Rehabilitation of lumbar multifidus dysfunction in low back pain: strengthening versus a motor re-education model

Regarding the article “Effects of three different training modalities on the cross sectional area of the lumbar multifidus muscle in people with low back pain,” I would like to extend my appreciation to Dr Danneels and his colleagues for their interest in contributing to the literature on this important and clinically relevant topic. Unfortunately, there are important design and methodological flaws inherent in this study which call into question its results and primary conclusions. I respectfully submit this review of the study, its findings, and the authors’ clinical recommendations.

The objective of the investigation of Danneels et al was to determine the potential for different exercise models to reverse the pathology related atrophy of the lumbar multifidus muscle in people with low back pain. As described by various researchers, the lumbar multifidus experiences a number of morphological and neurophysiological changes following low back injury. One of these changes is a segmental atrophy which develops at the level of pathology, on the symptomatic side and as quickly as 24 hours after the injury. Further, these changes have been shown to persist beyond the resolution of symptoms, and for at least five years after surgical intervention for intervertebral disc herniation. There is evidence that such findings are indicative of a neurologically mediated process rather than a simple disuse or weakness phenomenon.

In their study, Danneels and colleagues compared the motor re-education model, originally developed by Richardson et al and as studied by O’Sullivan and colleagues, against two variations of a traditional strengthening model. The first of these strength training variations utilised typical concentric and eccentric lumbar extensor loading motions. The other added a static or isometric component which was to be maintained between the concentric and eccentric phases of the exercise.

The authors concluded that, in order to correct the atrophy observed in the lumbar multifidus, patients should perform strengthening exercises targeting the lumbar extensors, ideally incorporating an isometric “pause” into these exercises. Danneels et al reported that this was the only exercise model tested that developed significant hypertrophy and improved strength in the multifidus atrophy seen in their experimental population.

These findings conflict with those of Hides and colleagues, who have published data showing correction of the pathology-induced lumbar multifidus atrophy using a considerably more specific and subtle activation of the multifidus muscle. Unfortunately, the method by which the authors measured the cross sectional area (CSA) of the multifidus muscle introduced a large degree of methodological error, calling into question the study’s harmfulness and therefore its clinical recommendations. Using computed tomography scanning, Danneels et al took measurements from three arbitrary levels of the lumbar spine (the L3 superior end plate, the L4 superior end plate, and the L4 inferior end plate) recording the CSA of the multifidus muscle at each of these levels bilaterally. They then summed the right and left multifidus CSA at each of these segments yielding in a single multifidus score for each level.

In preselecting the levels from which CSA measurements would be taken, it is possible, indeed likely, that the pathological level would have been missed entirely in at least some of the subjects. This is important because previous studies have shown that the multifidus muscle experiences its greatest loss in CSA at the primary level of pathology. Also problematic was the decision to sum the CSA scores at each of the preselected levels. A number of studies have shown that the lumbar multifidus, ipsilateral to the pathological side, experiences a cascade of neuro-morphological changes, including atrophy, in the presence of both acute and chronic pathology, whereas the multifidus contralateral to the pathological side experiences no such changes.

Taken together, the preselection of measurement levels and the summation of the bilateral multifidus CSA would have had the effect of attenuating any side to side differences in multifidus CSA even if a subject’s level of pathology happened to coincide with one of the levels from which measurements were obtained. Through either of these mechanisms, this measurement scheme would have introduced a substantial mass of healthy, non-motor dysfunctional muscle into each of the multifidus CSA measurements. The net effect of the measurement approach employed by Danneels et al, although probably intended to more discretely reflect the pathological side multifidus, was to bias the outcome. The experimental intervention toward an exercise model with the ability to cause hypertrophy in healthy muscle—that is, a strengthening exercise. It is likely that the “dynamic-static” exercise recommended by the authors as being most effective for correcting the pathological atrophy of the multifidus instead caused hypertrophy of the non-pathological, non-atrophied multifidus muscle segments included in the three measurement scores. Ultimately, the study’s recommendations are unsustainable given this flaw in methodology.

The low load multifidus activation exercise, developed by Richardson et al and used by O’Sullivan and colleagues, is to be performed as a co-contraction with the transversus abdominis muscle, and is intended to correct a neurologically mediated loss of normal multifidus muscle volume, not unlike that seen in the vastus medialis following trauma or surgery involving the knee joint. In studies in which the CSA of a pathological multifidus muscle has been compared with its contra-lateral and “healthy” segmental partner, this form of motor re-education exercise has been shown to normalise the CSA of the pathological multifidus in as little as four weeks.

