as non-surgical interventions for Morton's neuroma.[19] As surgical removal of a misdiagnosed neuroma may cause an exacerbation of the symptoms and even disability,[6] it is imperative to exhaust non-invasive approaches before contemplating surgery.

Longitudinal arch pain

Often, pain along the longitudinal arch occurs at the cuboid-metatarsal articulation. There are three mechanisms of injury associated with this condition: 1) acute injury from forceful plantar flexion and inversion of the foot and ankle, 2) compensatory weight bearing over the lateral border of the foot in response to antecedent medial plantar heel pain, and 3) biomechanical predisposition to a lateral forefoot sprain, such as in the presence of pes cavus deformity.[20] In treating this problem, mobilization, manipulation, long axis distraction, and orthotics have been suggested.[20,21]

Tendinopathy of the Achilles tendon

Posterior heel pain most commonly arises from the Achilles tendon. The clinical picture is pain and swelling immediately above the heel, with related impairment of physical function. The Achilles tendon is the conjoint tendon of the soleus and the two heads of the gastrocnemius muscles. Patients with insertional tendinopathy present with posterior heel pain, mostly in the middle region and insertion of the tendon. Overuse is the principal cause. Usual treatments include orthosis, stretching, anti-inflammatory medication and steroid injection, the latter of which should be performed only sparingly due to the potential risk of tendon rupture. Surgery is considered only when all nonoperative treatments have been tried.[22]

Nonspecific foot pain

In the absence of the above-mentioned specific causes of chronic foot pain, we suggest that a diagnosis of nonspecific foot pain is applicable. For nonspecific foot pain, we have utilized a treatment approach that appears to be promising from an anecdotal perspective, but has not yet been formally validated to date.

The proposed treatment assumes that nonspecific foot pain has, at least in part, a myofascial component which may be present either exclusively or in addition to the aforementioned specific causes of foot pain. Our use of ischemic compression and joint mobilization is therefore based on the theoretical rationale that myofascial trigger points (TrPs) may be located within muscles, ligaments, tendons, fascia, and articular capsules of the painful foot.[23,24] Previous studies (further detailed in the Discussion section) have shown that other manual myofascial therapy techniques such as friction massage, mobilization, and Graston Instrument Mobilization Technique have helped alleviate many foot problems.[9,13,14,16,21,25]

Study rationale

The objective of this study was to determine the efficacy of ischemic compression and joint mobilisation for the treatment of common nonspecific foot pain of presumed myofascial origin.

Our primary hypothesis was that private clinic patients with chronic foot pain who are treated with ischemic compression on trigger points, as pinpointed through palpation and patient corroboration, in combination with joint mobilization, would exhibit significant improvement in the severity of symptoms and functional status after 15 experimental treatments.

Methods

We conducted a controlled before-and-after study in which data was collected in two populations contemporaneously within a private clinic setting in Trois-Rivières, Québec. However, as both populations were exposed to an experimental treatment phase, each constituted a separate quasi-experimental study individually as well as a controlled before-and-after study collectively.

Each individual study involved a single group repeated measures design within a distinct cohort of foot pain patients. Study A was a before-and-after study of the effect of manual therapy in a cohort of custom orthotic users. Study B was a before-and-after study of the effect of delayed manual therapy in a cohort of non-users who were prescribed a soft prefabricated orthotic for five weeks before undergoing the experimental manual therapy approach over an additional five weeks. Therefore, during the first five weeks of data collection only the Study A cohort received experimental treatment while the Study B cohort was monitored contemporaneously without receiving experimental treatment.

All participants received experimental manual therapy from one of two experienced chiropractors (GH, AML). All data collection was performed by the principal investigator (GH) who was clearly aware of each participant's degree of progress throughout the study. A study statistician, on the other hand, was blinded to the types of treatments being administered and analyzed. The study was approved by the ethics committee of the Université du Québec à Trois-Rivières.

All subjects were recruited through local newspaper advertisements. In order to take part in one of the studies, participants had to be between 20 and 60 years of age and had to have suffered from daily foot pain for at least three months. Foot pain was defined as pain anywhere in the foot, either at rest or during movement. The intensity of the pain had to be rated at least a "6" on a 10 cm visual analogue scale. Participants agreed to receive, free of charge, 15 manual therapy treatments over five weeks (one month), at a frequency of three times per week.

The exclusion criteria for both studies included past surgery to the symptomatic foot, body weight greater than 200 pounds, a history of steroid or local anaesthetic injection to the foot within the past month, a current history of local tumour, infection, fracture, rheumatoid arthritis or any other active arthropathy.

As podiatry is taught at the Université du Québec à Trois-Rivières, most of the participants (readers of the local newspaper) were residents of the local community who had already seen a Doctor of Podiatric Medicine and were also already using custom orthotics. Arbitrarily, we recruited all participants who were already wearing a custom orthotic into Study A while participants not wearing a custom orthotic were recruited for Study B.

