Journal of Bodywork & Movement Therapies (2011) 15, 130–152

available at www.sciencedirect.com

ScienceDirect

journal homepage: www.elsevier.com/jbmt

Is a postural-structural-biomechanical model, within manual therapies, viable?: A JBMT debate

Leon Chaitow, ND DO

Honorary Fellow, University of Westminster, UK

Eyal Lederman DO: The fall of the postural-structural-biomechanical model in manual and physical therapies: Exemplified by lower back pain

With invited responses from:

Gary Fryer PhD
John Hannon DC
Robert Irvin DO
Diane Lee PT
Stuart McGill PhD

Introduction to the debate

Leon Chaitow ND DO

In recent years, a number of leading thinkers within manual and physical therapies have questioned traditional models of care.

Professor Eyal Lederman DO PhD is one such critic, of what he has termed the *postural-structural-biomechanical* (PSB) model. Using low back pain as a focus he has asserted that the PSB model is not viable — in fact that there is no reliably proven relationship between low back pain and posture, structure or biomechanical features. — and by implication, that attempts to treat and normalize such features, are meaningless, and a waste of time and resources. Lederman asserts: *"We can no longer justify the use of manual techniques to readjust, correct or balance-out the misaligned structure."* The suggestion is that rehabilitation strategies, motor re-education approaches, behaviorally focused methodologies — are the best means of achieving resolution and prevention of dysfunctional states such as low back pain, and that manual modalities have - at most - short-term effects, and are largely redundant (Lederman, 2010)

It can be counter-argued that unless and until postural balance, mobility (i.e. more normal ranges of motion, including fascial) strength and endurance features are restored, via manual therapy strategies, normal pain-free function may be far more difficult to achieve by means of rehabilitation strategies. (Irvin et al., 2007; Chaitow, 2010)

Dr Lederman's assertions challenge the practices of many — probably most — manual and physical therapists. Indeed, in his presentation of these ideas he challenges the very foundation of much of what is currently practiced by the majority of physiotherapists, osteopaths, chiropractors, massage therapists, as well as those engaged in many of the prevention and rehabilitation methodologies currently in use.

What Lederman suggests, as a replacement for what he terms the postural-structural-biomechanical model, can be evaluated in his invited article (below), that sets out his considered thoughts. Basically he proposes that what is required in any given case, is identification of the processes underlying the patient's condition, followed by provision of *"the stimulation/signals/management/care that will support/assist/facilitate change"* — what he terms a '*process approach*'. In itself this argument is not controversial — however the implications Lederman draws from his thesis goes on to suggest that passive manual treatment has little value in the healing, recovery, process.

Anyone who attended the recent (November 2010) 7th Interdisciplinary Congress on Low Back and Pelvic Pain would have heard presentations that, in many ways, echo and support Lederman's position (as well as many presentations that did not agree with it). For example O'Sullivan (2010) has advocated a biopsychosocial approach to back pain with particular focus on underlying mechanisms that may be driving pain disorders. Included in that model are evaluations of whether there have been adaptive or maladaptive motor responses to the condition. O'Sullivan's emphasis is therefore to divert patient care away from

1360-8592/$ - see front matter © 2011 Elsevier Ltd. All rights reserved.
doi:10.1016/j.jbmt.2011.01.004

manual treatment, towards cognitive and re-educational strategies - which is a less extreme position to that taken by Lederman.

In order to unpick these arguments, JBMT has invited five leading experts from the world of manual medicine to respond to Dr Lederman's thesis. These clinicians (from the world of osteopathy, chiropractic and physiotherapy) as well as researchers into biomechanical dysfunction and therapeutic methods, offer their perspectives,in alphabetical order, following Dr Lederman's paper.

It is important to observe that by no means all of these expert responses are wholly critical of Dr. Lederman's arguments, and ultimately it is for you, the reader, to decide which aspects of the debate are most persuasive, and where the relative clinical truth lies.

It is hoped that this examination of controversial ideas will lead to a better understanding of what we hold to be true, based on current evidence, as well as what we need to question, what we need to know, and how we need to practice.

References

Chaitow, L., 2010. Fascia Directed Therapies for the Treatment of Low Back Pain: Review and New Directions. Presentation: 7th Interdisciplinary World Congress on Low Back and Pelvic Pain. Los Angeles. November 11 2010.

Irvin, R.E., 2007. Why and how to optimize posture, chapter 16. In: Vleeming, et al. (Eds.), Movement, Stability and Lumbopelvic Pain, second ed. Elsevier, Edinburgh.

Lederman, E., March 2010. The fall of the postural-structural-biomechanical model in manual and physical therapies: exemplified by lower back pain. CPDO Online J. http://cpdo.net/jour/jour1.html.

O'Sullivan, P., 2010. Diagnosis and Classification of Chronic Low Back Disorders. Proceedings Book 7th Interdisciplinary World Congress on Low Back and Pelvic Pain, pp. 160—177.

The fall of the postural-structural-biomechanical model in manual and physical therapies: Exemplified by lower back pain

Eyal Lederman, PhD DO

CPDO Ltd, 15 Harberton Road, London N19 3JS, UK

Summary Manual and physical therapists often use a postural-structural-biomechanical (PSB) model to ascertain the causes of various musculoskeletal conditions. It is believed that postural deviations, body asymmetries and pathomechanics are the predisposing/maintaining factors for many musculoskeletal conditions. The PSB model also plays an important role in clinical assessment and management, including the choice of manual techniques and the exercise prescribed. However, the most important question is consistently being ignored — *can a person's physical shape/posture/structure/biomechanics be the cause of their lower back pain?*

.

Is development of LBP associated with PSB factors?

In the last two decades the PSB model has been eroded by clinical studies examining the relationship between PSB factors and lower back pain. The balance of evidence suggests that there is no association between LBP and PSB factors (see summary, Figure 1).

Prospective studies are particularly useful to examine the causal relationship between PSB factors and LBP. In these studies groups of asymptomatic individuals are assessed for PSB factors initially and tracked over several years noting the episodes of LBP. Other less ideal studies compare subjects with LBP to an asymptomatic group. However these studies can only be used to inform us about the changes that are due to the condition but they cannot indicate its cause, i.e. the consequence of LBP is not necessarily its cause. This distinction is important clinically. Often the PSB assessment is made when the patient is already in pain, once the individual/body has reorganized to cope with the condition.

E-mail address: cpd@cpdo.net.

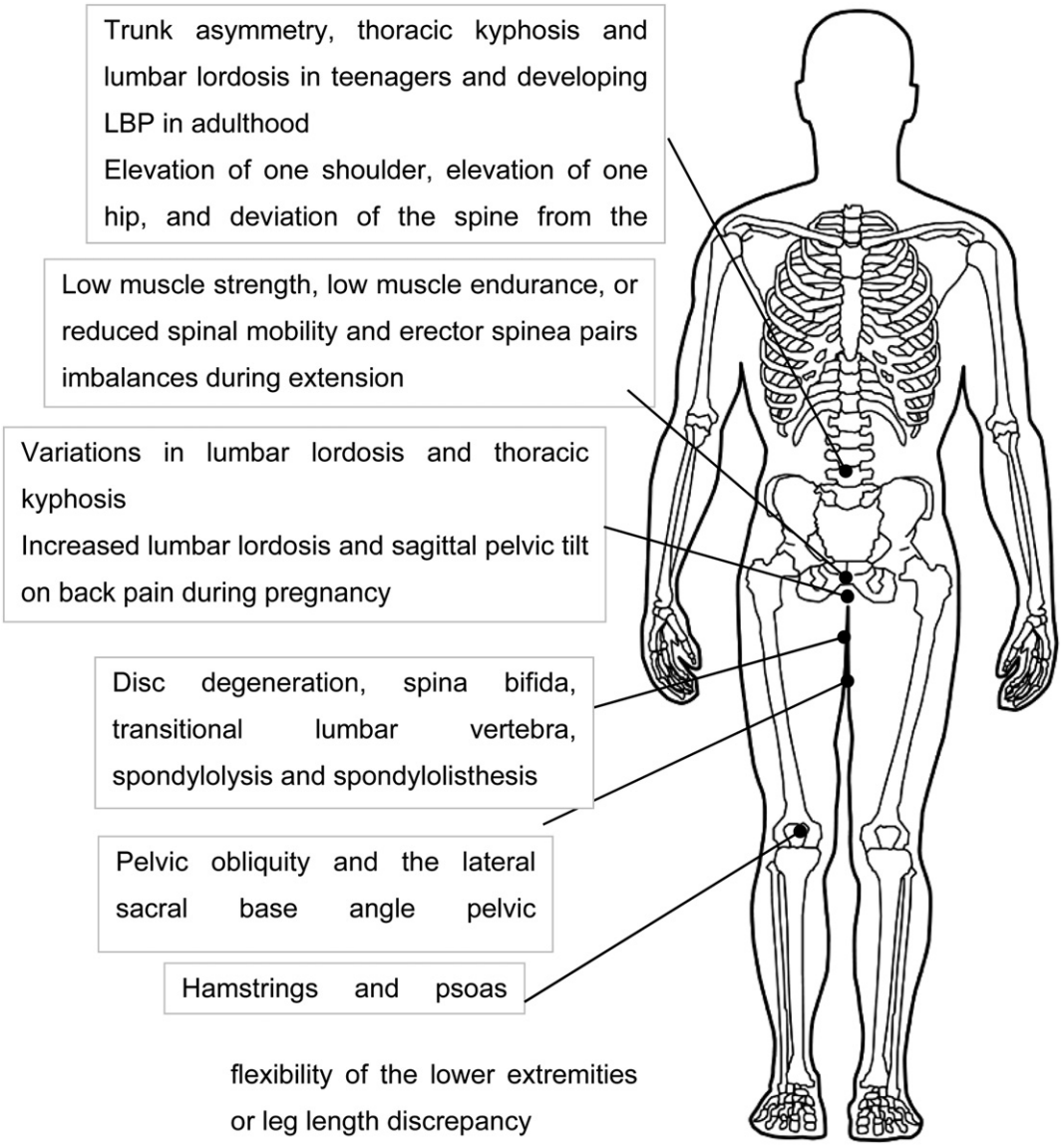

Figure 1 PSB actors not associated with LBP.

Spinal curves, asymmetry and motion

There was the lack of association between postural spinal asymmetry, thoracic kyphosis and lumbar lordosis in teenagers and developing LBP in adulthood (Papaioannou et al., 1982; Dieck, 1985; Poussa, 2005). Even obvious increases in lordosis and sagittal pelvic tilt during pregnancy lack an association with back pain (Franklin and Conner-Kerr, 1998). Stronger predictors of the development of back pain during pregnancy were body mass index, history of hypermobility and amenorrhea, low socioeconomic class, previous LBP, posterior fundal location of placenta and fetal weight to LBP with radiation to leg (Orvieto et al., 1990; Mogren and Pohjanen, 2005).

In adults, the extent of lumbar lordosis as well as the presence of scoliosis failed to show an association with back pain (Dieck, 1985; Haefeli et al., 2006; Norton, 2004; Christensen and Hartvigsen, 2008, syst. rev.) Also differences in regional lumbar spine angles or range of motion between the segments failed to show an association with the future development of LBP (Hellsing, 1988b; Burton and Tillotson, 1989; Hamberg-van Reenen HH 2007, syst review; Mitchell et al., 2008).

Segmental pathomechanics

One important area to examine is whether the profound biomechanical changes brought about by segmental biomechanics can give rise to lower back symptoms.

A systematic review from 1997 suggests an association between disc degeneration and non-specific low back pain (van Tulder et al., 1997). However, it might not be the cause of it -there is strong evidence that X-ray and MRI findings have no predictive value for future LBP or disability (Waddell and Burton, 2001, review). Several studies since have failed to show a clear relationship between spinal/disc degeneration and LBP (Savage et al., 1997; Borenstein et al., 2001; Jarvik et al., 2005; Carragee et al., 2005;

Kanayama et al., 2009; Kalichman et al., 2010). In a population-based study of 34,902 Danish twins 20—71 years of age there were no meaningful differences in the frequency in LBP between younger and older individuals (Leboeuf-Yde et al., 2009), although greater degenerative changes are expected in older individuals.

In studies that show some relationship between disc degeneration and LBP it has been suggested that the genes that play a part in the heritability of back pain also play a part in disc degeneration, i.e. pain may not be due to the mechanical changes in the spine but to shared biological factors (Battie et al., 2007). These hereditary factors are not associated with the shape of the back but linked to variations in the collagen and immune-repair system/processes between individuals (Paassilta et al., 2001; Valdes et al., 2005; Battié et al., 2009; Videman, 2009). It was demonstrated in twins that as much as 47%—66% of spinal degeneration is due to hereditary and shared environmental factors, whereas only 2%—10% of the degeneration can be explained by physical stresses imposed by strenuous occupations or sporting activities (Battié, 1995; Battié et al., 2009; Videman et al., 2006 and 2007).

No association has been found between congenital abnormalities in the lumbar spine and pain in that area (spina bifida, transitional lumbar vertebra, spondylolysis and spondylolisthesis: van Tulder et al., 1997, syst. review, Luoma et al., 2004; Brooks et al., 2009). Although spina bifida and transitional vertebra may not be the cause of LBP, they may determine the pain levels (Taskaynatan et al., 2005, weaker study).

Another popular and enduring biomechanical concept is the spinal "neutral zone". It claims to be related to stability and LBP (Panjabi, 1992a and b; Panjabi, 2003; Suni et al., 2006). This mechanical concept is derived from mathematical models and cadaver experiments on which an extensive amount of spinal joint damage had to be inflicted before the findings could fit the model (Gracovetsky, 2005). Since its inception three decades ago, no study exists to show a correlation between mechanical changes in the neutral zone changes and LBP (Leone et al., 2007, review).

The disparity between pathomechanics and symptomatology can be observed in other segmental conditions. For example, in an MRI study of patients with nerve root pain it was found that the degree of disc displacement, nerve root enhancement or nerve compression did not correlate with the magnitude of the patients' subjective pain or level of functional disability (Karppinen et al., 2001; see also Beattie et al., 2000). However there is a strong association between severe nerve compression, disc extrusion and distal leg pain (Beattie et al., 2000).

Non-spinal structures

Studies have also failed to identify an association between other structures beyond the spine and back pain. For example, there is no correlation between pelvic obliquity/asymmetry and the lateral sacral base angle and lower back pain (Dieck, 1985; Levangie, 1999a and b; Fann, 2002; Knutson, 2002).

