The cause of the abdominal pain commonly referred to as ‘side stitch’, but more accurately known as exercise related transient abdominal pain (ETAP), has until recently attracted more speculation than research. Explanations for the pain have included diaphragmatic ischaemia,1 stress on peritoneal ligaments,2 and irritation of the parietal peritoneum.3 Although the exact cause of the pain remains to be elucidated, a neurogenic explanation has not appeared in the literature.

We present the case of an elite runner who, after a thoracic spine trauma, developed severe and recurrent episodes of ETAP which were relieved by localised treatment. As a follow up study, we made observations on 17 other runners who often experience episodes of ETAP and found that palpation of specific facet joints could reproduce symptoms of ETAP shortly after an episode of the pain had been relieved.

Case study

The patient was a 25 year old man who had been actively involved in competitive running for about 16 years. During this time, he claimed that he occasionally experienced mild symptoms of ETAP during training and competition, although they were not disruptive to performance. After trauma to the thoracic spine in a trampoline accident, he began to experience ETAP with greater frequency and severity. Subsequently he would develop ETAP often when walking and consistently when running. The presentation of the pain essentially forced him from competition. He had received advice from several medical practitioners before being referred to our clinic about two years after the trauma. The symptoms had been present and persistent when running was attempted for this time period.

On questioning, it became apparent that several conditions that stress the spine exacerbated the experience of ETAP. For example, torso hyperextension coupled with the jolting movement of downhill running provoked the pain. In addition, tight erector spinae after a surfing session seemed to increase the likelihood of experiencing ETAP.

On assessment, vertebral hypomobility was observed at the T7–T8 level, presumably as a result of the trampoline accident. Accompanying this was hypermobility of the T8–T9 joint. Palpation of the left T8–T9 facet joint resulted in reproduction of sub-costal pain consistent with the patient’s exercise induced experience of ETAP. Further, the site of the pain reproduction was consistent with the dermatomal distribution arising from the T8–T9 level. Palpation of the joints above and below the joint caused no pain referral.

Localised treatment aimed at mobilising the T7–T8 joint was administered in the clinic, and the patient was given mobilising exercises to perform. After about four weeks of treatment, he reported a reduction in the symptoms of ETAP. He has since resumed competitive running.

Further observations

In response to the presentation of this case, we recruited 17 other runners who spasmodically experienced ETAP during training and competition. Ethical clearance was obtained from the Avondale College human research ethics committee. The subjects exercised on a treadmill after a meal until ETAP developed, at which time the exercise session was terminated. After the pain had completely disappeared, the subjects lay in a prone position and were submitted to a spinal assessment, in which facet palpation was performed along the length of the thoracic region. The subjects were not informed of the intent of the study but were asked to volunteer any symptoms of pain or discomfort that arose.

In eight cases (47%), the exact previous experience of ETAP was reproduced through palpation of the thoracic spine at various levels from T8 to T12. In another six cases (35%), the subjects reported lateral and anterior pain referral towards the site of ETAP as a result of the palpation. The longitudinal site of ETAP along the abdomen correlated significantly with the level of the thoracic spine producing symptoms on palpation. This result was observed when only the subjects reporting exact reproduction were analysed (r = 0.78, p<0.05) and also when all subjects reporting any form of pain referral were included in the analysis (r = 0.63, p<0.05). Further, the level of the spine producing symptoms on palpation and the site of ETAP were consistent with dermatomal distribution.

Discussion

There has been little or no mention in the literature of the possible role of the spine in the cause of ETAP, although there are several observations that indicate that its contribution may be noteworthy. Firstly, in 1941 Capps’ anecdotally observed that people with an increased kypholordotic postural alignment showed most susceptibility to the pain. To investigate this further, we recently confirmed in a study involving a comprehensive postural assessment of over 150 subjects that increased kyphosis was provocative of ETAP.4 This observation is interesting given that intercostal nerves arising from the thoracic region supply the anterior abdomen. In addition, it has been suggested that “jolting” of the torso is primarily responsible for evoking the pain,5 and this action is known to result in intervertebral compression.6 As a side issue, we documented that horse riding is one of the sporting activities most likely to induce ETAP.7 It has also been asserted that an episode of ETAP can be relieved through body inversion.8 Finally, we have argued that the characteristics of the pain, being well localised, lateral, and mostly sharp, are consistent with somatic rather than visceral abdominal pain.9 An explanation for the pain involving the intercostal nerve would account for these pain characteristics.