Written informed consent was obtained from each participant.

Experimental interventions

Ischemic compression treatment over trigger and/or tender points.

The diagnosis of a treatable TrP in this study involved the patient pinpointing the exact location of pain on the foot, and the attending chiropractor reproducing the patient's pain upon applying thumb-over-thumb pressure over the location in question. If the patient's pain was reproduced, ischemic compression treatment was administered during each visit, and consisted of a single application of 15-seconds of pressure (again using a thumb-over-thumb contact) over each treatable TrP identified in the foot. As the TrP could be very sensitive, the pressure was light during the first few seconds, and gradually increased to the point of the patient's maximum tolerance.

Patients were positioned in a standardized manner depending upon the area of the foot being treated:

1. Plantar fascia: The patient was in either a supine position with the legs extended or in a prone position with the knee flexed to 90 degrees.

2. Dorsal aspect of the foot: To target TrPs over the dorsal metatarsal or tarsal regions, ischemic compression was applied with the patient supine and the foot flat on the table. The pressure was applied perpendicularly to the dorsal surface of the foot. The therapist sometimes used his body weight to augment the application of pressure through the thumbs.

3. Lateral longitudinal arch: The patient would lie in a recumbent position with the affected side (foot) up, and the medial aspect of the affected foot resting on the table. In this position, the plantar surface of the cuboid bone was the most common site treated.

4. Achilles tendon: With the patient in a prone position, the tendon was palpated along its entire length. Pressure was applied perpendicularly to the skin surface over tender points anywhere along the length of the tendon. TrPs in the soleus and the gastrocnemius muscles were also treated.

5. Center of the fat pad of the heel: The patient was treated either in a prone or standing position with the dorsal surface of the forefoot facing down and resting flat on the table, and with the plantar surface of the heel exposed and facing up. Often, treatable TrPs were detected and therefore targeted over the entire plantar surface of the heel.

6. Anterior capsule of the foot: While the patient was supine, the therapist stabilized the foot by applying pressure to the ball of the foot (and therefore forceful ankle dorsiflexion) with his thigh. At the same time the ankle-mortise joint was probed deeply with the thumbs in order to test for the presence of treatable TrPs and/or movement restriction in the ankle. Particularly in this region of the foot, ischemic compression and joint mobilisation were often administered simultaneously.

Mobilization

Joint mobilisation consisted of applying forceful flexion or extension movement, within the patient's level of tolerance, through the hypomobile articulation for 15 seconds per administration. The following standardized approaches were used:

1. First metatarsophalangeal articulation (big toe): Depending on the direction of detected restriction, pressure for 15-seconds was applied in either extension, flexion, or both directions. Usually, treatment was given in the direction that was limited by pain. In symptomatic pa-

tients, pressure was applied very lightly during the first few seconds, then gradually increased to the patient's maximum tolerance. Thereafter, pressure was sustained without moving the joint any further. The same treatment was used for treating an underlying hallux rigidus. The diagnosis of a concomitant hallux rigidus was based on the presence of exquisite tenderness upon passive extension of the big toe, with or without bunion formation.[16]

2. Third and fourth metatarsophalangeal articulation (sometimes in the presence of concomitant Morton's neuralgia): Mobilisation was carried out with the patient in a supine position. Hyperirritability was tested for by hyperextending and hyperflexing the third and fourth metatarsophalangeal joints. If pain was reproduced, then the affected joint was treated by administering gradual, yet forceful, hyperextension or hyperflexion to tolerance, for 15 seconds per visit. Ischemic compression therapy was often concomitantly administered to treatable TrPs over the lateral aspects of metatarsophalangeal joints.

3. Generalized metatarsophalangeal stiffness: Simultaneous treatment to multiple metatarsophalangeal articulations was carried out with the patient in a supine position. The attending chiropractor would grasp the last four toes of the patient's foot by applying a thenar and hypothenar contact against the plantar surfaces of the toes with one hand, and by reinforcing his grasp with the other hand. All four toes were then simultaneously hyper-extended to the limit of the patient's tolerance and held in that position for 15 seconds. In most case this treatment was performed in combination with ischemic compression therapy over treatable TrPs in the ball of the foot.

Most patients suffered from the presence of concomitant specific foot conditions. In the presence of heel pain, plantar fasciitis, arch pain, and fat pad pain, TrPs located in the calf muscles were also treated. In the foot itself, there were typically one or two TrPs for each condition.

Patients were encouraged to taper off any existing analgesic medications as soon as possible. During treatments, patients were monitored for even the slightest signs of distress and were repetitively asked if the administered pressure or mobilisations were bearable.

Treatments were repeated until either hyperirritability was completely gone or a maximum of 15 visits had been administered. The presence of hyperirritability (pain reproduction) was the basis for the diagnosis of treatable TrPs or joint hypomobility. If the problem was a TrP, the tender point pinpointed by the patient was easily confirmed through palpation over the area in question. If the location was at an articulation, hyperirritability, if any, was detected by forcing the articulation to the end of flexion and/or extension.