Leg length differences as a cause for back pain has been debated for the last three decades. It is estimated that about 90% of the population has a leg length inequality with a mean of 5.2 mm. The evidence suggests that for most people anatomic leg length inequality is not clinically significant (Papaioannou et al., 1982; Grundy and Roberts, 1984; Dieck, 1985; Fann, 2002; Knutson, 2005, review), until the magnitude reaches approximately 20 mm (Gurney, 2002 review; Knutson, 2005, review). Although some earlier studies comparing people experiencing back pain with asymptomatic controls suggest a correlation (Giles and Taylor, 1981; Friberg, 1983 and 1992), more relevant are prospective studies in which no correlation was found between leg length inequality and LBP (Hellsing, 1988a; Soukka et al., 1991; Nadler, 1998).

Patients who have acquired their leg length differences later in life as consequence of disease or surgery may also help to shed light on the relationship between pathomechanics and LBP. Individuals who developed a shorter leg due to Perthe's disease had a poor correlation between leg length inequality, lumbar scoliosis and low-back disorders, assessed several decades after the onset of the condition (Yrjönen et al., 1992). In studies of patients who had marked changes in leg length due to hip fractures or replacement, such changes were not associated with back pain assessed several years after surgery (Gibson et al., 1983; Edeen et al., 1995; Parvizi et al., 2003).

One of the arguments in favor of an association between leg length differences and LBP is the supposed success of heel lifts in reducing back pain (Giles and Taylor, 1981; Gofton, 1985; Helliwell, 1985; Friberg, 1983 and 1992; Brady et al., 2003 review). However, all these studies failed to include controls or sham heel lift (such as inefficient soft foam lift).

Prospective studies of inflexibility of the lower extremities and hamstrings and psoas tightness also fail to predict future episodes of LBP (Hellsing, 1988c; Nadler, 1998).

As for foot biomechanics there is strong evidence that orthotic corrections have no effect on preventing back pain (Sahar et al., 2007, syst. review).

Surprisingly even whole body changes such as overweight/obesity have a low association with LBP (Leboeuf-Yde, 2000 syst. review). Contrary to common beliefs, a recent study has shown that cumulative or repetitive loading due to higher body mass (nearly 30 pounds on average) was not harmful to the discs. The study found a slight delay in disc desiccation (L1-L4) in the heavier men when compared with their lighter twin brothers (Videman et al., 2009).

Neuromuscular factors

Although not fully within the scope of this article, motor control of the trunk is relevant in relation to muscle function and posture. Certain neuromuscular components also failed to show a clear association with LBP.

Although earlier studies demonstrated an association between muscle endurance and LBP (Biering-Sørensen, 1984; Alaranta et al., 1995), a recent systematic review found strong evidence that low trunk muscle endurance is not associated with LBP (Hamberg-van Reenen, 2007 syst. rev). This review found inconclusive evidence for an association between low trunk muscle strength and LBP. Also there is no association between erector spinae pairs imbalances during extension and LBP (Reeves et al., 2006;

Hamberg-van Reenen, 2007 syst. rev; Van Nieuwenhuyse et al., 2009). Furthermore, no study to date has shown that back pain is due to timing differences in specific muscle such as transversus abdominis (see discussion Lederman, 2010b). These control changes have been observed only in individuals who already have back pain. They probably represent the outcome rather than the cause of back pain (see discussion Lederman, 2010a).

Two studies using the same methodology appear to demonstrate that in athletes a delayed reflex muscle response at the trunk could increase the risk of lower back as well as knee injury (Cholewicki et al., 2005; Zazulak et al., 2007). Unfortunately, the obvious was not examined in these studies — the reflex response to a sudden perturbation of the trunk should have been examined in other body areas (e.g. a control recording from the leg). This would have helped establish whether the injuries are due to delayed muscle onset-timing, specific to the trunk or, the alternative more plausible explanation that athletes with sluggish muscle reaction times/reflexes may be more susceptible to injury.

Postural behavior factors

An area that is often assessed in manual and physical therapy is how "correctly" a person is using their body — their "postural behavior". It is believed that prolonged postural stresses at work or sporting activities could be the cause of LBP. The results of recent systematic reviews challenge these widely held beliefs. These studies demonstrate lack of association between work-related posture and LBP. They include postures such as prolonged standing, bending, twisting, awkward postures (kneeling or squatting) sitting posture at work and prolonged sitting at work and leisure time (Hartvigsen et al., 2000 syst. review; Chen et al., 2009 syst. review; Bakker et al., 2009 syst. review; Roffey et al., 2010 syst. review; Wai et al., 2010, syst. review). Also physical leisure time activities such as sport or exercises, sitting, and prolonged standing/walking were found not to be associated with LBP (Bakker et al., 2009 syst. review). Heavy manual lifting is strongly associated with LBP, however the effect size is considered to be modest (Waddell and Burton, 2001 review).

Prediction of back pain by PSB assessment

In a recent prospective study on young workers ($n = 692$) examined by physical therapists, PSB factors failed to show a correlation with future development of LBP (Van Nieuwenhuyse et al., 2009). A number of factors were evaluated including iliac crest height inequality, scoliosis, lumbar flexion, extension and lateral flexion, length of hamstring muscles and strength testing in the motor distribution of L4/L5/S1.

Biological not mechanical

There seems to be a disparity between pathomechanics and the experience of a low back condition. In this biomechanical model the musculoskeletal system is seen as a precision engine where every system, organ and cell works in perfect harmony within itself and other body systems. However, the research suggests that biological systems contain reserve capacity to accommodate for loss and imperfections without failure or symptoms. Furthermore, within a biological dimension, structures such as the spine are capable of self-repair and are able to adapt and change according to needs and demands. Hence, the spine can undergo profound physical changes that are well tolerated without the development of a symptomatic condition.

Perhaps there is a critical level where PSB factors will exceed the reserve of the system. Clinically, this still remains unquantified (and probably unquantifiable). If we were to overlook this obstacle, the next hurdle to overcome is the reliability of assessing PSB factors. It is now well established that many of the examinations that assess PSB factors are either low on validity or reliability (McCaw and Bates, 1991; Mannello, 1992; Panzer, 1992; Levangie, 1999a; Hestbaek and Leboeuf-Yde, 2000; Seffinger et al., 2004; Dunk et al., 2004; van Trijffel et al., 2005; Hollerwöger, 2006; May et al., 2006; Paulet and Fryer, 2009). A third clinical hurdle to overcome is whether manual techniques or specific exercise are effective in modifying inherent PSB factors. Can foot mechanics, leg length differences, pelvic tilts, vertebral positions and spinal curves be permanently changed, solely, by these clinical tools?

There are no known studies that examine the influence of manual techniques on PSB factors in the medium- or long-term, in particular at the cessation of the treatment. However we know from the allied sciences that a herculean effort would be required to modify many of the inherent PSB factors (Maruyama et al., 2008; Maruyama, 2008; Lonstein, 1999; Marks and Qaimkhani, 2009; Willy et al., 2001; Harvey et al., 2002; Williams et al., 1986; Goldspink et al., 1992; Arnoczky et al., 2002; Bosch et al., 2002; Magnusson et al., 1995; Magnusson, 1998; Light et al., 1984; Roberts and Wilson, 1999. see full discussion of this topic and references in, Lederman, 2010a). As such, the therapeutic investment in correcting PSB factors is irrational, in particular, as it is unlikely to influence the course of the patient's LB condition.

Implications for practice

The lack of association between PSB factors and back pain has far-reaching implications for the way we conceptualize musculoskeletal conditions, the clinical examination and the goals/objectives of the techniques and the exercise prescribed.

From the evidence so far many of the clinical examinations assessing PSB factors have no obvious value in explaining why the patient has developed their back condition. It implies that the PSB model and the related clinical examinations are mostly redundant.

The lack of association between PSB factors and LBP has also important implications for what we aim to achieve and for our choice of techniques and exercise used to manage the condition. We can no longer justify the use of manual techniques to readjust, correct or balance-out the misaligned structure. There is an urgent need to redefine what

the therapeutic goals are, beyond relieving the patient's symptoms, e.g. is there any value in providing long-term maintenance/preventative treatments for asymptomatic individuals?

Furthermore, the therapeutic ideal of a "cure" may not be possible, as the underlying condition could still be present but is asymptomatic. Perhaps research and treatment should be directed towards finding better approaches to provide symptomatic relief during periods of pain as well as increasing the patient's participation in social, occupational and recreational activities (Waddell and Burton, 2001; Waddell et al., 2008; Kendall et al., 2009). This attitude may be more realistic than the idealized clinical aspiration to provide a permanent cure by correcting PSB factors.

Finally and more complex is the therapists' education in the various manual and physical therapies where the PSB model is dominant. If this model is flawed what is the alternative clinical model and who is capable of teaching it?

The alternative: a process approach

A clinical alternative to the PSB model is a Process Approach model. In this approach the aim is to identify the processes underlying the patient's condition and provide the stimulation/signals/management/care that will support/assist/facilitate change. This approach has been extensively discussed in Lederman E (2005) and will be discussed in a future article.

Summary and conclusion points

PSB asymmetries and imperfections are normal variations — not a pathology.
Neuromuscular and motor control variations are also normal.
The body has surplus capacity to tolerate such variation without loss to normal function or development of symptomatic conditions.
Pathomechanics do not determine symptomatology.
There is no relationship between the pre-existing PSB factors and back pain.
Correcting all PSB factors is not clinically attainable and is unlikely to change the future course of a lower back condition.

Clinically:

Observational or physical assessments of PSB factors have no value in elucidating the causes for back pain.
Clinical assessment of PSB factors assessed by manual and visual means may be unreliable.
Such assessments are likely to be redundant and can be safely removed from clinical practice. This excludes assessment that aim to identify serious pathologies.
PSB factors are unlikely to change in the long-term by manual techniques or even exercise, unless rigorously maintained (exercise).
A PSB model may introduce an element of therapeutic failure as the aims and goals of this approach may not be attainable by manual therapy or even exercise.

References

Alaranta, H., Luoto, S., Heliövaara, M., Hurri, H., 1995 Sep. Static back endurance and the risk of low-back pain. Clin. Biomech. (Bristol, Avon) 10 (6), 323—324.

Arnoczky, S.P., Tian, T., Lavagnino, M., Gardner, K., Schuler, P., Morse, P., 2002. Activation of stress-activated protein kinases (SAPK) in tendon cells following cyclic strain: the effects of strain frequency, strain magnitude, and cytosolic calcium. J. Orthop. Res. 20 (5), 947—952.

Bakker, E.W., Verhagen, A.P., van Trijffel, E., et al., Apr 15 2009. Spinal mechanical load as a risk factor for low back pain: a systematic review of prospective cohort studies. Spine (Phila Pa.) 34 (8), E281—E293.

Battié, M.C., Dec 15 1995. Volvo Award in clinical sciences. Determinants of lumbar disc degeneration. A study relating lifetime exposures and magnetic resonance imaging findings in identical twins. Spine 20 (24), 2601—2612.

Battié, M.C., et al., 2007. Heritability of low back pain and the role of disc degeneration. Pain 131, 272—280.

Battié, M.C., et al., 2009 Jan-Feb. The twin spine study: contributions to a changing view of disc degeneration. Spine J. 9 (1), 47—59.

Beattie, P.F., Meyers, S.P., Stratford, P., et al., Apr 1 2000. Associations between patient report of symptoms and anatomic impairment visible on lumbar magnetic resonance imaging. Spine 25 (7), 819—828.

Biering-Sørensen, F., 1984 Mar. Physical measurements as risk indicators for low-back trouble over a one-year period. Spine (Phila Pa. 1976) 9 (2), 106—119.

Borenstein, D.G., O'Mara Jr., J.W., Boden, S.D., et al., Sep 2001. The value of magnetic resonance imaging of the lumbar spine to predict low-back pain in asymptomatic subjects: a seven-year follow-up study. J. Bone Jt. Surg. Am. 83-A (9), 1306—1311.

Bosch, U., Zeichen, J., Skutek, M., Albers, I., van Griensven, M., Gassler, N., 2002. Effect of cyclical stretch on matrix synthesis of human patellar tendon cells. Unfallchirurg 105 (5), 437—442.

Brady, R.J., Dean, J.B., Skinner, T.M., Gross, M.T., May 2003. Limb length inequality: clinical implications for assessment and intervention. J. Orthop. Sports Phys. Ther. 33 (5), 221—234.

Brooks, B.K., Southam, S.L., Mlady, G.W., et al., Nov 13 2009. Lumbar spine spondylolysis in the adult population: using computed tomography to evaluate the possibility of adult onset lumbar spondylosis as a cause of back pain. Skeletal Radiol. [Epub ahead of print].

Burton, A.K., Tillotson, K.M., May 1989. Is recurrent low back trouble associated with increased lumbar sagittal mobility? J. Biomed. Eng. 11 (3), 245—248.

Carragee, E., et al., Jan-Feb 2005. Discographic, MRI and psychosocial determinants of low back pain disability and remission: a prospective study in subjects with benign persistent back pain. Spine J. 5 (1), 24—35.

Chen, S.M., Liu, M.F., Cook, J., et al., 2009 Jul. Sedentary lifestyle as a risk factor for low back pain: a systematic review. Int. Arch. Occup. Environ. Health 82 (7), 797—806. Epub 2009 Mar 20.

Cholewicki, J., Silfies, S.P., Shah, R.A., Greene, H.S., Reeves, N.P., Alvi, K., Goldberg, B., 2005 Dec 1. Delayed trunk muscle reflex responses increase the risk of low back injuries. Spine (Phila Pa.) 30 (23), 2614—2620.

Christensen, S.T., Hartvigsen, J., 2008 Nov-Dec. Spinal curves and health: a systematic critical review of the epidemiological literature dealing with associations between sagittal spinal curves and health. J. Manipulative Physiol. Ther. 31 (9), 690—714.

Dieck, G.S., Dec 1985. An epidemiologic study of the relationship between postural asymmetry in the teen years and subsequent back and neck pain. Spine 10 (10), 872—877.

Dunk, N.M., Chung, Y.Y., Compton, D.S., Callaghan, J.P., Feb 2004. The reliability of quantifying upright standing postures as a baseline diagnostic clinical tool. J. Manipulative Physiol. Ther. 27 (2), 91—96.

Edeen, J., Sharkey, P.F., Alexander, A.H., Apr 1995. Clinical significance of leg-length inequality after total hip arthroplasty. Am. J. Orthop. 24 (4), 347—351.

Fann, A.V., Dec 2002. The prevalence of postural asymmetry in people with and without chronic low back pain. Arch. Phys. Med. Rehabil. 83 (12), 1736—1738.

Franklin, M.E., Conner-Kerr, T., Sep 1998. An analysis of posture and back pain in the first and third trimesters of pregnancy. J. Orthop. Sports Phys. Ther. 28 (3), 133—138.