It is critical that both researchers and clinicians appreciate that a significant body of evidence now shows that atrophy seen in the multifidus muscle in people with low back dysfunction is representative of a form of impaired motor control, not simple disuse weakness. As such, traditional strengthening exercises will often fail to correct this fault, just as daily physical activities fail to maintain a normal segmental CSA at the pathological level. Certainly, the historical lack of success of the rehabilitation and medical professions in treating low back pain using the variety of strength based clinical models used over the last 50 years should serve as sufficient motivation to look to more evidence based models as an explanation for the condition. The motor control dysfunction model as developed over the past decade by a variety of researchers holds great promise, both as a basis for understanding the causes of back pain and in developing effective treatment strategies for our patients.

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References

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PostScript

The event side doctor: the role of the orthopaedic surgeon

Orthopaedic surgeons have long had a close association with sport. International sports players have similarly gone on to careers in orthopaedic surgery, examples being Jonathan Webb and JPR Williams.

Finding appropriate medical personnel to care for the needs of spectators and athletes at major sporting events is a challenge.1 Event organisers have required the skills of volunteer orthopaedic surgeons.2

I recently volunteered as a competitors’ doctor for the XVII Commonwealth Games in Victoria, Canada. As a specialist registrar in orthopaedics, and previously an emergency medicine registrar, I felt suitably skilled to be an event side doctor for the time trial, mountain biking, and road race events.

During the course of the events, I reviewed cyclists with dyspepsia and back muscle spasm. I also reviewed a cameraman with hay fever and one with eye irritation possibly secondary to the increased muscle mass in athletes. Serum creatine kinase was also found to be significantly higher in the athletes than control subjects (22.16 (0.87) vs 7.74 (0.29) pg/ml, p<0.001; values are mean (SEM)). Serum creatine kinase was also found to be significantly higher in athletes than controls (33.84 (4.33) vs 11.05 (1.73) IU, p<0.001; values are mean (SEM)).

The increased creatine kinase levels may be attributed to the increased muscle mass in athletes, and the increased serum ET-1 levels can be explained as a consequence of increased ET-1 production.1 This study, we found a difference in basal serum ET-1 levels between trained male athletes and normal matched male controls.

We studied 13 male professional football players (mean (SEM) age 28.6 (1.02) years; mean (SEM) body mass index 24.2 (1.2) kg/m²) and an equal number of sedentary or moderately physically active men (26 (1.3) years; body mass index 24.4 (1.8) kg/m²). All subjects gave written consent and had a negative family history of diabetes and hypertension. Blood samples were collected at 8 am after an overnight fast; all subjects remained at rest for 20 minutes in a supine position, before collection of the blood specimens. ET-1 concentration in serum was measured by radioimmunoasssay (Peninsula Lab Inc, Belmont, California, USA). Data were analysed by Student’s t test for independent samples.

The concentration of ET-1 in the serum was significantly higher in the athletes than control subjects (22.16 (0.87) vs 7.74 (0.29) pg/ml, p<0.001; values are mean (SEM)). Serum creatine kinase was also found to be significantly higher in athletes than controls (33.84 (4.33) vs 11.05 (1.73) IU, p<0.001; values are mean (SEM)).

The increased creatine kinase levels may be attributed to the increased muscle mass in athletes, and the increased serum ET-1 levels can be explained as a consequence of a widening of the vascular bed resulting from the increased muscle weight and size.

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References

Increased endothelin-1 levels in athletes

Endothelin-1 (ET-1), a potential vasoconstrictor peptide, may contribute to the exercise induced redistribution of blood flow in muscles.3,4 On the other hand, the latter parameter in athletes may be expanded secondarily as a consequence of increased ET-1 production.1 In this study, we found a difference in basal serum ET-1 levels between trained male athletes and normal matched male controls.

We studied 13 male professional football players (mean (SEM) age 28.6 (1.02) years; mean (SEM) body mass index 24.2 (1.2) kg/m²) and an equal number of sedentary or moderately physically active men (26 (1.3) years; body mass index 24.4 (1.8) kg/m²). All subjects gave written consent and had a negative family history of diabetes and hypertension. Blood samples were collected at 8 am after an overnight fast; all subjects remained at rest for 20 minutes in a supine position, before collection of the blood specimens. ET-1 concentration in serum was measured by radioimmunoasssay (Peninsula Lab Inc, Belmont, California, USA). Data were analysed by Student’s t test for independent samples.

