

Leaders

Factors contributing to low back pain in rowers

Competitive rowing is a highly aerobic sport requiring technical skills, motor coordination, adequate strength, and endurance.^{1–2} A number of authors^{1–6} have reported a significant incidence of low back pain among the rowing population. This paper identifies the factors that may influence the onset of low back pain.

During the rowing stroke, the magnitude of the forces on the lumbar spine is high. Hosea *et al*⁷ reported average compressive loads of 3919 N for men and 3330 N for women, while anterior shear forces were found to be 848 N and 717 N for men and women respectively. Peak compressive loads during the stroke were 6066 N and 5031 N for men and women respectively. Furthermore, for 70% of the stroke cycle, rowers are in a flexed posture.⁷ Hosea *et al*⁷ recorded flexion ranges averaging 28–30° which equates to 55% of maximum range of spinal flexion. Tensile stresses on the outer annulus of the intervertebral disc have been found to increase considerably above 50% flexion.⁸ The combination of flexion with compressive loading has been identified as a mechanism for injury to the lumbar spine structures.⁹ In addition to flexion and compression, sweep rowers also rotate their trunks. This combination can place considerably more stress through the facet joint capsules and ligaments and may facilitate damage to discs, although the evidence for the latter is inconclusive.⁹ For rowers, the time of day will also influence the magnitude of the forces on their lumbar spines. In order to have calm water to row in, and to fit in with other daily commitments (work and study), a large volume of rowing is undertaken in the early morning. It is known that lumbar discs imbibe fluid from the surrounding tissue overnight when the disc is unloaded.¹⁰ Adams *et al*¹¹ calculated that the bending stresses are three times greater on the spine in the morning, and therefore this mechanism may make the disc and other ligamentous structures more vulnerable to injury in the morning particularly during activities involving flexion. Therefore it is suggested that repeated flexion and extension movements of the lumbar spine without load are undertaken at a slow speed for at least 60 seconds before rowing. Based on *in vitro* work,¹² 15 to 20 cycles of motion over this interval will decrease the bending moment by 8–10%. This motion should be undertaken in the sitting position that closely simulates the posture of the rower in the boat, and the range of motion should be gradually increased with subsequent repetitions. These exercises could also be undertaken on a rowing ergometer with the resistance set to zero.

It has been suggested that, during repetitive loading, compressive forces above 4000 N may cause damage to vertebrae.¹³ In industry, studies have shown that prolonged and cyclic flexion can result in a 10-fold increase in exposure to low back pain.¹⁴ The repetitive cyclic action of rowing may predispose the rower to low back injury. In a single session, a rower may train for 90 minutes and cover 20–25 km over that time. This amounts to about 1800 cycles of flexion per session. Although there is considerable variation, *in vitro* studies of repetitive loading have shown that damage can occur to lumbar vertebrae over a few hundred cycles of repetitive motion.¹⁵ Other researchers¹⁶ have suggested that injury to lumbar spine structures may occur when the bending moment on the lumbar spine exceeds about 23 N.m during repetitive motion. During everyday

lifting activities, the bending moment rises to about 18 N.m at L5-S1.¹⁷ In rowing, because of the larger loads on the spine and the influence of early morning training, it is likely that the bending moment is much higher.¹⁷

The repetitive motion of rowing may also induce creep in the soft tissues leading to a decrease in the stiffness of the tissues through the range of motion and an increase in the total range of motion in the lumbar segments.^{18–19} It has been suggested²⁰ that this process may ultimately lead to instability. Furthermore, repetitive motion can also desensitise the mechanoreceptors in spinal ligaments. These receptors often have pathways that lead to reflex activation of muscle.²¹ After repetitive motion, protective muscle activity has been shown to be reduced, often for a number of hours after the exercise is completed.²¹ The ramification for rowers is that, during this period, the athlete may be more vulnerable to injury, even when they may not be experiencing high loading on the spine.