Friberg, O., Sep 1983. Clinical symptoms and biomechanics of lumbar spine and hip joint in leg length inequality. Spine (Phila Pa.) 8 (6), 643—651.

Friberg, O., Apr 1992. Results of radiologic measurements of leg-length inequality (LLI). Spine (Phila Pa.) 17 (4), 458—460.

Gibson, P.H., Papaioannou, T., Kenwright, J., Nov 1983. The influence on the spine of leg-length discrepancy after femoral fracture. J. Bone Jt. Surg. Br. 65 (5), 584—587.

Giles, L.G., Taylor, J.R., Sep-Oct 1981. Low-back pain associated with leg length inequality. Spine (Phila Pa. 1976) 6 (5), 510—521.

Gofton, J.P., Aug 1985. Persistent low back pain and leg length disparity. J. Rheumatol. 12 (4), 747—750.

Goldspink, G., et al., 1992. Gene expression skeletal muscle in response to stretch and force generation. Am. J. Physiol. 262, R356—R363.

Gracovetsky, S., 2005. Stability or controlled instability: evolution at work. In: Vleeming, A., et al. (Eds.), Movement, Stability and Lumbopelvic Pain, second ed. Elsevier 2007, (Chapter 14).

Grundy, P.F., Roberts, C.J., 1984 Aug 4. Does unequal leg length cause back pain? A case-control study. Lancet 2 (8397), 256—258.

Gurney, B., Apr 2002. Leg length discrepancy. Gait Posture 15 (2), 195—206.

Haefeli, R., Elfering, A., Kilian, R., et al., Feb 2006 1. Nonoperative treatment for adolescent idiopathic scoliosis: a 10- to 60-year follow-up with special reference to health-related quality of life. Spine 31 (3), 355—366.

Hamberg-van Reenen, H.H., Jul 2007. A systematic review of the relation between physical capacity and future low back and neck/shoulder pain. Pain 130 (1—2), 93—107.

Hartvigsen, J., et al., Sep 2000. Is sitting-while-at-work associated with low back pain? A systematic, critical literature review. Scand. J. Public Health 28 (3), 230—239.

Harvey, L., Herbert, R., Crosbie, J., 2002. Does stretching induce lasting increases in joint ROM? A systematic review. Physiother. Res. Int. 7 (1), 1—13.

Helliwell, M., 1985. Leg length inequality and low back pain. Practitioner. May 229 (1403), 483—485.

Hellsing, A.L., 1988a. Leg length inequality. A prospective study of young men during their military service. Ups J. Med. Sci. 93 (3), 245—253.

Hellsing, A.L., 1988c. Tightness of hamstring- and psoas major muscles. A prospective study of back pain in young men during their military service. Ups J. Med. Sci. 93 (3), 267—276.

Hellsing, A.L., 1988b. Passive lumbar mobility. A prospective study of back pain in young men during their military service. Ups J. Med. Sci. 93 (3), 255—265.

Hestbaek, L., Leboeuf-Yde, C., May 2000. Are chiropractic tests for the lumbo-pelvic spine reliable and valid? A systematic critical literature review. J. Manipulative Physiol. Ther. 23 (4), 258—275.

Hollerwöger, D., May 2006. Methodological quality and outcomes of studies addressing manual cervical spine examinations: a review. Man. Ther. 11 (2), 93—98. Epub 2006 Feb 17.

Jarvik, J.G., Hollingworth, W., Heagerty, P.J., et al., 2005. Three-year incidence of low back pain in an initially asymptomatic cohort: clinical and imaging risk factors. Spine 30, 1541—1548.

Kalichman, L., Kim, D.H., Li, L., Guermazi, A., Hunter, D.J., 2010. Computed tomography-evaluated features of spinal degeneration: prevalence, intercorrelation, and association with self-reported low back pain. Spine J. [Epub ahead of print].

Kanayama, M., Togawa, D., Takahashi, C., et al., Oct 2009. Cross-sectional magnetic resonance imaging study of lumbar disc degeneration in 200 healthy individuals. J. Neurosurg. Spine 11 (4), 501—507.

Karppinen, J., et al., Apr 1 2001. Severity of symptoms and signs in relation to magnetic resonance imaging findings among sciatic patients. Spine 26 (7) E149—E145.

Kendall, N.A.S., Burton, A.K., Main, C.J., Watson, P.J.on behalf of the Flags Think-Tank, 2009. Tackling Musculoskeletal Problems: A Guide for the Clinic and Workplace - Identifying Obstacles Using the Psychosocial Flags Framework. The Stationery Office, London. ISBN 0 11 703789 2.

Knutson, G.A., 2002. 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. 24, e1. doi:10.1067/mmt.2002.121414.

Knutson, G.A., Jul 20 2005. Anatomic and functional leg-length inequality: a review and recommendation for clinical decision-making. Part I, anatomic leg-length inequality: prevalence, magnitude, effects and clinical significance. Chiropr Osteopat 13, 11.

Leboeuf-Yde, C., 2000. Body weight and low back pain. A systematic literature review of 56 journal articles reporting on 65 epidemiologic studies. Spine.

Leboeuf-Yde, C., Nielsen, J., Kyvik, K.O., Fejer, R., Hartvigsen, J., Apr 20 2009. Pain in the lumbar, thoracic or cervical regions: do age and gender matter? A population-based study of 34,902 Danish twins 20-71 years of age. BMC Musculoskelet. Disord. 10, 39.

Lederman, E., 2005. The Science and Practice of Manual Therapy. Elsevier, Edinburgh.

Lederman, E., 2010a. Neuromuscular Rehabilitation in Manual and Physical Therapies. Elsevier, Edinburgh.

Lederman, E., 2010b. The myth of core stability. J. Bodyw Mov Ther. 14 (1), 84—98. Epub.

Leone, A., Guglielmi, G., Cassar-Pullicino, V.N., Bonomo, L., Oct 2007. Lumbar intervertebral instability: a review. Radiology 245 (1), 62—77.

Levangie, P.K., 1999a. The association between static pelvic asymmetry and low back pain. Spine 24 (12), 1234—1242.

Levangie, P.K., 1999b. Four clinical tests of sacroiliac joint dysfunction: the association of test results with Innominate torsion among patients with and without low back pain. Phys. Ther. 79 (11), 1043—1057.

Light, K.E., Nuzik, S., Personius, W., 1984. Low load prolonged stretch vs. high load brief stretch in treating knee contractures. Phys. Ther. 64, 330—333.

Lonstein, J.E., Jul 1999. Congenital spine deformities: scoliosis, kyphosis, and lordosis. Orthop. Clin. North Am. 30 (3), 387—405. viii.

Luoma, K., Vehmas, T., Raininko, R., et al., Jan 15 2004. Lumbosacral transitional vertebra: relation to disc degeneration and low back pain. Spine (Phila Pa.) 29 (2), 200—205.

Magnusson, S.P., 1998. Passive properties of human skeletal muscle during stretch maneuvers. A review. Scand. J. Med. Sci. Sports 8 (2), 65—77.

Magnusson, S.P., et al., 1995. Viscoelastic response to repeated static stretching in the human hamstring muscle. Scand. J. Med. Sci. Sports 5 (6), 342—347.

Mannello, D.M., Nov-Dec 1992. Leg length inequality. J. Manipulative Physiol. Ther. 15 (9), 576—590.

Marks, D.S., Qaimkhani, S.A., Aug 1 2009. The natural history of congenital scoliosis and kyphosis. Spine (Phila Pa.) 34 (17), 1751—1755.

Maruyama, T., 2008. Bracing adolescent idiopathic scoliosis: a systematic review of the literature of effective conservative treatment looking for end results 5 years after weaning. Disabil. Rehabil. 30 (10), 786—791.

Maruyama, T., Takeshita, K., Kitagawa, T., May 2008. Milwaukee brace today. Disabil. Rehabil. Assist. Technol. 3 (3), 136—138.

May, S., Littlewood, C., Bishop, A., 2006. Reliability of procedures used in the physical examination of non-specific low back pain: a systematic review. Aust. J. Physiother. 52 (2), 91—102.

McCaw, S.T., Bates, B.T., Mar 1991. Biomechanical implications of mild leg length inequality. Br. J. Sports Med. 25 (1), 10—13.

Mitchell, T., O'Sullivan, P.B., Burnett, A.F., et al., Nov 18 2008. Regional differences in lumbar spinal posture and the influence of low back pain. BMC Musculoskelet. Disord. 9, 152.

Mogren, I.M., Pohjanen, A.I., 2005. Low back pain and pelvic pain during pregnancy: prevalence and risk factors. Spine 30 (8), 983—991.

Nadler, S.F., 1998. Low back pain in college athletes: a prospective study correlating lower extremity Overuse or acquired Ligamentous laxity with low back pain. Spine 23 (7), 828—833.

Norton, B.J., Sep 2004. Differences in measurements of lumbar curvature related to gender and low back pain. J. Orthop. Sports Phys. Ther. 34 (9), 524—534.

Orvieto, R., et al., 1990. [Low-back pain during pregnancy]. Harefuah 119 (10), 330—331.

Paassilta, P., et al., 2001. Identification of a novel common genetic risk factor for lumbar disk disease. JAMA 285, 1843—1849.

Panjabi, M.M., 1992a. The stabilizing system of the spine. Part I. Function, dysfunction, adaptation, and enhancement. J. Spinal Disord. 5 (4), 383—389. discussion 397.

Panjabi, M.M., 1992b. The stabilizing system of the spine. Part II. Neutral zone and instability hypothesis. J. Spinal Disord. 5 (4), 390—396. discussion 397.

Panjabi, M.M., 2003. Clinical spinal instability and low back pain. J. Electromyogr. Kinesiol. 13, 371—379.

Panzer, D.M., Oct 1992. The reliability of lumbar motion palpation. J. Manipulative Physiol. Ther. 15 (8), 518—524.

Papaioannou, T., Stokes, I., Kenwright, J., 1982 Jan. Scoliosis associated with limb-length inequality. J. Bone Jt. Surg. Am. 64 (1), 59—62.

Parvizi, J., Sharkey, P.F., Bissett, G.A., et al., Dec 2003. Surgical treatment of limb-length discrepancy following total hip arthroplasty. J. Bone Jt. Surg. Am. 85-A (12), 2310—2317.

Paulet, T., Fryer, G., 2009. Inter-examiner reliability of palpation for tissue texture abnormality in the thoracic paraspinal region. IJOM 1 (3), 92—96.

Poussa, M.S., Aug 2005. Anthropometric measurements and growth as predictors of low-back pain: a cohort study of children followed up from the age of 11 to 22 years. Eur. Spine J. 14 (6), 595—598.

Reeves, P.N., Cholewicki, J., Silfies, S.P., 2006. Muscle activation imbalance and low-back injury in varsity athletes. J. Electromyogr. Kinesiol. 16, 264—272.

Roberts, J.M., Wilson, K., 1999. Effect of stretching duration on active and passive range of motion in the lower extremity. Br. J. Sports Med. 33 (4), 259—263.

Roffey, D.M., et al., 2010. Causal assessment of awkward occupational postures and low back pain: results of a systematic review. Spine J. 10, 89—99.

Sahar, T., et al., Oct 2007. Insoles for prevention and treatment of back pain. Cochrane Database Syst. Rev. 17 (4), CD005275.

Savage, R.A., Whitehouse, G.H., Roberts, N., 1997. The relationship between the magnetic resonance imaging appearance of the lumbar spine and low back pain, age and occupation in males. Eur. Spine J. 6 (2), 106—114.

Seffinger, M.A., et al., Oct 1 2004. Reliability of spinal palpation for diagnosis of back and neck pain: a systematic review of the literature. Spine (Phila Pa.) 29 (19), E413—E425.

Soukka, A., Alaranta, H., Tallroth, K., Heliövaara, M., Apr 1991. Leg-length inequality in people of working age. The association between mild inequality and low-back pain is questionable. Spine (Phila Pa.) 16 (4), 429—431.

Suni, J., et al., Aug 15 2006. Control of the lumbar neutral zone decreases low back pain and improves self-evaluated work ability: a 12-month randomized controlled study. Spine (Phila Pa. 1976) 31 (18), E611—E620.

Taskaynatan, M.A., Izci, Y., Ozgul, A., et al., Apr 15 2005. Clinical significance of congenital lumbosacral malformations in young male population with prolonged low back pain. Spine (Phila Pa.) 30 (8), E210—E213.

Valdes, A.M., Hassett, G., Hart, D.J., Spector, T.D., 2005. Radiographic progression of lumbar spine disc degeneration is influenced by variation at inflammatory genes: a candidate SNP association study in the Chingford cohort. Spine 30, 2445—2451.

Van Nieuwenhuyse, A., et al., Jan 5 2009. Physical characteristics of the back are not predictive of low back pain in healthy workers: a prospective study. BMC Musculoskelet. Disord. 10, 2.

van Trijffel, E., Anderegg, Q., Bossuyt, P.M., Lucas, C., Nov 2005. Inter-examiner reliability of passive assessment of intervertebral motion in the cervical and lumbar spine: a systematic review. Man. Ther. 10 (4), 256—269.

van Tulder, M.W., Assendelft, W.J., Koes, B.W., Bouter, L.M., Feb 15 1997. Spinal radiographic findings and nonspecific low back pain. A systematic review of observational studies. Spine (Phila Pa.) 22 (4), 427—434.

Videman, T., et al., Mar 15 2006. Determinants of the progression in lumbar degeneration: a 5-year follow-up study of adult male monozygotic twins. Spine 31 (6), 671—678.

Videman, T., Levälahti, E., Battié, M.C., Jun 1 2007. The effects of anthropometrics, lifting strength, and physical activities in disc degeneration. Spine (Phila Pa.) 32 (13), 1406—1413.

Videman, T., Gibbons, L.E., Kaprio, J., Battié, M.C., Nov 2009. Challenging the cumulative injury model: positive effects of greater body mass on disc degeneration. Spine J. 17 [Epub ahead of print].

Waddell, G., Burton, A.K., Mar 2001. Occupational health guidelines for the management of low back pain at work: evidence review. Occup. Med. (Lond) 51 (2), 124—135 (Review).

Waddell, G., Burton, A.K., Kendall, N.A.S., 2008. Vocational Rehabilitation — what Works, for Whom, and when? (Report for the Vocational Rehabilitation Task Group). The Stationery Office, London. ISBN 0 11 7038615.