Additional Interventions (Study B)

In study B, patients were initially treated for five weeks with only a soft prefabricated insole (Holiday, Pedag International, Germany). After five weeks each participant was invited to receive, free of charge, a course of experimental therapy as described above.

Outcome measures

Foot Function Index (FFI) questionnaire

The FFI was used to measure the impact of pathology on function in terms of pain, disability, and activity restriction. It is a self-administered index consisting of 23 items making up three sub-scales. The test-retest reliability of total and sub-scale scores ranges from 0.87 to 0.69. Internal consistency ranges from 0.96 to 0.73.[26] The FFI is an easily administered clinical index which provides a practical method of measuring foot function in an outpatient setting.[26]

Perceived Improvement Score (PIS).

The PIS is ascertained by asking a patient to rate his/her perceived degree of improvement on a numerical scale from 0 to 100%, where 0 means no improvement and 100% means complete improvement

Timing of measurements

In the Study A, FFI measurements were ascertained at baseline. Subsequently, follow-up FFI and PIS measurements were ascertained at one month post-enrolment (immediately after 15 treatments), two months post-enrolment (one month post-treatment) and seven months post-enrolment (6 months post-treatment).

In Study B, measurements were again obtained at enrolment (baseline). Subsequently, both FFI and PIS were ascertained at one month post-enrolment (after one month of soft orthotic use), and then at two months post-enrolment (immediately after completion of 15 manual therapy treatments in combination with ongoing soft orthotic use).

Statistical Analysis

The percent change in FFI was calculated by subtracting the baseline FFI score from the follow-up measurements, and then dividing by the baseline score. Percent change in PIS was measured directly from the raw questionnaire score and therefore required no statistical transformation.

To test the effects of experimental treatment over time, a one-way repeated-measures ANOVA was performed for each study. Multiple paired t-tests were performed to compare each follow-up measurement to the baseline measurement.

Also, to exploit the controlled before-and-after component of the data, the immediate post-treatment percent changes in the FFI and the PIS from the Study A group were compared to the corresponding one-month post-monitoring period changes in the Study B group using a t-test for independent samples. In this latter analysis Study B participants who were issued a prefabricated orthotic and only monitored during the first month were used as an external control group for Study A participants who were both active custom orthotic users and had received experimental manual therapy during the first month. For all analyses, statistical significance was set at a conventional level of $P < 0.05$.

Results

Figure 1 depicts the flow of patients and timing of measurements for Studies A and B. A total of forty-one patients met initial inclusion criteria and agreed to participate. Thirty-one participants were active users of custom orthotics and were therefore enrolled into Study A. Ten participants were not active custom orthotic users and were therefore enrolled into Study B. There were no statistically significant differences between the two groups in terms of baseline characteristics except for sex, in which case, women accounted for a much higher proportion of participants in Study A (Table 1). The most common specific diagnoses and/or locations of pain among participants involved the heel or Achilles tendon, the plantar fascia, and longitudinal arch (Table 2). In this trial, only three patients in the Study A group were lost to follow-up at six months.