Recently, it has been suggested that specific muscle activity can increase the stability of the lumbar spine. Research undertaken by Richardson and Jull²² and O'Sullivan *et al*²³ has focused on the importance of the transversus abdominus and the internal oblique abdominus groups, along with co-contraction of the multifidus muscles. These muscles have the potential to control the amount of movement in the lumbar segments, and their activation may therefore be useful in the prevention of low back pain in the rowing population. However, if these muscles are to control the amount of lumbar motion, they must be able to perform for long sustained periods. Roy *et al*²⁴ suggested that muscle fatigue may influence the incidence of low back pain in rowers. These researchers showed that rowers with low back pain became fatigued more easily than those without. Whether fatigue was a manifestation of the low back pathology or a factor that led to low back pain could not be determined. The ramifications of fatigue are related to the kinematics of the lumbar spine during the rowing stroke. If the erector spinae muscles are fatigued, the amount of lumbar flexion occurring during the rowing stroke may be increased, thereby increasing the bending moments on the spine. Such an increase may lead to additional strain on the passive structures of the spine such as ligaments and adjacent tissues.²⁴ More recently, Taimela *et al*²⁵ showed that fatigue of lumbar muscles affected proprioception. This study demonstrated that lumbar fatigue significantly impaired the ability of subjects to sense the position of their trunks when in flexion. For rowers, this may mean that, as they become fatigued, they may not be aware that they are moving into a more flexed posture.

Howell⁴ reported that 94% of rowers showed hypermobility of the lumbar spine, and this correlated strongly with the incidence of low back pain. It has been suggested⁶ that, to decrease the forces on the lumbar spine, rowers should adopt a less flexed lumbar spine, particularly at the catch phase when the oar is placed in the water. In this respect, if the pelvis could be rotated more anteriorly, less motion would be required in the lumbar spine. A major restraint to pelvic motion is the length and stiffness of the hamstring muscles. Studies by Gajdosik *et al*^{26–27} have shown that shorter hamstrings are associated with increases in range of lumbar and thoracic flexion. This has

consequences for rowing. If the athlete has short hamstrings, then to achieve the appropriate posture at the catch, he/she may overflex the spine. Hence it is important for rowers to include hamstring stretching exercises in their training programmes.

In summary, the large forces combined with the repetitive nature of the activity create the potential for injury to the lumbar spine structures during rowing. However, the warming up activities of rowers, the time at which they train during the day, the control of lumbar motion by specific muscle activation patterns, and the flexibility of the hamstring muscles can influence these forces. Incorporating these factors into training and rehabilitation programmes may lead to a reduction in the incidence of back injuries in rowers.

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Why exercise in paraplegia?

Spinal cord injury leads to two dramatic changes: not only is there loss of muscle function and a large amount of muscle, but also susceptibility to inactivity related diseases, such as obesity, insulin resistance, type II diabetes, and coronary heart disease, increases.¹ Previously, one of the major problems and causes of death for people with spinal cord injuries was infection, but recently, coronary heart disease has become more prominent. The possibilities for exercise in people with spinal cord injuries are limited to either performing voluntary exercise with non-paralysed muscle groups—for example, arm exercises, especially in the paraplegic—or subjecting themselves to electrically induced exercise through stimulation of motor nerves either with surface electrodes or after implantation of electrodes.