Wai, E.K., et al., 2010. Causal assessment of occupational bending or twisting and low back pain: results of a systematic review. Spine J. 10, 76–88.
Williams, P., Watt, P., Bicik, V., Goldspink, G., 1986. Effect of stretch combined with electrical stimulation on the type of sarcomeres produced at the end of muscle fibers. Exp. Neurol. 93, 500–509.
Willy, R.W., Kyle, B.A., Moore, S.A., Chleboun, G.S., 2001. Effect of cessation and resumption of static hamstring muscle stretching on joint range of motion. Orthop. Sports Phys. Ther. 31 (3), 138–144.
Yrjönen, T., Hoikka, V., Poussa, M., Osterman, K., Dec 1992. Leg-length inequality and low-back pain after Perthes' disease: a 28-47-year follow-up of 96 patients. J. Spinal Disord. 5 (4), 443–447.
Zazulak, B.T., Hewett, T.E., Reeves, N.P., Goldberg, B., Cholewicki, J., Jul 2007. Deficits in neuromuscular control of the trunk predict knee injury risk: a prospective biomechanical-epidemiologic study. Am. J. Sports Med. 35 (7), 1123–1130. Epub 2007 Apr 27.

Further Reading Full article

Lederman, E., 2010. The fall of the postural-structural-biomechanical model in manual and physical therapies: exemplified by lower back pain. CPDO Online J. http://cpdo.net/jour/jour1.html March 2010.
Lederman, E., 2005. The Science and Practice of Manual Therapy. Elsevier, Edinburgh.
Lederman, E., 2010. Neuromuscular Rehabilitation in Manual and Physical Therapies. Elsevier, Edinburgh.

Invited response

Gary Fryer, PhD, BSc(Osteopathy), ND [a,b,c]

[a] *School of Biomedical and Health Sciences, Victoria University, Melbourne, Australia*
[b] *Institute of Sport, Exercise and Active Living, Victoria University, Melbourne, Australia*
[c] *A.T. Still Research Institute, Kirksville, MO, USA*

The philosophical foundations of many manual therapy disciplines, including osteopathy, have been deeply rooted in the postural structural model (PSM) paradigm, which emphasizes the role of altered posture, anatomical structure, and biomechanics as a cause of pain and disturbed function. This paradigm has been reinforced by catch cries such as 'structure governs function' and by enduring concepts of misaligned or malpositioned joints. Lederman has highlighted the lack of scientific support for abnormal posture as a cause of low back pain and provided a well-reasoned argument that research has eroded many aspects and practices associated with PSM. While I applaud Lederman's critical approach to this model and agree that postural and biomechanical factors have been overemphasized in the past, I believe it would be a mistake to conclude that posture and biomechanical factors have no relevance in the assessment of patients with pain.

The causes of spinal pain are unclear, but pain is multifactorial, and an overemphasis of any one aspect, such as mechanical factors, is inappropriate. Lederman correctly draws attention to the literature that shows most diagnostic findings associated with PSM cannot be used to differentiate individuals with pain from those without pain or to predict whether individuals are more likely to have pain. This literature does not, however, demonstrate that postural factors have no influence on pain. Lederman states that the most important question is consistently being ignored: 'can a person's physical shape/posture/structure/biomechanics be the cause of their lower back pain?' The cause of back pain is multi-factorial and may involve genetic, biopsychosocial, and environmental factors. The key questions, therefore, are not whether physical shape or posture can cause lower back pain, but whether these factors influence or contribute to back pain and, if so, whether practitioners can identify and treat these factors to influence the health of patients. The studies reviewed by Lederman typically examine the influence of a single PSM factor (e.g., pelvic asymmetry), without accounting for other PSM factors or other multidimensional aspects of pain. Comparison of posture between individuals may not be meaningful because posture is highly variable and idiosyncratic, but exaggeration of an individual's postural pattern may be more clinically relevant, as may treatment aimed at minimizing deviations. For example, examination of head carriage in a group of office workers may not reveal those individuals with pain, but exaggeration of head forward posture in a worker with recurrent pain will likely aggravate the pain, whereas attention to better posture alleviates it.

Although direct evidence for the association of posture with low back pain is lacking, there is a strong theoretical rationale of why posture may influence pain. Mechanical loading on ligaments, either sustained or cyclic, causes viscoelastic change, hysteresis, strain, inflammation, and abnormal motor activity (Solomonow, 2006). Thus,

E-mail address: gary.fryer@vu.edu.au.

abnormal load on ligaments and other structures may occur due to chronic asymmetrical or suboptimal posture, and may contribute to exhausting an individual's adaptive reserve, which results in strain and pain.

Evidence also suggests that asymmetrical or suboptimal posture may affect function. Increases in thoracic kyphosis are associated with higher multisegmental spinal loads and trunk muscle forces in upright stance (Briggs et al., 2007). Although there is considerable controversy about assessment of the sacroiliac joints, asymmetrical sacroiliac motion may be predictive for pelvic pain (Buyruk et al., 1995; Buyruk et al., 1999; Damen, Stijnen et al., 2002; Damen, Buyruk et al., 2002). Additionally, subtle pelvic torsion has been reported to cause asymmetrical loading on the lumbar and thoracic tissues (Al-Eisa, Egan et al., 2006a; 2006b).

Pain clearly affects movement. Subjects with low back pain have been reported to have greater asymmetry between right and left paraspinal muscle contraction (Oddsson and De Luca, 2003) and altered activation during trunk flexion and re-extension. Pain affects proprioception and the precision of the control of movement (Gill and Callaghan, 1998; Taimela et al., 1999; Leinonen et al., 2002; Grip et al., 2007; Lee et al., 2007). Although these motion abnormalities are consequences of pain rather than causes, motion and postural control are assessed as part of PSM and are potentially useful to guide management and assess the success of treatment.

The influence of suboptimal posture on pain is more clearly demonstrated for the head and neck. Several studies support these clinical observations and demonstrate associations with altered cervical posture and neck pain. Yip et al. (Yip et al., 2008) reported that subjects with neck pain demonstrated greater forward head posture (CV angle) than those without pain and that the greater the forward head posture the greater the disability. Lau et al. (Lau, Cheung et al., 2010) found the upper thoracic angle was a good predictor for presence of neck pain, even better than the CV angle. Other researchers have also reported greater cervical lordosis in neck pain patients. Maintaining a neutral head posture and avoiding a forward head position is advantageous in reducing sustained upper and lower trapezius activity (Weon et al., 2010). Furthermore, structural and biomechanical factors, such as knee valgus motion, knee flexion (Myer, Ford et al., 2010a; 2010b), altered joint kinematics, and postural stability (Paterno et al., 2010), have been useful in predicting athletes at risk of anterior cruciate ligament injury.

I believe a broad interpretation of PSM assessment, including observation of global posture (during standing and in occupational tasks), range of motion, and movement patterns, provides useful information to the clinician. As Lederman highlights, the lack of reliability and validity of many of our diagnostic palpatory tests should concern practitioners who use this therapeutic paradigm to detect minute differences in asymmetry of landmarks or motion of spinal segments. However, the clinical usefulness of identifying minor asymmetry of pelvic and spinal landmarks is highly dubious, may lead to erroneous beliefs by therapists and patients of 'bones out of place', and may contribute to fear avoidance behavior. Assessment of global posture (during standing and in occupational tasks), functional movement, muscle strength, and palpation for tenderness are likely to have clinical utility, and although these factors may be consequences of pain and injury rather than causes, they are biomechanical findings that may help guide management. However, any physical findings must be placed in context with clinical history and a biopsychosocial approach. There is no question that the importance of PSM factors have been overstated by sub-groups of manual therapists in the past and consideration of the multidimensional nature of pain is essential for holistic, patient-centered care.

References

Al-Eisa, E., Egan, D., et al., 2006a. Effects of pelvic asymmetry and low back pain on trunk kinematics during sitting: a comparison with standing. Spine 31 (5), E135—E143.

Al-Eisa, E., Egan, D., et al., 2006b. Effects of pelvic skeletal asymmetry on trunk movement: three-dimensional analysis in healthy individuals versus patients with mechanical low back pain. Spine 31 (3), E71—E79.

Briggs, A.M., van Dieen, J.H., et al., 2007. Thoracic kyphosis affects spinal loads and trunk muscle force. Phys. Ther. 87 (5), 595—607.

Buyruk, H.M., Snijders, C.J., et al., 1995. The measurements of sacroiliac joint stiffness with colour Doppler imaging: a study on healthy subjects. Eur. J. Radiol. 21 (2), 117—121.

Buyruk, H.M., Stam, H.J., et al., 1999. Measurement of sacroiliac joint stiffness in peripartum pelvic pain patients with Doppler imaging of vibrations (DIV). Eur. J. Obstet. Gynecol. Reprod. Biol. 83 (2), 159—163.

Damen, L., Stijnen, T., et al., 2002. Reliability of sacroiliac joint laxity measurement with Doppler imaging of vibrations. Ultrasound Med. Biol. 28 (4), 407—414.

Damen, L.M., Buyruk, H.M.P., et al., 2002. The Prognostic value of Asymmetric laxity of the sacroiliac joints in pregnancy-related pelvic pain. Spine 27 (24), 2820—2824.

Gill, K.P., Callaghan, M.J., 1998. The measurement of lumbar proprioception in individuals with and without low back pain. Spine 23 (3), 371—377.

Grip, H., Sundelin, G., et al., 2007. Variations in the axis of motion during head repositioning - A comparison of subjects with whiplash-associated disorders or non-specific neck pain and healthy controls. Clin. Biomech. doi: 10.1016/j.clinbiomech.2007.05.008.

Lau, K.T., Cheung, K.Y., et al., 2010. Relationships between sagittal postures of thoracic and cervical spine, presence of neck pain, neck pain severity and disability. Man. Ther. 15 (5), 457—462.

Lee, H.Y., Wang, J.D., et al., 2007. Association between cervicocephalic kinesthetic sensibility and frequency of subclinical neck pain. Man. Ther. doi: 10.1016/j.math.2007.04.001.

Leinonen, V., Maatta, S., et al., 2002. Impaired lumbar movement perception in association with postural stability and motor- and somatosensory- evoked potentials in lumbar spinal stenosis. Spine 27 (9), 975—983.

Myer, G.D., Ford, K.R., et al., 2010a. New method to identify athletes at high risk of ACL injury using clinic-based measurements and freeware computer analysis. Br. J. Sports Med.

Myer, G.D., Ford, K.R., et al., 2010b. Development and validation of a clinic-based prediction tool to identify female athletes at high risk for anterior cruciate ligament injury. Am. J. Sports Med. 38 (10), 2025–2033.
Oddsson, L.I.E., De Luca, C.J., 2003. Activation imbalances in lumbar spine muscles in the presence of chronic low back pain. J. Appl. Physiol. 94, 1410–1420.
Paterno, M.V., Schmitt, L.C., et al., 2010. Biomechanical measures during landing and postural stability predict second anterior cruciate ligament injury after anterior cruciate ligament reconstruction and return to sport. Am. J. Sports Med. 38 (10), 1968–1978.
Solomonow, M., 2006. Sensory – Motor control of ligaments and associated neuromuscular disorders. J. Electromyogr. Kinesiol. 16 (6), 549–567.
Taimela, S., Kankaanpaa, M., et al., 1999. The effect of lumbar fatigue on the ability to sense a change in lumbar position. Spine 24 (13), 1322–1327.
Weon, J.H., Oh, J.S., et al., 2010. Influence of forward head posture on scapular upward rotators during isometric shoulder flexion. J. Bodyw Mov Ther. 14 (4), 367–374.
Yip, C.H., Chiu, T.T., et al., 2008. The relationship between head posture and severity and disability of patients with neck pain. Man. Ther. 13 (2), 148–154.

Invited response: (Without science, there must be art)

John C. Hannon, DC *

1141 Pacific Street, Suite B, San Luis Obispo, CA 93401, USA

KEYWORDS
Lederman;
Lower back pain;
Asymmetry;
Posture;
Manual therapy;
Relaxation;
Predictability;
Movement excellence

Summary In the target article, Lederman reminds us we do not know the causes of back pain. Further, we cannot count on perceived asymmetry, biomechanical dysfunction and muscle imbalance to guide treatment.

Early osteopaths and chiropractors believed where there is no science, there must be fervor. This reviewer suggests where there is no science, there must be art. We can train ourselves, as Professor Lederman has done, to read widely, think deeply and debate well. We can open communication between scientists and clinicians. And, we also can find inspiration in artistry.

Artists spend decades discovering how to steer their craft. An early student of movement control, Nicholai Bernstein showed that expert blacksmiths have neither intra-operator nor inter-operator repeatability. They never swing their arms exactly the same yet every expert predictably produces the desired outcome.

This paper encourages learning from the artistry of expert massage therapists. Instead of a narrow focus detecting diagnoses and dysfunction, they have a softer and wider focus. They create a comfortable ambiance and they tend to spend more time with their clients.

In addition to learning from bodyworkers and movement therapists, cultivation of the arts may also deepen empathy, communication skills and personal conviction in ways that wordlessly help patients find our work trustworthy.

Introduction

For more than 23 years Professor Lederman has spoken in a strong and clear voice about issues at the core of rehabilitation. He can be counted on to ask searching questions that derive from a deep concern with the quality of physical examination and manual therapy. In the target article, Lederman (2010) speaks out again, this time about what he sees as three hurdles to effective manual therapy.

First, he notes the impossibility of defining the causes of back pain. Humans are resourceful and work around asymmetries and imbalances. Lederman reminds us that perceived asymmetry (be it postural, structural or biomechanical) cannot be counted on to guide treatment. He cautions us that muscle imbalance (be it strength, length or endurance) also is an untrustworthy guide. Therefore, what he calls the 'postural-structural-

* Tel.: +1 805 542 9925; fax: +1 805 541 2391.
E-mail address: Jhannon@digitalputty.com.

biomechanical model' (PSB) is bound to be erratic and ineffective in guiding treatment. His second hurdle is the lack of reliability in the assessment tools used in manual therapy. Lastly, he notes that remodeling aberrant muscles and connective tissues is too time-intensive to expect good compliance.

It is true, manual therapy is in a dreadful mess. It always has been and always will be. That is one of its splendors. Its complexity prevents convenient packaging; its metaphors change with time to accommodate the latest assumptions (and sometimes even scientific findings). Perhaps the urge to classify; to economize the intellect, produced the chimeric[1] aberrations spotlighted by his paper. In any case, we have Lederman to thank for his collection of articles[2] that deride the idea of simple causation.

Before addressing his concerns, consider another example of muddle-headed health care masquerading as science; one where even vast investments of money, prestige and power have proved useless: America's unsuccessful 35 years fighting the War on Cancer[3]. Even with annual spending of up to 20 billion dollars on research (and 200 billion dollars on treatment) cancer incidence in the USA leads the world and American cancer death rates refuse to drop.