The extent to which the thoracic intercostal nerves may contribute to the experience of ETAP is worthy of further investigation. It seems plausible that, in some cases mechanical compression of the nerve root may refer pain distally, resulting in abdominal pain. Alternatively, irritation of the nerve may sensitize it to stimuli such that the threshold required for activation is lessened. Hence, in this study, palpation after the pain had been relieved may have allowed tissues innervated by the intercostal nerves, such as the abdominal musculature or parietal peritoneum, to recreate sensations of pain.

Although the significance of the observations documented in this report remains to be fully elucidated, they do indicate that the spine may play a role in the genesis of ETAP. Further studies are required to determine the extent to which the spinal nerves are involved and the potential benefits of improving spinal integrity in ETAP sufferers. Certainly, the spine should be considered when treating patients with recurrent symptoms of ETAP.

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References


Science versus opinion

Dr Berger recently responded1 to my article in the British Journal of Sports Medicine entitled: Berger in retrospect: effect of varied weight training programmes on strength.2 Dr Berger presented no scientific evidence to support his opinion on single versus multiple sets, cited references that were irrelevant to the topic, and challenged the reported statistical analyses.

Dr Berger claimed that most athletic and therapeutic professionals have added credence and support to the belief that multiple sets are required for optimal gains in strength because they use multiple sets in their practice and research (p 372).1 However, just

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using multiple sets in practice is not evidence that multiple sets are superior to a single set; it merely perpetuates an unsubstantiated belief. In my retrospective study of 57 studies that reported no significant difference in strength gains as a result of performing a greater number of sets, Dr Berger failed to cite a single study to support his opinion on the benefits of multiple sets. In my study, Dr Berger’s own follow up studies, which are described in my retrospect, failed to support his opinion—that is, a greater number of sets did not result in a significantly greater strength gain in either of his follow up studies.

Dr Berger claimed that some early studies compared different weight training programs and cited five studies. However, these studies (described below) are irrelevant to the issue of single versus multiple set resistance training. Delorme et al (196) treated eight young men and 11 women with poliomyelitis-weakened and atrophied knee extensor muscles. Their goal was to increase muscular strength and hypertrophy. The patients performed three sets of 10 repetitions (intensity not reported) of knee extension and leg press exercises four times per week for one to four months. Isometric force increased 1–200%. No statistical pre to post training analysis was reported. All the patients performed three sets of each exercise; consequently, there was no comparison of single versus multiple sets. Krusen randomly assigned military aged male (n = 23) and female (n = 1) patients with poliomyelitis to one of two knee extensor strength training protocols (sessions per week not reported). One group performed five repetitions with 25% 5RM, five repetitions with 50% 5RM, and five repetitions with 75% 5RM twice a day for 3–23 weeks. The other group performed five repetitions with 100% 5RM, five repetitions with 75% 5RM, and five repetitions with 50% 5RM, with limited range of motion on the last two sets twice a day for 3–19 weeks. Krusen claimed that, to rule out the difference in intensity, 13 subjects in each group were matched for strength and compared after five weeks of training. Knee extension 1RM increased in both respective groups (74.5% and 60.0%), with no significant differences in the groups in strength gains. A comparison of single versus multiple sets was not possible because both groups performed three sets of exercise. The actual comparison was between a low intensity protocol (five repetitions with 75% 5RM) and high intensity protocol (five repetitions with 100% 5RM), which resulted in similar strength gains.

McMorris and Elkins trained the right triceps of nine healthy men and three women (20–29 years of age) with four sets of exercise five times a week for 12 weeks. One group performed four sets of 10 repetitions with 25%, 50%, 75%, and 100% 10RM in ascending order, while the other group performed four sets of 10 repetitions with 100%, 75%, 50%, and 25% 10RM in descending order. McMorris and Elkins reported a 5.5% greater gain in strength for the group that performed the descending order. There was no statistical analysis reported. Because all the patients performed four sets of each exercise, no comparison of single versus multiple sets was possible. Krusen and Houtz reported their observations of what they described as 620 experiments in 17 male and female competitive athletes. Wrist flexion and extension strength varied from 3 to 40 sets of 25 repetitions with 25–100% 25RM three to five times a week for 8–20 weeks. There was no statistical analysis reported, and more importantly, no comparison of single versus multiple sets.