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References
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Web site: www.basics.org.uk

Athletes Heart Symposium
17 December 2002, University College London, UK
An international multidisciplinary symposium for physiologists, cardiologists, sports scientists, and physicians in associations with the Physiological Society.
Further details: Lynn Coombs, Cardiac Department, Homerton University Hospital, Homerton Row, London E9 6SR, UK; email: ahssymp@btinternet.com

2nd World Congress of Science and Medicine in Cricket
4–7 February 2003, University of Port Elizabeth, South Africa
Further details: Dr Richard Stretch, University of Port Elizabeth, PO Box 1600, Port Elizabeth 6000, South Africa; tel: +27 41 5042584; fax: +27 41 5832605; email: sparas@upc.ac.za

The 2003 NSW Conference of Science and Medicine in Sport
1 March 2003, AJC Convention Centre, Alison Road, Randwick, NSW, Australia
Keynote speaker: Professor Nikolai Bogduk, University of Newcastle.
Further details: www.smansw.com.au or email smansw@dsr.nsw.gov.au

SMX 2003
22–23 March 2003, University of Melbourne, Victoria, Australia
The Victorial Conference of Science and Medicine in Sport and Exercise in conjunction with The Gastrolyte VIS International Science and Football Symposium.
Further details: members@vic.sma.org.au

Sports Medicine Seminar at the Hong Kong Sevens
27 March 2003, Hong Kong
This will be the first of an annual conference on Sports Medicine to coincide with the premier 7s event. Please visit the website www.droid.cuhk.edu.hk/events/sms.htm.
Further details: Iain Stewart, National Diagnostic Imaging, Woden, ACT 2606, Australia; tel: +61 2 6282 2888; email: ncdi@ozemail.com.au

Vth World Congress on Science & Football
April 2003, Lisbon, Portugal
Further details: Dr J Cabri; email: jcabri@fmb.ual.pt
Web site: http://www.fmb.ual.pt/wesf

2003 SMA Queensland State Conference
3–4 May 2003, Nara SeaWorld Resort, Gold Coast, Australia
Speakers: Dr John Best, Medical Director for the 2003 Rugby World Cup; Associate Professor Kim Bennell, Director, Centre for Sports Medicine Research and Education, (School of Physiotherapy), University of Melbourne, Victoria, Australia.
Further details: www.sportsmedicine.com.au

3rd Quebec International Symposium on Cardiopulmonary Rehabilitation Evidence Based Interventions: Science to the Art of Cardiopulmonary Rehabilitation
11–13 May 2003, Quebec City Convention Center, Quebec, Canada
Call for abstracts deadline is 1 November 2002. The abstract submission form and complete programme can be printed from the web site.
Further details: email: Jean.Jobin@med.ulaval.ca
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The 6th STMS World Congress on Medicine and Science in Tennis in conjunction with the LTA 2004 Sports Science, Sports Medicine and Performance Coaching Conference
Keynote speakers include Professor Per Renstrom (SWE), Professor Peter Jokl (USA), Professor Savio Woo (USA), Dr Carol Otis (USA), Dr Mark Safran (USA), Dr Ben Kibler (USA), Prof Bruce Elliott (AUS), and Professor Ron Maughan (UK).
Further details: Dr Michael Turner, The Lawn Tennis Association, The Queen’s Club, London W14 9EG, UK; email: michael.turner@lta.org.uk

International XVII Puijo Symposium
25–28 June 2003, Kuopio, Finland
This symposium “Physical activity and Health—Gender Differences Across the Lifespan.”
Further details: Ms Auli Korhonen, Project Secretary, Puijo Research Institute of Exercise Medicine, Puijo Symposium Secretariat, Haapaniementie 16, 70100 Kuopio, Finland; tel: +358 17 288 4422; fax: +358 17 288 4488; email: puijo.symposium@uku.fi

Winners of the annual BASEM Prizes
Dr Eileen Mackie (Clopidogrel inhibits platelet activation and exercise induced ischaemia in stable coronary artery disease) and Mrs Eleanor Curry (Role of exercise in multiple sclerosis) (joint winners).
The poster prize was won by Dr Stuart Reid (Injury patterns and injury prevention strategies in the winter sports population attending the English medical centre in Val d’Isère).

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Intercollegiate Academic Board of Sport and Exercise Medicine Diploma Exam
The following were successful diplomates in the Intercollegiate Academic Board of Sport and Exercise Medicine Diploma Exam: 7 July 2000
• Dr Prabodh C Agarwal
• Dr Robert Bleakney
• Dr Trevor W Fleet
8 November 2000
• Dr James P Robson
• Dr Samantha L Fee
• Dr David C Watkins
• Dr RS Prabu

Further information contact: Donald AD Macleod, Chairman, Intercollegiate Academic Board of Sport and Exercise Medicine.

www.basem.co.uk
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