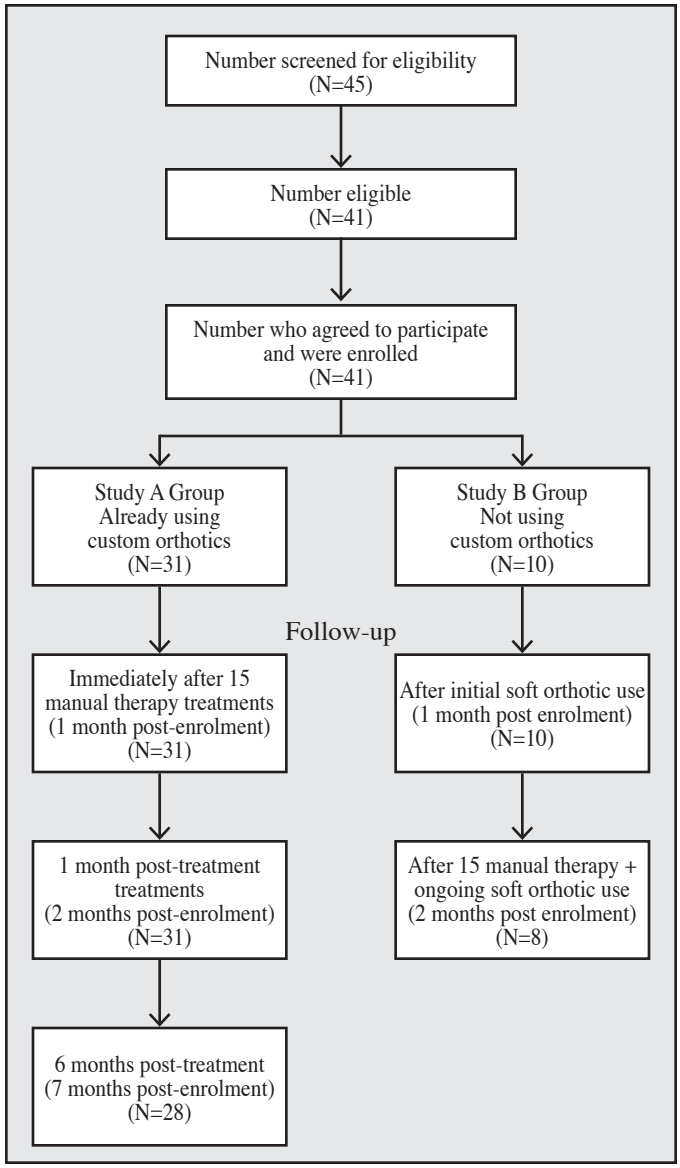

Figure 1:
Patient flow chart.

Foot Function Index (FFI)

In the Study A group, the mean FFI score (and standard deviation [SD]) was 89.5 (SD, 26.2) at baseline (see Table 1); 51.4 (SD, 33.7) after 15 treatments; 47.5 (SD, 32.5) at one month post-treatment, and 37.4 (SD, 28.2) at six months post-treatment. Table 3 shows the corresponding improvement in percent from baseline at each follow-up point. The Study A group maintained a significant reduction in the mean FFI score at all three follow-up evaluations (one-way ANOVA: $F(2, 18) = 30.664$, $p < 0.01$).

Table 1:
Baseline characteristics of participants.

	Study A Group	Study B Group
n	31	10
Women	22	4
Mean age	45	43
Mean duration of symptoms (years)	3.7	4
Baseline FFI questionnaire	89	94

Table 2:
Concomitant specific diagnoses and locations of otherwise nonspecific foot pain among participants.

Diagnosis or location	n (%)
Heel pain (Achilles tendon)	19 (46)
Plantar fasciitis	13 (31)
Painful arches (interior or exterior)	11 (27)
Metatarsalgia	10 (24)
Big toe pain	7 (17)
Pain in other toes	6 (15)
Ankle pain	3 (7)
Anterior tarsus pain	2 (5)
Painful fat pad	1 (2)
Morton's neuroma	1 (2)

*Some patients had multiple diagnoses, therefore, total of percentages exceeds 100.

For the Study B group, the repeated-measures ANOVA showed that a statistically significant improvement occurred only after the period of experimental manual therapy. The mean FFI score was 94.3 (SD, 40.7) at baseline (see Table 1); 77,7 (SD, 32.6) after five weeks (one month) of monitoring and soft orthotic use; and 30.7 (SD, 18.2) after 15 experimental manual therapy treatments (one-way ANOVA $F(1, 9) = 11.412$, $p < 0.01$) at 1 month post-treatment. Again, the corresponding percent improvement from baseline is shown in Table 3 for each follow-up time point.

Figure 2 depicts the mean changes in FFI in both groups over time. Mean FFI and corresponding SDs are presented in Table 3. Admittedly, the two study groups (i.e., active users and nonusers of custom orthotics) are likely systematically different in terms of unmeasured confounders. However, for exploratory purposes only, we tested for, and found, a significant difference ($t(39) = 2.678$, $p = 0.011$) between the two groups at one month post-enrollment. Again, this time point corresponds to the time at which Study A participants completed 15 experimental manual therapy treatments and Study B participants completed one month of monitoring and soft orthotic use without any manual therapy. Overall, the mean percentage of improvement (and standard deviation) was 47% (SD, 30.4) and 19% (SD, 19.5) for the Study A and Study B groups, respectively.

Perceived Improvement Scores (PIS)

The Study A group mean score (and SD) was 58% (SD,

Table 3:
Mean percent improvement in Foot Functional Index (FFI) and Perceived Improvement Scale (PIS) scores.

	Study A		Study B	
	FFI [a]	PIS [b]	FFI [a]	PIS [b]
1 month post-enrolment	47 (30) (after 15 trt's)	58 (26) (after 15 trt's)	19 (19) (after 1 mo. of soft orthotic use)	31 (23) (after 1 mo. of soft orthotic use)
2 months post-enrolment	49 (29) (1 mo. post-trt.)	57 (28) (1 mo. post-trt.)	64 (31) (after 15 trt's)	78 (16) (after 15 trt's)
7 months post-enrolment	56 (33) (6 mo. post-trt.)	58 (33) (6 mo. post-trt.)		