Despite these staggering costs in treasure and misery, organized medicine refuses to shift its focus to cancer prevention through tobacco abstinence, dietary improvements and environmental remedies.

Pain and psychology

Wall (1977) discusses the emotions that surround pain[4] (such as fear, anxiety, dislike and urgency). This paper echoes Wall's wish to move away from merely studying pain stimuli, instead, we gain advantage by learning to recognize (and calm) the reactive states triggered by pain. Unmentioned in Lederman is how to address these tense body reactions although psychology[5] is mentioned (in two sentences): "[B]eing a human … means that the structure is within the awareness. It is also under the influence of our emotions as well as the will."

3 straw men and the fall of PSB?

Lederman's title states a fall is coming. But was a single model ever raised up? Consider the three straw man fallacies found in the target paper. Contrary to his title, no model is shared by manual therapists. Lederman notes PSB is a 'basic belief' and it is 'frequently used in clinic' but without citations backing up this claim. It is more likely that practitioners, at different points in their careers, believe in some aspects of what Lederman presents as the PSB model.

Similarly, no one is identified who believes Lederman's portrayal of PSB's mechanical model as the "utopian view of the body [working] in perfect precision/synchrony." Lastly, he oversimplifies; for instance, he manhandles the vast field of clinical management into a tiny and impossible dyad where lower back problems are either too severe to treat or too minor to matter.

[1] Chimeric in that a false tidiness was created by those manual therapists who strove to yoke the concepts (and tests) of two different species: bone-cutting orthopedists and the vitalists of yore.

[2] In support of his claim of the absence of scientific support he cites (but does not summarize) about 60 articles and systematic reviews. This reviewer is not a scientist, but this seems too meager a serving for so important a topic.

[3] The research, diagnosis and treatment of cancer and low back pain share characteristics that Professor Lederman describes. Each demonstrates muddle-headed thinking, prejudicially rigid behaviors and vested interests. Aggarwal et al., (2009) explain the "War on Cancer" was declared by President Nixon, in 1974, as an attempt to make the wealthiest country into the healthiest country. In the last 20 years, 60,000 compounds have been tested against 60 human cancer cell lines without a single effective anticancer candidate identified. Many cell-lines are cross-contaminated and the cell-lines spontaneously mutate over time. But, despite these confounding factors, note that cell-line research forms the basis of 75% of all cancer research publications. It is not surprising that these studies are poor indicators of efficacy in humans. Making things more muddle-headed is the absence of any animal model that is predictive of cancer. Nor are there any biomarkers reliable enough to assess cancer risk or to assess treatment. Why are rational scientists, despite their access to prestige, funding, and sophisticated scientific tools, unwilling to move forward and stake out new territory? Perhaps it is an aggressive refusal to consider what Nabokov (1983) calls the "most dread enemy of the visionary: the snake of doubt, the coiled consciousness that his quest is an illusion".

[4] Wall notes that "I have never sensed a pure pain… If I sense a pain, it comes in a packet with such changes as fear, loathing, anxiety, dislike, urgency, etc. … There is a second peculiarity of pain which differentiates it from most of our sensory experience. Pain is given. Most of our sensory experience is taken.' And, on p. 366 he states: "A great deal of the body's machinery is involved in maintaining a stable internal environment. If this shifts from its normal range, a series of more and more complex and elaborate reactions are triggered. These states of awareness have many properties in common. One is urgency. … So great is the urge to do something that these states have been associated each with its drive and we would add to the commonly accepted drives to eat and drink, a drive to preserve the body intact. … Pain is associated with the turning on of the drive for reaction. The nature of the reaction will depend on a different set of circumstances. This shifts pain away from the stage of event detection towards a stage of reaction decision."

[5] Psychology is also mentioned on p.10 of the target article. But notice a trend in his books. Over time there has been a whittling away of his discussion of the psychological side of manual therapy. Psychology shared in the book's title and fully one third of the 1997 book. The 2005 edition retains a section entitled Psychology and Psychophysiological Processes but the section shrinks to about 20% of the text. The 2010 edition's table of contents no longer highlights psychology although the subject still crops up in the text. The cpdo.net website (home to Lederman's online journal which published the target article) lists Tsafi Lederman (UKCP registered psychotherapist) as co-author of the Lederman 2005 psychology section. The 2005 preface states: "I would like to acknowledge Tsafi Lederman's special contribution to Section 3 of both editions [2005 and 1997] of the book."

As refreshing as it is to see straw men swept away with energy and vigor, that energy would have been better used in the describing his Process Approach model. Similarly, more clarification is needed to understand lower back pain through Lederman's biological rather than mechanical lens.

The value of art in manual therapy

Current perceptions of manual therapy are not always elevated. Recent survey articles (Duffy, 2010; Hulen, 2008) consign manual therapy (with minimal commentary) to a nondescript grab-bag of complementary and alternative therapies. But, this is squabbling between competing interests since both allopathic medicine and manual therapy (physical therapy, osteopathy and chiropractic) share heroic assumptions. They often take on disease and shoot for a cure. But consider the other form of manual therapy that is either too humble (or too smart) to join the fracas: massage.

Interestingly, preference for massage (mostly paid out-of-pocket rather than by insurance) sometimes beats out manipulation[6]. Massage therapy training builds competency based upon not only science[7] but also art. Perhaps it is time for some chiropractors, osteopaths and physical therapists to take lessons from massage therapists[8].

Consider the typical massage table, it is well padded, wide and often heated; all features designed to encourage relaxation and a feeling of security. The massage therapist, freed from the responsibility of diagnosis, has the luxury of being solicitous of the client's comfort. Another luxury often enjoyed by massage therapists is a longer treatment time. Much can happen in an hour, particularly for those who find it difficult to relax.

The most important asset the best massage therapists have, in this reviewer's opinion, is their ability to sense what is right about their clients. Diagnosis-driven manual therapy tends to focus on dysfunction/disorder/disease. Instead of finding fault, we can also look for optimal function contributing to movement excellence. Using this as our starting point, perhaps we can amplify, and distribute this ease and coordinated function into other regions of the body.

Huygens and Goldstein

Meijer (2001) tells the story of Charles V. Almost 450 years ago, this inveterate clock collector, wanted his many clocks to synchronize while striking the hours. This problem vexed him to the point of springing him out of bed to adjust the laggards. Sadly for Charles, it was a century later when Huygens found a solution. He suspended clocks from a metal bar. Suspension permitted gravity to orient each clock in concert while the resonance of the metal bar entrained their rhythms.

Huygens found an esthetic solution to an engineering problem. It is possible that a similar artistic solution may aid earnest manual therapists, dizzied by Lederman's divergent findings.

Goldstein (1939/1995) describes a central dilemma in biology; that is, life must be taken apart to be studied scientifically and this process has generated "a multitude of isolated facts … but [w]hat do the phenomena, arising out of the isolating procedure, teach us about the essence (the intrinsic nature) of an organism? How, from such phenomena, do we come to an understanding of the behavior of the individual organism?"

The internal arts

Lederman echoes Goldstein. One way out of this quandary is to devote a career to understanding movement excellence, the better to recognize and encourage it in our patients. Patients trust our skills, knowledge and intuition when they believe we are doing our best for them. Empathetic[9] conversation, as well as touch, helps patients to trust.

[6] The following studies suggest that, for some, massage has a more elevated status than manual therapy: 1/. Chenot et al., 2007 studied Germans with low back pain and found the prevalence of these modalities (descending order): local heat (34%), massage (31%), spinal manipulation (26%). 2/. Fleming et al., 2007 in a study of 908 chronic pain patients receiving opioids as a primary treatment (lower back pain was the largest group). Massage therapy was used by (27%), followed by chiropractic treatment (18%), acupuncture (7%), and yoga (6%). 3/. Wang et al., 2005, in a survey of pregnant women, listed acceptance in descending order: massage (61.4%) compared to acupuncture (44.6%), relaxation (42.6%), yoga (40.6%), and chiropractic (36.6%). 4/. Carneiro and Rittenberg 2010 considered the role of exercise and alternative treatments for lower back pain and endorsed yoga and acupuncture rather than manual therapy. 5/. Wang et al., 2003, in a follow-up survey (1235 respondents) of the use of complementary and alternative treatments by surgical patients at Yale-New Haven hospital, found the most prevalent treatment was self-prayer followed by relaxation techniques, herbal medicine, massage/reflexology followed by chiropractic. 6/. Nichols and Harrigan 2006 found athletic usage of massage 25% higher than manual therapy. 7/. Rawsthorne et al., 1999, surveyed alternative medicine use by patients with inflammatory bowel disease at clinics in Winnipeg, Cork, Stockholm and Los Angeles. They found that half the patients ($n = 289$) used some form of alternative medicine. In descending order: exercise (28%), prayer (18%), counseling (13%), massage (11%), chiropractic (11%), and relaxation (10%).

[7] Some might say this science is merely descriptive as in gross anatomy and basic biomechanics but, massage therapists, bodyworkers and movement therapists have joined the collaborative table as evidenced by their attendance at the recent International Fascia congresses and their authorship (and readership) of this Journal.

[8] Lederman 1997 suggested: "Manual therapists are encouraged to use all manual techniques, even those which are traditionally outside their own discipline: the most effective techniques can be used to facilitate the patient's health…"

[9] Lewit 2010 notes "The locomotor system … is the largest and most intricate system in the human body. … [describing current therapists] we have learned to utilize increasingly complicated equipment while at the same time neglecting the evidence of our eyes and in particular of our hands, as well as forgetting to communicate with our patients. However, by comparison with communicating with the patient, observing with our eyes and sensing with our hands, no piece of high-tech equipment is able to yield such a wealth of varied information. All this is heightened by the capacity for feedback—indeed empathy—between practitioner and patient."

This contemplates that we choose to consciously mature our qualities of patience, sincerity, honesty and serenity. We ourselves may need to deepen these qualities to recognize excellence of movement, particularly of the minuscule respiratory excursions and the diminutive postural releases that accompany the act of relaxation[10]. We can learn from Huygens whose innovation bridged esthetics and engineering.

Plumbing profound applications from basic principles also takes time. This reviewer believes that learning how to observe, palpate and respond to the patient's varying levels of relaxation is a fruitful use of time. This inward focus may balance the need to stay current with research findings; Donald Knuth, the computer science pioneer, explains on his website why he does not respond to (most) emails. He says that unlike most people, he does not want to stay on top of things; instead, he wants to get to the bottom of things.

An additional way to get to the bottom of things may be cultivation of the arts. Not only by watching dance, martial arts and Olympic sporting events in slow-motion but also by reading sufficient poetry, music or drama[11] to be deeply moved by art. The point is to develop empathy and increase our points of view by cherishing art.

There are scientists and clinicians who, like Professor Lederman, teach, treat and train. We can learn from these people. For instance, Lee et al. (2008) help women with pregnancy-related pelvic girdle pain. Without compromising continence or respiration, they accomplish effective lumbopelvic transfer of loads by maintaining optimal joint axes and sufficient intra-abdominal pressure.

Also interested in force transmission are Brown and McGill (2010) who compared ultrasound and electromyography findings in the examination of abdominal wall contraction. They suggest that the composite laminate-like organization of the wall may affect force transmission. They too merge their findings and those of other researchers into therapeutic approaches.

But we should also develop the art of encouraging patients to reach beyond passivity. Salmonsen et al. (2010) make a distinction between patients as passive recipients of treatment and those that choose to become explorers. They note that such explorers "have claims to knowledge and actions in which science does not have a privileged place". Instead, explorers scout for techniques and concepts that are meaningful in their everyday life.

Conclusion

Like an unkempt bazaar where idealists and freebooters rub shoulders and caveat emptor reigns, manual therapy is a blooming confusion where the earnest either wrangle their patients within paradigmatic constraints or forge new beginnings from the broken remnants of old exemplars.

This reviewer believes the healing arts reach their potential when practitioners find passion and devotion in being of service. Empathy and communication are needed to inspire those we serve and this takes time. We need to move beyond passive care and to harness curiosity, will and imagination. A scientific background may be essential but, when causation is confused and reliability nonexistent, the foundations of healing must be based on art.

Competing interests

This reviewer has no competing interests.

References

Aggarwal, B.B., Danda, D., Gupta, S., Gehlot, P., 2009. Models for prevention and treatment of cancer: problems vs promises. Biochem. Pharmacol. 78 (9), 1083—1094.

Brown, S.H.M., McGill, S.M., 2010. A comparison of ultrasound and electromyography measures of force and activation to examine the mechanics of abdominal wall contraction. Clin. Biomech. (Bristol, Avon) 25 (2), 115—123.

Carneiro, K.A., Rittenberg, J.D., 2010. The role of exercise and alternative treatments for low back pain. Phys. Med. Rehabil. Clin. N. Am. 21 (4), 777—792.

Chenot, J.F., Becker, A., Leonhardt, C., Keller, S., Donner-Banzhoff, N., Baum, E., Pfingsten, M., Hildebrandt, J., Basler, H.D., Kochen, M.M., 2007. Use of complementary alternative medicine for low back pain consulting in general practice: a cohort study. BMC Complement. Altern. Med. 7, 42. doi:10.1186/1472-6882-7-42.

Duffy, R.L., 2010. Low back pain: an approach to diagnosis and management. Prim. Care Clin. Off. Pract. 37, 729—741.

Fleming, S., Rabago, D.P., Mundt, M.P., Fleming, M.F., 2007. CAM therapies among primary care patients using opioid for chronic pain. BMC Complement. Altern. Med. 7, 15. doi:10.1186/1472-6882-7-15.

Goldstein, K., 1939/1995. The Organism: A Holistic Approach to Biology Derived from Pathological Data in Man. Zone Books. MIT Press, Cambridge, p. 27.

Hannon, J.C., 2006. Wartenberg Part 3: relaxation training, centration and skeletal opposition: a conceptual model. J. Bodywork Move. Therapies 10, 179—196.

Hulen, C.A., 2008. Nonoperative treatment of low back pain. Semin. Spine Surg. 20 (2), 102—112.

Lederman, E., 1997. Fundamentals of Manual Therapy: Physiology, Neurology and Psychology. Churchill Livingstone, New York, p. viii.

Lederman, E., 2005. The Science and Practice of Manual Therapy. Elsevier, Edinburgh, p. xiv.

Lederman, E., 2010. The fall of the postural-structural-biomechanical model in manual and physical therapies: exemplified by lower back pain. CPDO Online J., 1—14. www.cpdo.net.