Whereas voluntary arm exercise can provide a certain stimulus to the cardiorespiratory system, it has recently been shown that stimulation of paralysed lower extremity muscles alone or in combination with arm cranking will not only increase energy combustion, but also activate more muscle groups and thus influence metabolic changes such as insulin resistance in a potentially better way. After the use of electrical stimulation for bladder and intestines, the possibility of stimulating paralysed muscle in a functional manner came to the fore at the beginning of the 1980s and allowed the development of a computerised bicycle (FES).² The use of such a bicycle for functional electrical stimulation has been shown not only to improve maximal oxygen uptake and endurance of the stimulated muscles, but also to cause muscle hypertrophy and muscle fibre shift from fast twitch type 2X to 2A.³

In addition to these effects, oxidative enzyme activity has also been shown to increase after several weeks of training.⁴ This occurs at a faster rate than the shift in fibre type, indicating different time patterns for the adaptation of these two systems. In addition, the collagen in muscle adapts to electrical stimulation, and it has been shown that type 4 collagen, which is predominant in the basal membrane, increases its turnover without any net increase in total amount, indicating possible reorganisation of this connective tissue.⁵ In addition to these effects, expression of the protein used for glucose transport (Glut4) increases with training and so does insulin stimulated glucose uptake in the muscle.⁶

Finally, it has been shown that functional electrical stimulation of paralysed legs increases bone mineral content of the tibial region. In studies using FES bicycling, high frequencies were used for stimulation, and no type I fibres were observed after this training. However, stimulation with lower frequencies actually seems to produce an increase in mRNA for myosin heavy chain type I after several weeks of training.⁷

In combination, the effects of functional electrical stimulation counteract the enzyme activity associated changes in people with spinal cord injuries and should thereby have a preventive effect.

In addition to these effects, electrical stimulation of partially paralysed muscle groups such as wrist extensor and muscles in tetraplegic people has been shown to result in improved function and endurance of the affected arm allowing more daily functions to be performed than before the training programme.⁸

Finally, it seems that training in people with spinal cord injuries improves their general wellbeing, temperature regulation, and sleeping patterns and reduces pressure sores, all important effects in addition to those mentioned above. It is therefore vital to encourage physical activity, including the use of electrical stimulation devices, in this group of patients in order to prevent diseases associated with physical inactivity. Such diseases not only occur in this group of people, but also reflect the general pattern in our modern inactive society. Results obtained in research on people with spinal cord injuries may therefore help to provide a basis for recommendations on exercise in the general population also.

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Magnetic resonance technology in training and sports

When muscles are used to perform physical activity they must metabolise available fuel to generate energy for contraction. The harder a muscle must work, the more fuel is required. The relation between how hard muscles must work and their need for fuel is an area of intense interest in the study of human performance. In the past, intramuscular energy metabolism has been measured directly by muscle biopsies,¹ which are invasive. During the last two decades, the sophistication of magnetic resonance (MR) technology has steadily improved. Using magnetic resonance spectroscopy (MRS), it is now possible to detect non-invasively changes in a number of important intramuscular fuel sources, such as muscle glycogen,^{2–5} during exercise and recovery. Magnetic resonance imaging (MRI) has been used for some time to examine anatomical effects of sport and training.⁶ Recently it has become possible to measure exercise induced physiological changes with MRI and use information from these measurements to determine muscle activity patterns.^{7–9} These more recent advances open up a new range of possibilities to use MR technology not only as a diagnostic tool, as in the past, but in a proactive manner to assess human performance.

Although MRS cannot completely replace the direct biochemical measurements obtained from muscle biopsy samples, it offers distinct advantages that are not available with biopsies. It provides a non-invasive direct measurement of muscle energy metabolite concentrations (glycogen, creatine phosphate, glucose 6-phosphate, inorganic phosphate, and lactate) with better time resolution, repeatability, and somewhat better precision.^{2–5} The drawbacks of MRS (the availability of expensive equipment and an inability to distinguish between muscle fibre types) are offset by the muscle biopsy technique.³ When MRS and muscle biopsy samples are obtained concurrently, the small amount of tissue obtained in the biopsy sample (50–80 mg muscle) does not need to be used to determine muscle glycogen concentration and can be used to assess other important metabolic indicators such as enzymatic activities. This complementary nature of MRS and muscle biopsy means that, when used in combination, they become a powerful

tool for optimising athletic training programmes. In such a programme, MRS samples obtained from individual athletes may be used to (a) monitor the effectiveness of different carbohydrate loading protocols, (b) optimise the efficiency of training schedules and avoid overtraining, (c) assess metabolic recovery from training sessions, and (d) measure the athlete's state of readiness to participate in an event. Ultimately MRS and biopsy measurements are indicators of an athlete's physical condition at a specific point in time (pre-season, mid-season, end of the season), and therefore are of great benefit in optimising an athlete's performance and minimising the risk of injury.