[a] Percent improvement in FFI = ((follow-up FFI – baseline FFI)/baseline FFI) x 100%; with standard deviation of calculated improvement scores in parentheses

[b] Percent PIS as directly reported on the Percent Improvement Scale; standard deviation of PIS scores in parentheses

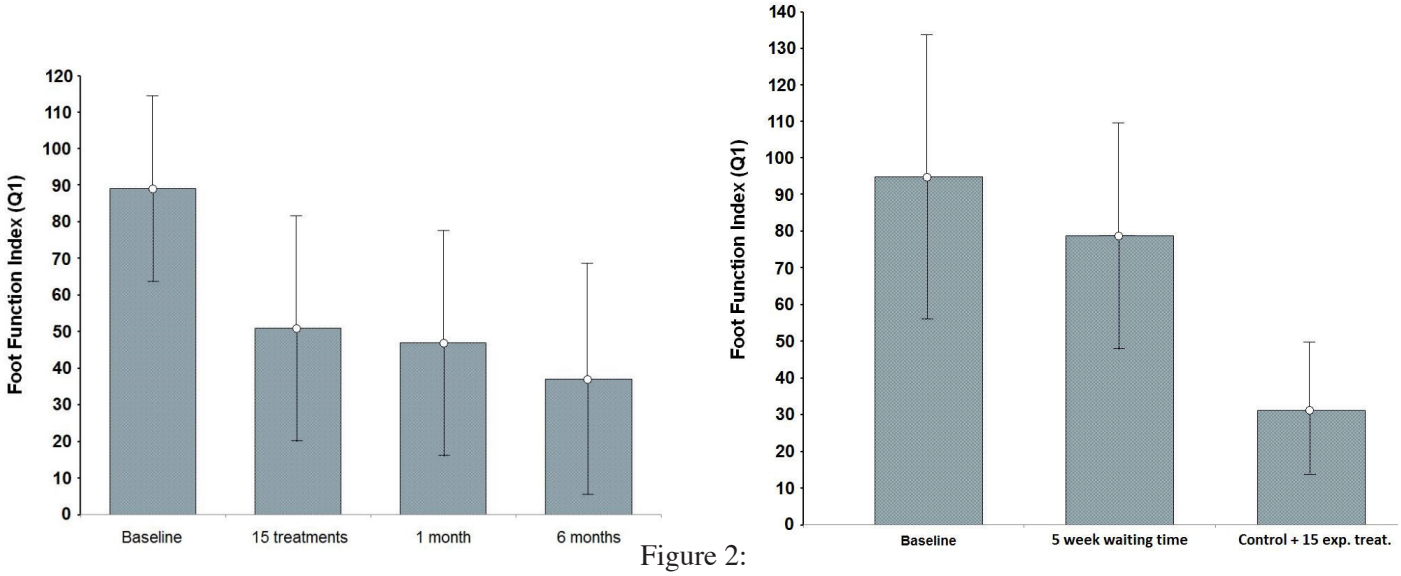

Figure 2:
Mean and standard deviation bars for foot function over time.

26.2) after 15 treatments; 57% (SD, 28.5) one month post-treatment, and 58% (SD, 31) at six months post-treatment (one-way ANOVA: $F(2, 60) = 0.007$, $p > 0.05$). There was no significant difference in the PIS between any of the three follow-up evaluations.

For the Study B group, the mean PIS (and SD) was 31% (SD, 22.9) after one month of prefabricated soft orthotic use without experimental manual therapy, and 78% (SD, 14) after 15 treatments with experimental manual therapy. The repeated-measures ANOVA yielded a significant increase in the PIS only after the experimental manual therapy period ($F(1, 9) = 67.314$, $p < 0.001$).

Between study cohorts, a significant difference was tested for, and detected between the groups A and B at one month post-enrolment, which corresponds to the time that the Study A group completed 15 manual treatments ($t(39) = 2.920$, $p = 0.006$),and the Study B group had completed one month of monitoring and soft orthotic use without any manual therapy. Mean PIS and (and SDs) at each individual time are presented in Table 3. Across all time points overall, the mean PIS was 58% (SD, 26.2) and 31% (SD, 22.9) for the Study A group and Study B groups, respectively.

In these studies, we did not observe any significant adverse effects from treatment. There were few reports (three) of increased sensitivity in the feet among some participants after the first few treatments, however this sensitivity invariably diminished subsequently.

The sum of the above frequency counts exceed 100% as many patients had more than one foot problem.

Discussion

The main findings of the two studies are that significant improvements in outcomes were detected after immediate as well as delayed experimental treatment. In Study A, FFI scores improved significantly from baseline following 15 sessions (i.e., one month) of experimental manual therapy. Similarly, in Study B, FFI scores were seen to be significantly greater only after implementation of delayed experimental manual therapy.

A comparison between the two study cohorts also suggested a benefit in favor of experimental manual therapy. In this regard, mean improvement in foot pain related disability (as assessed by the FFI) was more improved in the Study A group (47%) than the Study B group (19%). A similar benefit in the Study A cohort was observed in terms of self-perceived improvement (58% in the Study A cohort versus 31% in the Study B cohort). On the one hand, these between-cohort comparisons should be interpreted cautiously as they are potentially confounded. Theoretically, for example, orthotic users in Study A may have been better off socioeconomically and there-

fore more able to afford orthotics. Similarly, they might have been more securely employed and therefore better covered by extended health insurance in comparison to nonusers of custom orthotics in Study B. Either circumstance could have been associated with a superior initial outcome in the Study A cohort independent of any effect of immediately-administered experimental therapy.