Lee, D.G., Lee, L.J., McLaughlin, L., 2008. Stability, continence and breathing: the role of fascia following pregnancy and delivery. J. Bodywork Move. Therapies 12 (4), 333—348.

Lewit, K., 2010. Manipulative Therapy. Elsevier, Edinburgh. p. 380.

Meijer, O.G., 2001. Making things happen: an introduction to the history of movement science. In: Latash, M.L., Zatsiorsky, V.M. (Eds.), 2001 Classics in Movement Science. Human Kinetics, Champaign, pp. 1—57.

Nabokov, V., 1983. In: Bowers, F. (Ed.), Lectures on Don Quixote. Harcourt Brace Jovanovich, San Diego, p. 68.

Nichols, A.W., Harrigan, R., 2006. Complementary and alternative medicine usage by intercollegiate athletes. Clin. J. Sport. Med. 16 (3), 232—237.

[10] One exploration of these distinctions is described in Hannon (2006).

[11] Please substitute whatever forms of art fit.

Rawsthorne, P., Shanahan, F., Cronin, N.C., Anton, P.A., Lofberg, R., Bohman, L., Bernstein, C.N., 1999. An international survey of the use and attitudes regarding alternative medicine by patients with inflammatory bowel disease. Am. J. Gastroenterol. 94 (5), 1298—1303.

Salamonsen, A., Launso, L., Kruse, T.E., Eriksen, S.H., 2010. Understanding unexpected courses of multiple sclerosis among patients using complementary and alternative medicine: a travel from recipient to explorer. Int. J. Qual. Stud. Health Well-being 5, 5032. doi:10.3402/qhw.v5i2.5032.

Wall, P.D., 1977. Why do we not understand pain? In: Duncan, R., Weston-Smith (Eds.), The Encyclopaedia of Ignorance. Pocket Books, New York, pp. 363, 366.

Wang, S.M., Caldwell-Andrews, A.A., Kain, Z.N., 2003. The use of complementary and alternative medicines by surgical patients: a follow-up survey study. Anesth. Analg 97 (4), 1010—1015.

Wang, S.M., DeZinno, P., Fermo, L., William, K., Caldwell-Andrews, A.A., Bravemen, F., Kain, Z.N., 2005. Complementary and alternative medicine for low-back pain in pregnancy: a cross-sectional survey. J. Altern. Complement. Med. 11 (3), 459—464.

Invited response (The postural structural model, with boundary conditions)

Robert Edwin Irvin, DO *

Department of Osteopathic Manipulative Medicine, Oklahoma State University Health Science Center, 1111 W. 17th St., Tulsa, OK 74107, USA

Introduction

I do not rebut Dr. Lederman's conclusions from the clinical science of posture (Lederman, 2010), but rather the contemporary science and therapeutics of posture itself.

Research within the last 30 years of the predictive value of postural imbalance for back pain yields results interpreted by several investigators (Grundy and Roberts, 1984, and Dieck et al., 1985), as evidence that posture is, at best, a weak player in the etiology of such pain. Grundy and Roberts found that disparity of the lower limbs is not a significant predictor for the history of low back pain. Dieck found that postural asymmetries in the coronal plane, after 25 years, had no predictive value for back or neck pain. Fann (2002) found that the history of low back pain does not correlate with the amount of pelvic unlevelness. Interpretation of these findings as being evidence of absent or weak causality of postural imbalance for pain is overly narrow for reason that while postural imbalance, an ubiquitous finding, is *not predictive* of pain (an example of observational causality: See A and you routinely see B) (Rapoport, 1954), clinical research evidences (Irvin, 1986, Hoffman, 1994, Irvin, 1998, Lipton et al., 2009) that sufficient *reduction* of postural imbalance *is strongly predictive* of enduring *reduction or alleviation* of musculoskeletal pain throughout the body, where no primary biologic disease of the musculoskeletal system otherwise exists (an example of manipulable causality: Change A and B routinely changes).

The preponderance of studies with outcomes of therapeutic strategies aimed to reduce the tissue changes associated with postural imbalance show weak or no benefit because of several problems in what is presupposed about the causality of postural imbalance for pain. Experimental design and the postural therapeutics that ensue from these presuppositions are then similarly weakened and are mistaken as evidence of lack of determination between postural imbalance and pain.

One problem is that postural imbalance as etiology of pathomechanical disease is more complex than is presumed by the dyad of cause and effect. Fundamentally at play is postural imbalance as origin of most mechanical stress throughout the body, in contrast to cause in terms of tissue changes that that are proximate to the effect.

How is postural imbalance the origin of most mechanical stress? Except for astronauts, those in air flight, and aquanauts, most mechanical activity occur on the ground (and also in the field of gravity). The shape, attitude and activity of the body with respect to gravitation (posture), and ground support are the initial and ongoing conditions from which all mechanical actions proceed. Logically, posture, and its corollary, imbalanced posture, is the origin of most human mechanical actions, and thereby of much, even most, mechanically mediated disease.

For which origin, experimental design and the therapeutic methods to be tested must be informed of the postural systematic (see Figure 1) (Irvin, 1998, Irvin, 2007) if they are to be a valid test of postural imbalance as it relates to pathomechanical disease. The postural systematic as origin is comprised of multiple and interacting factors which cannot be reduced to a single cause.

* 6620 Bryant Irvin Road, Suite 100, Fort Worth, Texas 76132, USA. Tel.: +1 817 346 6656.

E-mail address: roberti@airmail.net.

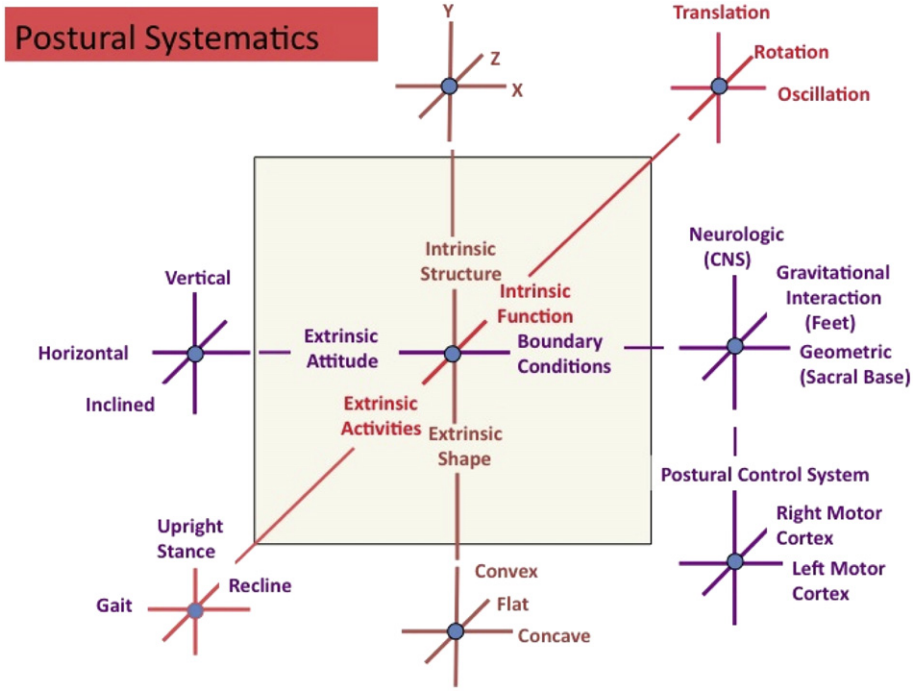

Figure 1 A schematic of the essential constituents of the postural systematic that extends from a common origin that represents their operational linkage and which is comprised of 6 essential characters for human posture: intrinsic structures, intrinsic functions, boundary conditions, extrinsic attitude, extrinsic activity, and extrinsic shape.

Central to the postural systematic is a composite of six natures in their environs of gravitation and ground support.

1. Extrinsic shape, in terms of convex, concave, and flat.
2. Extrinsic attitude, in terms of vertical, inclined, and horizontal.
3. Extrinsic activity, in terms of gait, upright stance (standing or seated) and recline.
4. Intrinsic structures, with respect to the 3 spatial freedoms, x, y, and z.
5. Intrinsic functions, with respect to the 3 freedoms that are translation, rotation, and oscillation.

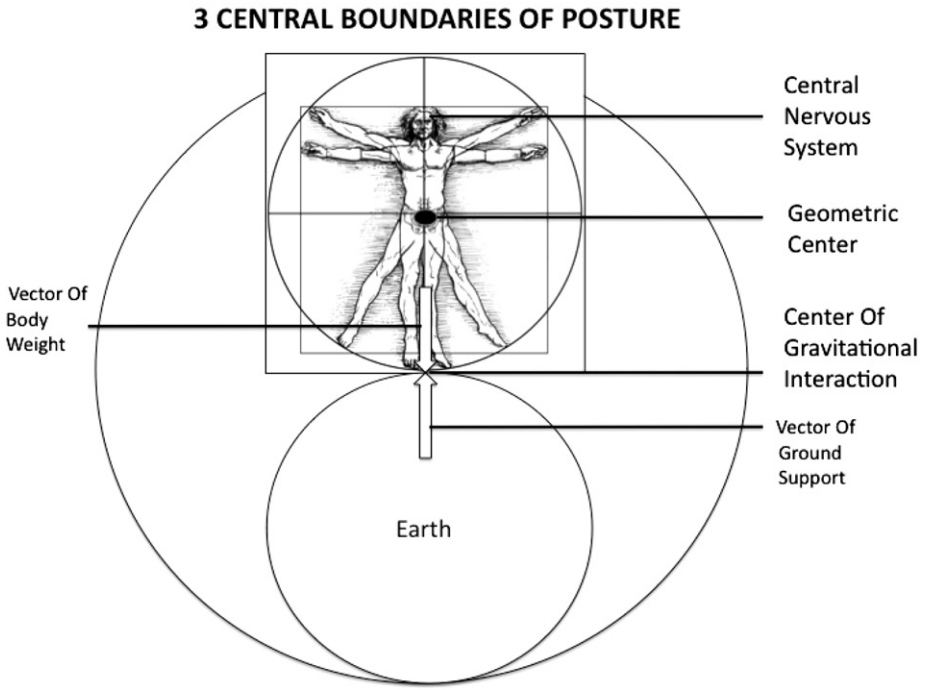

Figure 2 An illustration of the 3 central boundaries of posture: the feet, the sacral base, and the CNS; all having ground support in the field of gravity.

6. Boundary conditions, which are initial and ongoing, and which limit and regulate overall postural array, these boundaries being the feet, the sacral base, and the CNS (see Figure 2).

The pivotal character of the feet, sacral base and CNS as boundary conditions for overall posture is a function of these structures being central to each of 3 sub-systems that comprise the postural system. By virtue of this operational centrality for postural systematics, each boundary is linked directly to all aspects of the musculoskeletal system. These 3 central postural boundaries are the feet, comprised of 3 arches, the sacrum with 3 articular surfaces, and the CNS, comprised of the right and left motor cortices and the postural control system within the brainstem which regulates postural array via motor control.

1) The feet are central to the equal and opposing vectors of body weight and ground support.
2) The sacral base is approximately geometrically central to the outstretched frame.
3) The postural control system in the brainstem of the central nervous system interacts with the entirety of posture via motor control, to affect the most economic stance, activity and shape, and thereby to minimize mechanical stress

An immediate therapeutic advantage from awareness of this postural schematic is that each aspect of the postural system is operationally linked with all other aspects. This linkage provides potential for its coherent manipulation towards an improved posture. An additional advantage is from recognition and according treatment of the boundary (initial and ongoing) conditions for posture, and thereby routinely achieves broad and enduring correction of postural imbalance. This concerted method for treatment of mechanically mediated disease is in contrast to a causal strategy aimed primarily to treat tissue changes proximate to the respective effect, and without synchronous correction of the boundary conditions that mediate postural imbalance.

Where causality is assumed to be the sole determinative mode, a number of difficulties in experimental design occur, which difficulties disappear in the context of origin. For instance, the tissue changes associated with imbalanced posture, and which are emphasized in contemporary treatment, are intermediate between the imbalanced posture and the effect that is pain. Hence, treatment of these changes is not directly corrective of the core postural imbalance, but instead reduces the accumulated disorder that can proceed from postural imbalance. While sufficient reduction of the disordered load can affect pain in individual cases, this benefit is less certain in a representative population because the origin (postural imbalance) is still in play.

A second difficulty, and a misstep, is to reduce the frame of reference for causality to a cause that is physically proximate (contiguous) to effect. Origin can yield the effect of pain, with or without intermediate processes, (e.g. tissue changes proximate to pain) a fact complicating experimental design. For instance, does manipulation of the low back enduringly reduce low back pain? The preponderance of previous studies conclude not. By not recognizing that postural imbalance is a pan-systemic disorder, being neither proximate to nor remote from effect, this partial view does not take into account that the effects on tissues are typically not for a single region, but instead are multi-regional. Treatment aimed to modify a local manifestation of postural imbalance, such as tissue restriction or skeletal asymmetry proximate to where there is pain, is susceptible to failure as the pan-systemic imbalance is an ongoing pressure towards the continuance of a given effect.

Thirdly, the network of disordered tissues throughout the body constitutes a load of disorder, which is resistant to change from treatment of a single region. Within which network, focal or local treatment is an inadequately small frame of reference for the embedded focus of pain.

A fourth problem, and one for experimental design, is that, practically, rather than treating one region for one pain, operators that provide manual or physical manipulation more commonly employ complex, pan corporeal therapeutics directed to counter multi-regional tissue disorder associated with postural imbalance. There is poor inter-operator reproducibility and uniformity for this complex therapeutics. Further, such therapeutics are tailored to each individual case, and thereby cannot practically follow a protocol for uniformity of treatment of multiple subjects, albeit with similar symptoms. This complexity is a nightmare for those who would apply the austere model of causality that prefers a singular therapeutic variable in order to test a singular and hypothetical causality.

If the operator is informed of this postural systematic and accordingly corrects the boundary conditions for posture, implementation of a pelvic lift has little complexity. Taking an imprint of the feet from which the orthotics are crafted routinely individualizes custom foot orthotics. The chief objective for additional physical and manual manipulation is to reduce the accumulated load of tissue disorder, and thereby decrease resistance to postural symmetrization as the postural boundaries are corrected. Thereby, operators can coherently reduce from many directions the pan-systemic load of tissue disorder, and by whatever accepted therapeutic modes available, even as the boundary conditions are being corrected.

A fifth problem is that prior studies of the effects either of foot orthotics or pelvic leveling alone neglects the contribution of the boundary not corrected, which can obscure the benefits from this partial correction. Nonetheless, where the pelvic obliquity alone is corrected, in combination with manual reduction of disorders of soft tissue, pan-systemic alleviation of the greater portion of chronic pain routinely follows (Irvin, 1998).