MRI is another non-invasive method that has the potential to be a powerful training tool, and it is much more universally available than MRS. Muscles that have actively participated in the performance of an exercise appear hyperintense on MR images.⁷ It is thought that this increase in MRI signal results from movement of fluid into the exercised muscle, brought about by increased metabolic activity in the muscle.^{8–9} Electromyography (EMG) measures neural activation as differences in electrical activity across the muscle membrane and has been used traditionally to measure muscle activity. It has the advantage that it is sensitive to small changes in electrical activity and it can detect the onset of neural fatigue.¹⁰ However, it cannot be used non-invasively to study deep muscles, and it can only study the muscles that it is set up to study.¹⁰ MRI can be used to study both surface muscles and deep muscles non-invasively, and may be a better indicator of how hard a muscle has worked.¹⁰ As with MRS and muscle biopsy, there is a great potential to use MRI and EMG in combination to optimise a training programme. Traditional MRI methods can be used to study an athlete's anatomy, making measurements such as heart chamber volumes (particularly left ventricle) and arterial development (measured as the arterial diameter of major arteries). Both of these variables are augmented by training and therefore are a measure of the degree of training of an athlete.¹¹ Functional MRI methods can be used to assess the muscle activation patterns that contribute to the complex biomechanical

movements involved in sports.¹⁰ This technology can be used to (a) identify muscles that are activated and (b) assess the extent of activation of each muscle relative to that muscle's maximum capacity to perform work.^{9, 10} Information obtained from functional MRI measurements can be used to compile individual databases of each athlete's muscle activation patterns when he or she is at peak performance. This information can be valuable if the athlete is injured or if there is a pronounced decline in peak performance. Functional MRI measurements of identical exercise obtained under such conditions, when compared with information from the athlete's database, could provide insight into injury induced changes in muscle activation patterns. Functional MRI may also be used to monitor an athlete's recovery from an injury. As with MRS, MRI provides a measure of an athlete's physical conditioning at a specific point in time.

In summary, it is possible that, by combining MRI and MRS with more traditional methods, we may create an organised training and evaluation tool capable of elevating human performance to a new level. At this level we would be able to (a) minimise instances of overtraining and therefore reduce overtraining injuries, (b) optimise event readiness thereby reducing injuries that are associated with fatigue during an event, and (c) optimise injury recovery programmes so as to reduce the incidence

of reinjury. These reductions could make a significant impact on sports related injuries in elite and professional athletes.

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Stretching before exercise: an evidence based approach

Clinicians are under increasing pressure to base their treatment of patients on research findings—that is, to practice evidence based medicine.¹ Although some authors argue that only research from human randomised clinical trials (RCTs) should be used to determine clinical management,² an alternative is to consider the study design (RCT, cohort, basic science, etc) as one of many variables, and that no evidence should be discarded a priori. In other words, the careful interpretation of all evidence is, and has always been, the real art of medicine.³ This editorial explores these concepts using the sport medicine example of promoting stretching before exercise to prevent injury. In summary, a previous critical review of both clinical and basic science literature suggested that such stretching would not prevent injury.⁴ This conclusion was subsequently supported by a large RCT published five months later.⁵ Had the review relied only on previous RCT data, or even RCT and cohort data, the conclusions would likely have been the opposite, and incorrect.