On the other hand, the results of the between-cohort comparisons are consistent with those of our within-cohort findings. Our findings are clinically important and particularly compelling within Study B, which included a five-week monitoring phase prior to the administration of experimental treatment. In Study B, the greatest improvements occurred only after the experimental treatment phase (from 19% pre-treatment to 64% post-treatment improvement in FFI, and from 31% pre-treatment to 78% post-treatment for PIS).

We partially attribute our observed outcomes to the targeted treatment of myofascial trigger points. By definition, a trigger point is a hypersensitive zone of harder than normal consistency, which triggers pain when stimulated.[27,28,29,30] The most pathognomonic symptom of myofascial pain syndrome is the presence of pressure sensitive palpable nodules that replicate the chief complaint of the patient.[30] Trigger points are thought to develop after trauma, overuse or prolonged spasm of muscle.[29,31] Ischemic compression has been demonstrated as being effective in the treatment of TrPs.[27,32,33] When the treatment is given carefully, by paying attention to the patient's slightest reaction and asking if the pain is bearable, it is likely to be well tolerated.

We also partially attribute our observed outcomes to the mobilization component of our management approach. Joint mobilization involves manual techniques without thrusting or sudden movement. Repetitive passive movement of a skeletal joint is performed with the aim of achieving a therapeutic effect.[34] In the present studies, the need for joint mobilization was determined by detection of a loss of joint play and/or provocation of pain during forceful flexion, extension, or rotation of the articulation of the foot. Subsequently, treatment consisted of forcing passive movement through the painful articulation, to the patient's limit of tolerance, and then maintaining that forceful contact for a duration of 15 seconds. This technique was repeated at each visit until the pain was completely eliminated or until the end of the 15 treatments.

We have found few clinical trials using mobilization, ischemic compressions, or massage to counter foot pain. Existing studies were conducted on patients with plantar fasciitis and hallux problems. Brantingham conducted a randomized clinical trial of the treatment of hallux abductovalgus (bunions).[17] Sixty subjects were randomized to two groups. The experimental group received mobilization of the first metatarsophalangeal articulation, in conjunction with cryotherapy and adjustment of all other putative fixations found in the foot and ankle. The placebo group received de-tuned microcurrent therapy. Six treatments were given over a two-week period. Outcomes were measured weekly throughout the treatment period and then at only one week post-treatment. In terms of objective findings, the experimental group showed a statistically significant improvement post-treatment, which was not the case in the control group. In terms of patients' subjective response to treatment, both groups experienced a statistically significant decrease in pain perception; however, the experimental group achieved a statistically significant improvement at one week whereas the placebo group did so only at three weeks. Furthermore, a statistically significant improvement in foot pain and related disability (as measured using the Foot Function Index) was observed in the experimental group only.[17]

Another study by Brantingham, this time on plantar fasciitis, involved a retrospective review of files of 29 patients who were treated with manipulations, physiotherapy, orthotics, and soft tissue massage of the plantar fascia.[35] Twenty-two of the 29 patients demonstrated excellent results (defined as greater than 75% pain reduction) following an average of eight treatments.

Sweeting et al. conducted a systematic review of the effectiveness of stretching in the treatment of plantar heel pain.[36] Six studies involving 365 symptomatic patients were included in the analysis. They reported that most patients with plantar heel pain who stretch tend to improve over time in terms of both pain and function, but when stretching is compared to other interventions, including sham treatment, no statistically significant benefit was observed.

Study limitations

Our studies had several limitations. We did not assess the reliability of our diagnostic protocol for detecting trigger points and foot dysfunction in advance. However, our

studies were intended to be preliminary investigations of the effectiveness of a manual therapy protocol on trigger points and joint hypomobility, as customarily diagnosed in everyday clinical practice.

We acknowledge that our outcomes were collected by the principal investigator who was aware of both the study questions and the phase of treatment for each participant at each follow-up visit. Having utilized only two treating doctors, our interventionists were not necessarily representative of other chiropractors in the general community. Furthermore, while we detected statistically significant differences, our sample sizes were small and therefore our point estimates are likely unstable and will require further corroboration in larger studies.