Adults with multi-regional chronic pain, not due to metabolic disease, and for whom the sacrum is leveled by a pelvic lift beneath the heel on the low side, have enduring reduction of the number of regions with pain by $\sim$70% (experimental results), and with marked reduction of pain for those regions remaining symptomatic (the later being anecdotal) (see Figure 3).

Where the feet and ankles are corrected, the sacral base is leveled (both seated and standing), and accrued restriction of soft tissues relieved, $>$90% of mechanically mediated pain is enduringly relieved (anecdotal).

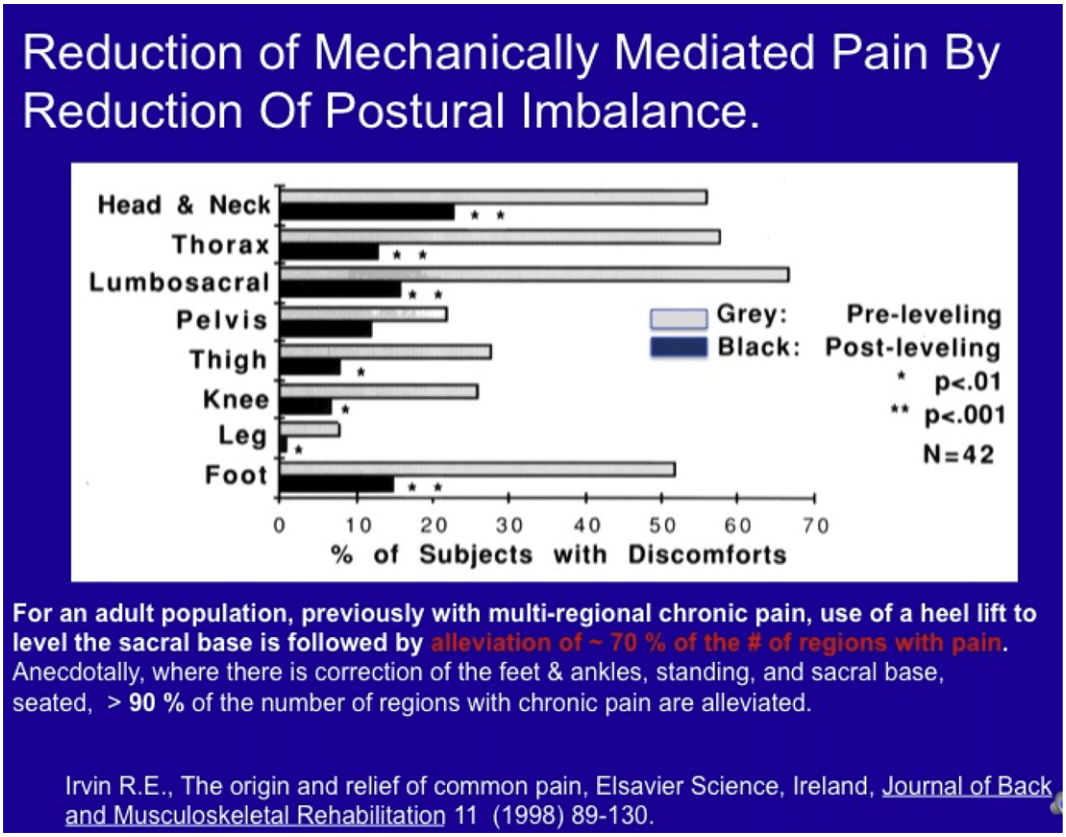

Figure 3 A graphic reflection of pan-systemic alleviation of the greater portion of chronic pain by correction of pelvic obliquity, combined with manual manipulation to reduce tissue restriction throughout all regions.

Independent research of the effects of leveling the unlevel standing pelvis, estimated by physical exam, combined with use of custom orthotics to correct the shape of the feet and promote the vertical alignment of the ankles, with manual manipulation to reduce accumulated restriction, results in alleviation of ~80% of reported chronic pain (Lipton et al., 2009).

A final problem is that two (Irvin, 1998; Lipton et al., 2009) of three (Hoffman and Hoffman, 1994) studies that demonstrate the strong effect of postural balancing on chronic pain by correction of boundary conditions in addition to manual reduction of disordered tissues, do not include either a control population or a sham heel lift. Perhaps for reason of this, and for reason of the significant effects reflected by all three, these studies are not mentioned in Dr. Lederman's paper that asserts the fall of the postural—structural—biomechanical model. This claim follows reasonably from the preponderance of experimental outcomes for most studies with the aforementioned flaws, relative to the actual nature of postural imbalance.

Admittedly, to demonstrate a cause and effect hypothesis, an experiment must often show that, for example, a phenomenon occurs after a certain treatment is given to a subject, and that the phenomenon does not occur in the absence of the treatment. A reasonable exception to this requirement for a control population is where the phenomenon being tested is known not to occur, otherwise. For instance, if one were to evidence that a particular treatment returns the dead to life, it would add no credence to the experimental outcome to assemble a control population of corpses for which treatment was withheld. Further, one might ably fool a corpse with a soft foam lift, but not a conscious human.

Strong reduction or enduring alleviation of chronic pain throughout all regions of the body that is mechanically mediated is known not to occur spontaneously for a representative population, nor has it been achieved experimentally by any other therapeutic method.

Another practical example of a lack of need for controls is the significant reduction of lumbar scoliosis by use of a pelvic lift to level the sacral base (Irvin, 1991). For a representative population, scoliosis is known not to significantly reduce spontaneously, or by any non-surgical method, other than by leveling the sacral base.

Altogether, manual and physical therapeutics for postural imbalance and its effects, while often effective on a case-by-case basis, are short a necessary practice of synchronously correcting the boundary conditions for posture. Where both the pes planovalgus and the sacral base obliquity are corrected via orthotics, there are routinely achieved large and enduring reductions of chronic, multi-regional pain in representative populations.

Contemporary experimental design, aimed to validly test the operational linkage between postural imbalance and pain, must be in accord with a broadened model for determinism that is informed of the nature of origin systematics and its pan-systemic and widely varied potential for generation of intermediate causes and eventual effects. While we cannot reliably predict the intermediate causes and effects

from postural imbalance (observational causality), we can reliably predict the large and enduring reductive outcomes for these effects from correction of postural imbalance, satisfying the determinism for manipulable causality.

The postural-structural-biomechanical model has not fallen, but rather shall rise like the Phoenix from the ashes of prior research and of therapeutic methods that were not, until the near future, sufficiently informed of a more full nature of posture than is recognized contemporarily. Our proper concern is not with the success and failure of past practices, but rather with the advancement of future practices so informed and thereby having the greatest efficacy, least cost, and least risk for our patients.

References

Dieck, G., et al., 1985. An epidemiologic study of the relationship between postural asymmetry in the teen years and subsequent back and neck pain. Spine 10, 872—877.

Fann, A.V., 2002. The prevalence of postural asymmetry in people with and without chronic low back pain. Arch. Phys. Med. Rehabil. 83 (12), 1736—1738.

Grundy, P.F., Roberts, C.J., 1984. Does unequal leg length cause back pain? A case-control study. Lancet 2, 256—258.

Hoffman, K., Hoffman, K., 1994. Effects of adding sacral base leveling to osteopathic manipulative treatment of back pain: a pilot study. J. Am. Osteopathic Assoc. 3, 217—322.

Irvin, R.E., 1991. Reduction of lumbar scoliosis by the use of heel lift to level the sacral base. J. Amer. Osteo. Assoc. 91 (1), 34—44.

Irvin, R.E., 1986. Postural Balancing: a regimen for the routine reversal of chronic somatic dysfunction, abstracted. J. Am. Osteopathic Assoc. 86 608/125.

Irvin, R.E., 1998. The origin and relief of common pain. Elsevier Science Ireland. J. Back Musculoskelet. Rehabil. 11, 89—130.

Irvin, R.E., 2007. Why and how to optimize posture, chapter 16. In: Vleeming, et al. (Eds.), Movement, Stability and Lumbopelvic Pain, second ed. Elsevier, Edinburgh.

Lederman, E., March 2010. The fall of the postural-structural-biomechanical model in manual and physical therapies: exemplified by lower back pain. Online J., 1—14. WWW.CPDO.net.

Lipton, J.A., et al., 2009. The use of heel lifts and custom orthotics in reducing self-reported chronic musculoskeletal pain scores. Am. Acad. Osteopathy J. 19 (1), 15—20.

Rapoport, A., 1954. Operational Philosophy: Integrating Knowledge and Action. Harper and Brothers Publishers, New York. pp. 57—64.

Invited response (Evidence and clinical experience: the challenge when they conflict)

Diane Lee, BSR, FCAMT, CGIMS

Diane Lee & Associates, Discover Physio, White Rock, BC, Canada

According to the Canadian Institutes of Health Research,

> *Knowledge translation is defined as a dynamic and iterative process that includes synthesis, dissemination, exchange and ethically-sound application of knowledge to improve the health of Canadians, provide more effective health services and products and strengthen the health care system. (CIHR, 2009, Salbach, 2010).*

In his article, The fall of the postural-structural-biomechanical model in manual and physical therapies: Exemplified by lower back pain, Lederman attempts to translate knowledge gained from some research pertaining to a postural-structural-biomechanical (PSB) model and makes conclusions regarding:

1. the role a PSB may or may not play in causing lower back pain and
2. how effective a PSB model is for the treatment of lower back pain.

What is the PSB model and who uses it? According to Lederman, the PSB model is somewhat vague and appears to encompass almost every approach known for the assessment and treatment of low back pain including physiotherapy, osteopathy, chiropractic, massage therapy, manual therapy, bodywork, craniosacral therapy, visceral therapy, Rolfing, Structural Integration, and all forms of exercise or movement training including, but not limited to, motor control training, neurodynamics, McKenzie back exercise, personal training, kinesiology, Yoga, and Pilates. Simply put, he uses certain studies to support his conclusion that:

1. factors inherent to his defined PSB model do not cause back pain and furthermore, that
2. the 'therapeutic investment in correcting postural-structural-biomechancal factors is irrational, in particular, as it is unlikely to influence the course of the patient's condition'.

He summarizes by suggesting that a PSB model is unnecessarily complex, has no value in elucidating the cause of low back pain, that the assessment tools (manual and visual) are unreliable and can be removed from clinical practice, and that PSB factors are unlikely to change in the long term with exercise and warns that this model may introduce an element of therapeutic failure as the aims and goals of this approach may not be attainable by manual therapy or even exercise.

E-mail address: dianelee@dianelee.ca.

This is a potentially dangerous interpretation of a limited, and specifically chosen, number of studies, and while it is important to reflect on the knowledge gained from these studies, it is also important to note that they do not represent the entire body of scientific evidence we now have for understanding the multi-factorial nature of individuals with back pain. This paper has clearly reinforced why nothing significant is gained when individuals with the same pain are grouped together for any investigation. Individuals with low back pain clearly are not a homogenous group; and it is not new knowledge that PSB factors do not cause pain.

> *As we all know, general diagnoses such as low back pain or hip pain do not often relate to the cause or to the underlying nature of the condition (Sahrmann, 1988).*

Clinicians have long recognized, and it is becoming more evident in recent scientific research, that patients with low back pain are heterogenous, and consist of multiple sub-groups with different combinations of underlying impairments, or mechanisms, driving their pain (physical and psychosocial), and these sub-groups require different treatment approaches for best outcomes (Dankaerts et al., 2006, 2007; Fersum et al., 2009; Fritz et al., 2003; Lee D and Lee LJ, 2011, O'Sullivan, 2005). The pursuit of valid ways to identify sub-groups of patients with low back pain has become increasingly prominent in the literature. In fact, a large part of the recent 7th Interdisciplinary World Congress on Low Back and Pelvic Pain (Vleeming and Fitzgerald, 2010) was dedicated to research that investigated sub-groups of individuals with LBP so that the multiple, and diverse, biopsychosocial factors that dominate the multiple sub-groups could be better understood and subsequently managed. Given that multiple factors are known to contribute to pain, it is also unrealistic to expect that one single type of treatment (manual therapy, dry needling, exercise training, or education about pain etc.) will resolve a patient's presenting pain and improve their quality of life. Thus studies which apply a single modality within the PSB model to all subjects with low back pain and then compare the outcome of one intervention to another, rarely show a consistent positive effect across repeated studies. The evidence cited by Lederman in this article clearly supports this. But does that mean that the intervention investigated was ineffective and should therefore be abandoned as he suggests, or should we consider the methodology of the particular study and perhaps question the validity of the findings?

There is a large body of evidence not cited in this article that provides a bigger, more accurate picture of the state of the science pertaining to individuals with back and pelvic pain and how this knowledge is being translated into clinical practice (see the reference lists provided in the proceedings of the 7th Interdisciplinary World Congress on Low Back and Pelvic Pain as well as recent European Guidelines for pelvic pain (Vleeming et al., 2008) for a broader view of the current evidence on this topic). You may ask, is there evidence to ever support the use of a postural-structural-biomechanical model? What is a postural-structural-model anyway? What is evidence? Where do we gain knowledge to practice our professions? What knowledge should we pay attention to?

Every day in clinical practice, health care practitioners meet patients seeking help for their loss of function (disability) and pain. Clinicians are keenly aware of the need to be effective in clinical practice and many feel that this requires being evidence-based, that is why we read articles that summarize evidence and why it is so important that the knowledge gained from the evidence be accurately translated. Sackett, Straus, Richardson, Rosenberg and Haynes (Sackett et al., 2000) define evidence-based practice as 'the integration of best research evidence, with clinical expertise and patient values.' They note that,

> *External clinical evidence can inform, but can never replace individual clinical expertise, and it is this expertise that decides whether the external evidence applies to the patient at all, and if so, how it should be integrated into a clinical decision.*

It is unlikely that there will ever be enough research evidence for every situation met in clinical practice and therefore sound clinical reasoning skills will be needed together with clinical expertise to bridge the gap between what science suggests and what we need to know practically to treat patients with disability and pain. What is clinical expertise? According to Ericsson and Smith (1991), 'expertise has been defined as having the ability to do the right thing at the right time.'

Given the diversity of individuals with low back pain, is it possible to classify, or subgroup, every individual who presents with low back pain? Clinically, I strongly agree with Jones and Rivett (2004) who note that:

> *Given the same painful impairment, no two individuals will have exactly the same experience and behavior because how they manifest their pain or illness is shaped in part by who they are.*

So, how do we resolve the dilemma of knowing what to do when someone presents in our office with low back pain? What evidence, protocol or guideline should we follow? Is there, or will there ever be, enough evidence to totally guide clinical practice? This is not likely and, actually, is highly unrealistic. We need frameworks within which to organize all the knowledge being accumulated both from research and clinical expertise as well as the ability to use sound clinical reasoning skills to develop prescriptive, individual treatment programs that address the multiple and diverse mechanisms driving the low back pain; one person at a time (Lee LJ and Lee D 2011).