Was there ever any evidence to suggest that stretching before exercise prevents injury? In 1983 Ekstrand *et al*⁶ found that a group of elite soccer teams randomised to an intervention of warming up and stretching before exercise, leg guards, special shoes, taping ankles, controlled rehabilitation, education, and close supervision had 75% fewer injuries than the control group. There was one other RCT and a quasi-experimental study that also supported this conclusion,^{7, 8} both using at least warm up as a co-intervention.

Clinical evidence suggesting that stretching before exercise does not prevent injuries has also been reported. van Mechelen⁹ published an RCT showing that the intervention had no effect, but many subjects were non-compliant. If we look at “less strong evidence”, both Walter *et al*¹⁰ and

Macara *et al*¹¹ published cohort studies that suggested that stretching before exercise was not beneficial, and there have been several cross sectional studies as well.^{12, 13} Of course, there were significant limitations to all of these studies.

In summary, the RCTs could easily be interpreted to suggest a probable effect using strict evidence based medicine guidelines. The use of cohort studies may weaken the conclusion, but would be unlikely to reverse it. Understanding the basic scientific research allows one to put this clinical evidence into perspective and explain results that may appear contradictory.

Firstly, some people believe that a compliant muscle is less likely to be injured. From the basic science research, we find that an increase in tissue compliance due to temperature,¹⁴ immobilisation,¹⁵ or fatigue^{16, 17} is associated with a decreased ability to absorb energy. Although this is not the equivalent of stretching, no basic science research shows that an increase in compliance is associated with a greater ability to absorb energy. Secondly, most injuries are believed to occur during eccentric contractions,¹⁸ which can cause damage within the normal range of motion because of heterogeneity of sarcomere lengths.^{19–22} If injuries usually occur within the normal range of motion, why would an increased range of motion prevent injuries? Thirdly, even mild stretching can cause damage at the cytoskeletal level.²³ Fourthly, stretching somehow increases tolerance to pain—that is, it has an analgesic effect.^{24–26} It does not seem prudent to decrease one's tolerance to pain, possibly create some damage at the cytoskeletal level, and then exercise this damaged anaesthetised muscle. Of note, there is no basic science evidence to suggest that stretching would decrease injuries. Fifthly, there are some basic science data to suggest that a warm up may help to prevent

injuries.²⁷ Understanding these principles, we can now explain the apparent contradiction in the clinical literature. Re-examining the RCTs that support stretching before exercise, one finds that all of them included at least one other effective co-intervention—for example, warm up, leg guards, etc.⁶⁻⁸ Therefore it is not surprising that these RCTs found less injuries in the intervention group. On the other hand, the cohort studies,^{10,11} and the RCT by van Mechelen *et al*,⁹ controlled for these co-interventions in the analysis stage. Therefore, although formally a “weaker design”, the studies suggesting that stretching before exercise is not beneficial should be weighted as stronger because the analysis was more appropriate. However, this was only recognised because the basic science was reviewed.

In conclusion, the strength of any literature review can be gauged by its ability to predict what future research studies eventually show. The inclusion of all the evidence available led to a conclusion that was supported by a subsequent, well conducted, large RCT. Had the evidence in the review article⁴ been limited to only RCTs as proposed by some authors, the conclusion would have probably been different, and inaccurate. This may be one reason why many meta-analyses fail to predict the outcome of future large RCTs. Further, we must remember that much of medicine in general, and sport medicine in particular, is based on historical precedent. When historical precedents are based solely on hypotheses that have more recently been proved incorrect, the clinician must choose to (a) continue treatment on the basis of a known incorrect idea of pathophysiology or (b) change to a treatment based on current knowledge of pathophysiology and pathobiology. Of course, the potential side effects of any new treatment (likely to be unknown) must also be weighed against the potential side effects of the historical treatment (more likely to be known). The art, and even science, of medicine then becomes the ability to weigh all the available information at hand without discriminating a priori, and to be able to judge which is most appropriate for the patient seated across the desk.

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