Also, our findings are potentially vulnerable to various threats to validity that are specifically associated with quasi-experimental studies.[37] One important threat to the validity of findings from a before-and-after study is the potential effect of history, meaning that some events occurring externally to the study, but concurrently with treatment, may affect outcomes independently of the experimental treatment. However, to our knowledge, our study participants were not systematically exposed to any events in the community (e.g., a public health initiative for foot disorders) that might have coincidentally caused the effects that we observed in our studies. A more likely threat to validity in our studies is maturation, or the effect of naturally occurring changes in outcomes (i.e., foot pain and related disability) over time, which could easily be confused with the effect of experimental treatment. However, this phenomenon is unlikely to account for the fact that the timing of clinical improvement among both of our study cohorts coincided precisely with the timing of two differently timed intervention periods (early treatment in Study A, and delayed treatment in Study B). Finally, regression to the mean is yet another potential threat to the validity of our observed effects. Admittedly, our study participants may have been seeking treatment (and were therefore enrolled) at a time when they were particularly disabled by their symptoms. In such patients, a natural regression from higher to lower disability status over the study period might have occurred independently of treatment, however regression to the mean would also not explain the observed consistency in the timing of observed improvements immediately after each of two differently timed experimental treatment periods. Moreover, the Study B cohort was exposed to an initial five-week monitoring period, at the end of which little to no regression of baseline outcomes was observed.

In Study B, our findings were potentially susceptible to the effect of experimental mortality in that two subjects declined experimental treatment after the initial monitoring phase and were therefore not included in the analysis. Finally, participants were not blatantly aware of the researchers' expectations but may have had personal expectations for improvements in outcomes following experimental manual therapy exclusively.

Conclusion

Our findings constitute preliminary evidence that myofascial therapy consisting of ischemic compressions and joint mobilizations may reduce the symptoms of patients suffering from chronic non-specific foot pain. The study intervention was heterogeneous and tailored to each individual patient, however it was applied pragmatically and consistent with the way in which it would be administered customarily in everyday clinical practice. A treatment period of five weeks was associated with improved self-reported outcomes. We hope these preliminary findings provide a foundation for other researchers to further evaluate the effectiveness of our approach within randomized controlled trials.

References

1. Hill CL, Gill TQ, Menz HB, Taylor AW. Prevalence and correlates of foot pain in a population-based study. J Am Med Assoc. 1987;77:308-311.
2. DeMaio M, Paine R, Mangine RE et al. Plantar fasciitis. Orthopedics.1993; 16:1153-63.
3. Nix S, Smith M, Vicenzino B. Prevalence of hallux valgus in the general population: a systematic review and meta-analysis. J Foot Ankle Res. 2010;3:21.
4. Polkinghorn BS. Posterior calcaneal subluxation: an important consideration in chiropractic treatment of plantar fasciitis (heel spur syndrome). Chirop Sport Med. 1995; 9(2):44-51.
5. Stuber K, Kristmanson K. Conservative therapy for plantar fasciitis: a narrative review of randomized controlled trials. J Can Chirop Assoc. 2006; 50(2):118-33.
6. Pack LG, Julien PH. Differential diagnosis of lesser metatarsalgia. Clin Podiat Med Surg. 1990; 7(4):573-7.
7. Kasmahl EM, Kosmahl EK. Painful plantar heel, plantar fasciitis, and calcaneal spurs: etiology and treatment. J Orthop Sport Ther. 1987;9(1):17-24.
8. Davison WT, Hyde TE, Reicher DL, Werner JS. Grand