While many therapists may use components of what Lederman has vaguely defined as a PSB model, in reality the interventions are used in an integrated and variable manner specific to the individual's needs, in context of the health of the whole person. This is very different to the assertion made by Lederman that clinicians view the body as a mechanical entity. It is highly likely that the research on individual components of the PSB model that Lederman cites is not at all reflective of the multi-modal, patient centered approach that integrates postural, structural and biomechanical factors used by clinicians. Clinicians need to publish more individual case reports and collaborate with researchers so that the scientific evidence can more accurately reflect what is occurring in clinical practice.

Science can provide us with an abundance of knowledge to challenge, refine, reshape, and validate our clinical

practice, but it cannot provide all of the information needed in any individual patient encounter; it does not paint the whole picture of the patient. In order to effectively treat patients, clinicians need to have well-organized knowledge including propositional (knowledge ratified by research trials), non-propositional (professional craft or 'knowing how' knowledge) and personal (knowledge gained from personal experiences) (Jones and Rivett, 2004). It is indeed a process, one that requires you to be informed (read the evidence or systematic reviews, journals, attend conferences), skilled (expose yourself to multiple approaches, techniques, training and integrate what is effective for you and your patients in the clinic) and capable of critical thinking (don't merely accept everyone's conclusions, reflect on how they resonate with your own experience in the clinic) to know when the evidence, or interpretation of the evidence, should, or should not, transform your clinical practice. The evidence cited in this article, as well as the interpretation of its author, have not convinced me that his representation of the postural-structural-biomechanical model in manual and physical therapies is accurate, or that it should fall.

References

Canadian Institutes of Health Research, 2009. About knowledge translation 2009 Ottawa: The Institutes. http://www.cihr-irsc.gc.ca/e/29418.html.

Dankaerts, W., O'Sullivan, P.B., Straker, L.M., Burnett, A.F., Skouen, J.S., 2006. The inter-examiner reliability of a classification method for non-specific chronic low back pain patients with motor control impairment. Man. Ther. 11 (1), 28.

Dankaerts, W., O'Sullivan, P., Burnett, A., Straker, L., 2007. The use of a mechanism-based classification system to evaluate and direct management of a patient with non-specific chronic low back pain and motor control impairment - A case report. Man. Ther. 12 (2), 181.

Ericsson, K.A., Smith, 1991. Towards a General Theory of Expertise: Prospects and Limits. Cambridge University Press, New York.

Fersum, K., O'Sullivan, P., Kvåle, A., Skouen, J., 2009. Inter-examiner reliability of a classification system for patients with non-specific low back pain. Man. Ther. 14, 555—561.

Fritz, J., Delitto, A., Erhard, R., 2003. Comparison of classification-based physical therapy with therapy based on clinical practice guidelines for patients with acute low back pain: a randomized clinical trial. Spine 28 (13), 1363—1372.

Jones, M.A., Rivett, D., 2004. Introduction to clinical reasoning. In: Jones, M.A., Rivett, D.A. (Eds.), Clinical Reasoning for Manual Therapists. Elsevier, Edinburgh, p 3.

Lee, L.J., Lee, D., 2011. Clinical practice - the reality for clinicians. Ch. 7. In: Lee, D. (Ed.), The Pelvic Girdle, fourth ed. Elsevier, Edinburgh.

Lee, D., Lee, L.J., 2011. Clinical reasoning, treatment planning and case reports. Ch. 9. In: Lee, D. (Ed.), The Pelvic Girdle, fourth ed. Elsevier, Edinburgh.

O'Sullivan, P., 2005. Diagnosis and classification of chronic low back pain disorders: maladaptive movement and motor control impairments as underlying mechanism. Man. Ther. 10 (4), 242—255.

Sackett, D.L., Straus, S., Richardson, W.S., Rosenberg, Haynes R B, 2000. Evidence-based Medicine. How to Practice and Teach EBM. Elsevier Science, New York.

Sahrmann, S.A., 1988. Diagnosis by the physical therapist — a prerequisite for treatment. Phys. Ther. 68 (11), 1703.

Salbach, N.M., 2010. Knowledge translation, evidence-based practice, and you. Physiother. Can. 62 (4), 293.

Vleeming, A., Albert, H.B., Ostgaard, H.C., et al., 2008. European guidelines for the diagnosis and treatment of pelvic girdle pain. Eur. Spine 17 (6), 794.

Vleeming, A., Fitzgerald, C. http://www.worldcongresslbp.com.

Invited response

Stuart McGill, BPE, MSc, PhD

Spine Biomechanics Laboratory, University of Waterloo, Canada

I have generalized philosophical concerns together with concerns over substantive issues regarding this paper. The topics are worthy of discussion, however, the sensational title sets the expectation for solid evidence and rigor in developing an issue and the counterpoint. The author repeatedly used a strategy in the framing of a question to make it easily dismissible, and then declared that "there is no evidence to support XXXX". The arguments put forth in many instances were unidimensional, incomplete, based on undeveloped expressions of concepts, and neglectful of developed bodies of knowledge together with their interactions. Critique of cited works was, for the most part, absent. I will also declare at the outset that I am sympathetic to several positions that the author took. But this must not affect the job assigned to me here. My points are largely directed towards the process of creating compelling argument — the appropriate treatment and interpretation of specific papers and quotes I will leave largely to others.

Back pain is not a homogeneous condition. Any therapeutic approach that helps one individual will exacerbate another. Epidemiological studies of "back pain" will never reveal cause and effect, or efficacy, since each individual will respond to a different approach, and different dosage. Thus a controlled study on "back pain" will result in the conclusion of "no effect". However, when patients are categorized into sub-groups based on pathomechanics, or pain patterns, or history, or even psychosocial variables,

E-mail address: mcgill@uwaterloo.ca.

results in the evolving literature are generally positive. This paper ignored these studies together with those showing the role of biomechanical factors in the causation pathway. As an exercise in logic let's replace the discussion of "back pain" with "leg pain". No study of non-specific leg pain would be expected to reveal cause/effect (the genesis of pain could be due to mechanical, vascular, hereditary, or many other factors). Similarly no credible individual would use such logic to state there is no evidence of links between mechanical factors and non-specific leg pain. Nor would it be published. However, a very specific "leg disorder" such as subsequent ACL disruption in female basketball players has been substantially reduced by a specific intervention to alter biomechanics (e.g. Hewett et al., 1999). One could argue that the treatment effect is underestimated. Even in these controlled clinical trials everyone in the group received the same treatment. However, in excellent clinical practice each intervention is tuned and adjusted to each patient suggesting that efficacy in real life on a patient by patient basis is probably better than any controlled trial would suggest. Well presented examples of the interactions between individual anatomical, morphological, mechanical and neuro-mechanical factors have been compiled by McLean and Beaulieu (2010), implying the need for individual assessment and treatment. Dr. Lederman contends that these relationships do not exist for the spine. The evolution of the spine literature lags that on the knee. Yet evidence exists on disc shape, to choose just one example from our own laboratory (Yates et al., 2010), influencing the patterns of annulus disruption from specific modes of loading resulting from specific movement patterns. This supports individual assessment of anatomical features to justify appropriate clinical effort to alter offending movement. The author rightly questions the ability of pained individuals to change movement patterns over longer terms but studies of the barriers and variables leading to success do exist and acknowledge the need for more study to elucidate the optimal motor learning approach for each individual. This does not mean that "no evidence exists". The complex interactions of structure and biomechanics ignored in the paper under consideration suggest that evidence from controlled trials of back pain must be coupled and interpreted with treatment studies of appropriately sub-classified patients, even to the point of case studies, to delineate the complex interactions.

Definitions of terms are needed particularly when entire bodies of science are summarily dismissed. "Biomechanics" includes kinematics and kinetics. The links between movement patterns, resultant tissue stresses, and pain are well documented in the injury pathway literature but were ignored in this paper. "Biomechanics" in the title, following "The fall of" infers a discussion of this literature. The title does not match the content of the paper.

The links between optimal health and biomechanical factors form a "U" shaped function. Too little loading causes mechanical disruption at the cellular level and disease, and too much also creates mechanically modulated tissue disruption and disorders. Flawed movement (biomechanics) creates stress concentrations well documented to alter the course of back pain. The literature is extensive describing detrimental changes from too little and too much load on an individual's spine that was obtained from many experimental approaches. However, interpretation of the literature on the effects of biomechanics and loading need to be rigorous enough to critique the load exposure, and whether it was leading toward the bettering or worsening of the symptoms. The authors' treatment of this concept throughout the paper was inappropriately dismissive.

This paper neglected the complexity of the many systems and interactions that obscure clear explanations. Having been involved in spine research I am aware of the limitations of capturing the complexities in a short paper. However, the author's example of interpretation of medical images forms a case in point. The static images are commonly acquired in an unloaded, recumbent posture. Dynamic images (multiple x-ray, fluoroscopy, dynamic MRI) show much more movement pathology where the dynamics can be directly related to pain. These techniques, developing literature and evidence were ignored. Discogenic pain, for example, has been shown to follow a natural history where the zenith of pain is associated with the unstable phase but that eventually ends with a very desiccated disc on a medical image and where the pain has subsequently "burned out". Then, abundant evidence shows, the history continues as changes in joint function influence the mechanical loading of the facet joints. Eventually these become arthritic shifting the pain source from disc to facet. Central sensitization further influences the process and links between pain, mechanical/functional factors and patient corrections to wind down the heightened neural response. The implication of this literature is that the links between pain and biomechanical factors are variables over the natural history of back pain. Integrated natural history concepts were ignored in the paper and obscured a mature interpretation of what this collection of evidence really shows — that being that the links between pain, biomechanics and an image are complex variables, are patient specific, and rigor is required to understand them.

Professor Lederman selected a few studies that suggested no causation of back pain from structural and neuromuscular asymmetry. Yet he chose not to report or critique those studies that have shown predictive links, particularly asymmetry in hip related variables that predict future back pain. Some studies were reported suggesting that neuromuscular asymmetry does predict subsequent first time back injury but these were dismissed. One example was his dismissal of Cholewicki et al's (2005) and Zazulak et al's (2007) work that suggested those athletes with delayed muscle reflex response rates were more likely to develop back or knee injuries. This is difficult longitudinal work. Dr Lederman writes "Unfortunately, the obvious was not examined in other body areas (e.g. controlled recording in the leg). This would have helped establish whether the injuries are due to delayed muscle response onset timing specific to the trunk or, the alternative more plausible explanation that athletes with sluggish muscle reaction times/reflexes may be more susceptible to injury". First, the Cholewicki study noted the predictive variable was in muscle shut-off latency time — not onset timing. Second, the Zazulak study never mentioned muscle response. Zazulak et al reported predictive associations between back pain and future knee pain/injury based on movement parameters. These substantial inaccuracies aside, the back is part of a skeletal linkage and skilled

clinicians assess the linkage — they are familiar with the literature linking restricted hip motion for example, with the development of future back pain (the literature from sport and military examples was ignored). A broader integration of the neuromuscular/biomechanical literature offers insight into the mechanisms and consequences of anatomical linkages (knees and back in this case), and neuromuscular variables that impact the entire linkage. Extending Dr Lederman's logic, and dismissive word choice - In response, "Is this not obvious"? Dr Lederman chose to write about the "unfortunate" oversights of others who have dedicated themselves to performing difficult work and holding it up for peer review. Errors in misrepresenting cited work followed by criticism is not forgivable. Credibility of those who choose to dismiss others within academic debate requires evidence of their own contributions to the scientific literature.

The scientific process incorporates the principle that the critic of a widely held position bears the "burden of proof" to support an alternate view. Dr Lederman concludes "The PSB model introduces unnecessary complexity and hurdles to practice. The first hurdle… is the inability to identify/define the critical level where PSB factors contribute to the individuals back pain. This critical level is impossible to predict on an individual basis". This opinion was stated in the face of broad evidence regarding provocative testing of the patient that determines the motions, postures or applied loads that cause pain. This was ignored. Further there is more literature pertaining to the pain being eliminated or reduced when the offending trigger level of the biomechanical variable is removed. A critique of this literature would have been essential for the authors' opinion to be credible and demonstrative that the burden was carried.

This is the second paper from Dr Lederman where he has declared an approach to practice mythical (the myth of core stability) or fallen (this paper). As with the "Myth of core stability" failure to really define what is meant by core stability defeated a rigorous discussion to pull out elements that are helpful — an entire clinical approach was soiled. Similarly, this paper ignored important research findings and confused posture, therapeutic exercise mechanism and efficacy, movement kinematics and kinetics, static anatomic structure, dynamic joint function, neurological function, pain mechanisms, natural histories of different spine structure damage, patient classification, and tissue loading, to name just a few. The process of establishing cause and effect, understanding of mechanism and creating an evidence base for clinical practice, requires a scholarly integration of the evidence. Dismissing singular papers, without the integrative context does not pass the test of appropriate rigor. In a positive sense the scientific community will work to further understanding of the relationships that form the evidence. Discussion is wonderful — exciting, educational and entertaining. However, sensational titles, and similarly sensational concluding statements, sets the expectation of critical analysis, fairness and rigor.

References

Cholewicki, J., Silfies, S.P., Shah, R.A., Greene, H.S., Reeves, N.P., Alvi, K., Goldberg, B., 2005. Delayed trunk muscle reflex responses increase the risk of low back injuries. Spine 30 (23), 2614—2620.

Hewett, T.E., Lindenfeld, T.N., Riccobene, J.V., Noyes, F.R., 1999. The effect of neuromuscular training on the incidence of knee injury in female athletes: a prospective study. Am. J. Sports Med. 27, 699—706.

McLean, S., Beaulieu, M.L., 2010. Complex integrative Morphological and mechanical contribution to ACL injury risk. Exerc. Sports Sci. Rev. 38 (4), 192—200.

Yates, J., Giangregorio, L., McGill, S.M., 2010. The influence of international disc shape on the pathway of posterior/posterior lateral partial herniation. Spine 35 (7), 734—739.

Zazulak, B., Hewett, T.E., Reeves, N.P., Goldberg, B., Cholewicki, J., 2007. Deficits in neuromuscular control of the trunk predict knee injury risk: a prospective biomechanical-epidemiologic study. Am. J. Sports Med. 35 (7), 1123—1130.