Rounds: Heel pain. J Neuromusculoskeletal System. 1997; 5(1):39-46.
9. Shapiro SL. Heel pain management starts with correct differential diagnosis. Biomechanics. 1997; 4(9):25-27.
10. Dimou ES, Brantingham JW, Wood T. A randomized controlled trial of chiropractic manipulation and achilles stretching vs. orthotics for the treatment of plantar fasciitis. J Am Chirop Assoc. 2004; 41(9):32-42.
11. Gill LH. Plantar fasciitis: diagnosis and conservative management. J Am Acad Orthop Surg. 1997; 5(2):109-117.
12. Speed C. A systematic review of shockwave therapies in soft tissue conditions: focussing on the evidence. Br J Sports Med. 2014; 48(21):1538-1542.
13. Brantingham JW, Chang MN, Gendreau DF, Price JL. The effect of chiropractic adjusting, exercises and modalities on a 32-year-old professional male golfer with hallux rigidus. Clinical Chiropractic. 2007; 10(2):91-96.
14. GlascoW, Glasco G. Conservative evaluation and intervention of a sport-related injury: turf toe. Sport Chirop Rehab. 1998; 12:2.
15. Ferrari J, Higgins JPT, Prior TD. Intervention for treating Hallux Valgus and Bunions (Review).The Cochrane Library 2007; Issue 4.
16. Manral DB. Hallux rigidus: a case report of successful chiropractic management and review of the literature. J Chirop Med. 2004; 3:1.
17. Brantingham JW, Guiry S, Kretzmann HH et al. A pilot study of the efficacy of a conservative chiropractic protocol using graded mobilization, manipulation and ice in the treatment of symptomatic hallux abductovalgus bunion. Clinical Chiropractic. 2005; 8:117-133.
18. Quirk R. Metatarsalgia. Austr Fam Phys. 1996; 25(6):863-69.
19. Thomson CE, Gibson JNA, Martin D. Interventions for the treatment of Morton's Neuroma (Review). The Cochrane Library 2007, Issue 4.
20. Subonick S. Peroneal Cuboid Syndrome: an often overlooked cause of lateral column foot pain. Chiropractic Technique. 1998; 10(4):156-162.
21. Watt LH. Conservative chiropractic management of recalcitrant foot pain after fasciotomy: a retrospective case review. J Manip Physiol Thera. 2006; 29(5):398-402.
22. Solan M, Davies M. Management of insertional tendinopathy of the achilles tendon. Foot Ankle Clin Am. 2007; 12:597-615.
23. Travel JG, Simons DG. Myofascial Pain and Dysfunction; the Trigger Point Manual, Vol.1. Philadelphia: Williams and Wilkins;1983. p.19.
24. Graff-Radford SB. Myofascial pain: diagnosis and management. Current Pain and Headache Reports. 2004; 8:463-467.
25. Looney B, Srokose T, Fernandez-de la Penas C, Cleland JA. Graston instrument soft tissue mobilization and home stretching for the management of plantar heel pain: a case series. J Manip Physiol Thera. 2010; 32(2):138-141.
26. Budiman-Mak E, Conrad KJ, Roach KE. The foot function index: a measure of foot pain and disability. J Clin Epidemiol. 1991; 44(6):561-570.
27. Gerwin RD, Dommerholt J, Shah JP. An expansion of Simons' integrated hypothesis of trigger point formation. Current Pain and Headache Report. 2004; 8:468-475.
28. Alvarez DJ, Rockwell PG. Trigger points: diagnosis and management. Am Fam Phys. 2002; 65(4):653-660.
29. Schneider MJ. Tender points/fibromyalgia vs. trigger points/myofascial pain syndrome: a need for clarity in terminology and differential diagnosis. J Manip Physiol Thera. 1995; 18(6):398-406.
30. Borg-Stein J, Stein J. Trigger points and tender points. Rheumatic Dis N Am. 1996; 22(2):305-323.
31. Fischer AA. New approaches in the treatment of myofascial pain. Phys Med Rehab Clin Am. 1997; 8(1):153-169.
32. Fernandez-de-la-Penas C, Alonso-Blanco C, Fernandez-Carnero J et al. The immediate effect of ischemic compression technique and transverse friction massage on tenderness of active and latent myofascial trigger points: a pilot study. J Bodywork and Mov Thera. 2006; 10:3-9.
33. Hou CR, Tsai LC, Cheng KF et al. Immediate effects of various physical therapeutic modalities on cervical myofascial pain and trigger points sensibility. Arch Phys Med Rehabil. 2002; 83:1406-1414.
34. Wikipedia/Joint-mobilization 2012.
35. Brantingham JW, Snyder WR, Dishman RW et al. Plantar fasciitis. Chiropractic Technique. 1992;4:3.
36. Sweeting D, Parish B, Hooper L et al. The effectiveness of manual stretching in the treatment of plantar heel pain: a systematic review. J Foot Ankle Res. 2011; 4:19.
37. Harris AD, McGregor DE, Perencevich EN et al. The use and interpretation of quasi-experimental studies in medical informatics. J Am Med Informatics Assoc. 2006; 13(1):16-23.

Note: Dr. Guy Hains DC passed away on October 13, 2014. He published a number of clinical studies over the last decade and made significant contributions to chiropractic research.

Trigger Point Dry Needling: An Evidenced and Clinical-Based Approach
Jan Dommerholt and Cesar Fernandez-de-las-Peñas
Churchill Livingstone, 2013, 280 pp.,
Hardcover, Price: CAN $108.00
ISBN: 978-0-7020-4601-8

While trigger point dry needling is becoming a popular treatment choice for myofascial pain within North America, there are few texts that adequately present this topic in a research driven format while maintaining a clinical approach. With *Trigger Point Dry Needling: An Evidenced and Clinical-Based Approach,* Jan Dommerholt and Cesar Fernandez-de-las-Penas accomplish both.

Part one of this text presents the basic concepts and theories that surround myofascial trigger points, offering the theorized mechanisms of dry needling from a basic science and clinical research perspective. This section reviews adverse events as well as offering guidelines to improve safety for both the patient and the clinician (aiming to reduce needle stick injury risk).

Part two of this text reviews commonly treated muscles within six regional areas, each introduced with clinical research. Excellent photography and detailed instruction helps to illustrate the practical application of dry needling techniques.

Part three reviews alternate approaches to dry needling techniques, such as superficial dry needling and intramuscular stimulation (IMS). A chapter presenting dry needling from a western medical acupuncture perspective adds depth and balance to the text as a whole.

I would recommend this text to clinicians seeking a reference manual in this technique. The text is well written and referenced, proving to be helpful in answering questions surrounding the clinical rationale and theories around dry needling as well as offering an excellent instructional review of the technique itself.

Robert Rodine, BSc, DC, FCCS(C)
Private Practice
Smiths Falls, Ontario, K7A 1M9
Tel: +1 613 205 0978
Email: robrodine@hotmail.com