McGovern and Luscombe reported on a two part investigation. In part 1, 10 healthy men (20–30 years of age) were divided into two groups (A and B). Group A performed one warm up set of unilateral quadriceps exercise for five repetitions with 50% 10RM, followed by one set of 10 repetitions with the 10RM. Group B performed one range of motion warm up set (no resistance), followed by three sets of 10 repetitions with 100%, 75%, and 50% 10RM, respectively, and one minute rest between sets. A third group (C) of five male orthopaedic patients (20–30 years of age) performed three sets of 10 repetitions with 50%, 75%, and 100% 10RM respectively. The groups trained five times a week for three weeks. No pre to post training or between group statistical analysis was reported. However, the authors stated that the increase in 1RM was similar for groups A, B, and C (7.7, 8.7, and 7.7 kg respectively) in part 1. In part 2, male and female orthopaedic patients (20–30 years of age) trained five times a week for four weeks. Half the patients performed one set of 10RM and the other half performed three sets of 10 repetitions with 50%, 75%, and 100% 10RM. The two groups of patients showed similar gains in 10RM (5.8 and 6.0 kg). McGovern and Luscombe concluded that the two training protocols produced similar strength gains, with a time saving advantage to the single set group.

The study by McGovern and Luscombe was the only one of the five studies cited by Dr Berger that compared single versus multiple sets, and they reported similar strength gains as a result of one set or three sets of exercise.

Dr Berger claimed that the medical applications of strength training for physical therapy have been significantly increased by multiple sets, and that one set was the exception (p 372). In 1951, Delorme and Watkins specifically stated that increasing the resistance after each of three sets provided an advantage for warming up the muscles in patients suffering from poliomyelitis, but probably did not contribute to the increase in muscular strength. They concluded that performing one set or three sets of 10RM would produce similar increases in strength (pp 27–8). Contrary to Dr Berger’s claim that one set was an exception, and contrary to Dr Berger’s study where his nine groups performed one, two, or three maximal sets, Delorme et al, Delorme and Elkins, and McGovern and Luscombe actually trained all their subjects with only one maximal set.

Dr Berger claimed that he was not the genesis of an unsubstantiated belief (p 372). In fact, I noted in my retrospect that Dr Berger’s study—not he personally—was perhaps the genesis of the unsubstantiated belief that multiple sets are superior to a single set for strength gains (p 319). The flow chart (fig 1, p 320) in my retrospective clearly shows that Dr Berger’s study was the only strength training study cited.

Dr Berger stated that therapists and coaches are flinging down the gauntlet in support of multiple sets (p 372). Apparently, Dr Berger is claiming that therapists and coaches are challenging others to show that multiple sets are not superior. However, a primary tenet of science is that the burden of proof rests entirely on the proponent of the claim. If Dr Berger and the therapists and coaches he refers to claim that multiple sets are superior, then the entire burden of proof is on them to support that claim with peer reviewed strength training studies.

Dr Berger claimed that in 1996, practitioners in the field would have confronted him to express their disagreement (p 372). It is not my fault that the reviewers and the editor of Research Quarterly initially failed as gatekeepers and that in the last 40 years other scientists in the field have not challenged Dr Berger’s medical applications, statistical analysis, and conflicting conclusions.

Dr Berger noted that Arthur Jones, who developed and sold Nautilus machines, was a proponent of single sets and that he has never presented any acrobatic evidence to support his belief (p 373). Mr Jones was never mentioned or cited in my retrospect because Mr. Jones’ beliefs, or anyone who agrees or disagrees with those beliefs, are completely irrelevant to Dr Berger’s study, my retrospect, and Dr Berger’s ‘response’ to my retrospective.

Dr Berger claimed that the data in tables 1, 2, and 3 of my retrospect were inappropriately cited according to a different protocol (p 373). In fact, it was Dr Berger who used the statistical analyses that I reported in my retrospect. The data in table 1 (p 320) of my retrospect, which show the increase in 1RM bench press strength for each of Dr Berger’s nine groups, were gleaned directly from Dr Berger’s table 1 (p 172). The data in table 2 (p 320) of my retrospect, which show the groups that differed significantly at the 0.05 level of probability, were from Dr Berger’s table 4 (p 176). When I questioned Dr Berger about the three typographical errors in his table 4, he said that he was not aware of these errors and claimed that no one else had ever questioned them before our telephone conversation in 1998. The correct data are shown in table 2 of my retrospective (p 320). Table 3 (p 321) in the retrospective shows that for the groups who performed the same number of repetitions (two, six, or 10), seven out of nine of Dr Berger’s comparisons showed no significant difference in the magnitude of strength gain. For example, the strength gain in the 3 × 6 group was significantly greater than the 2 × 6 group, but not greater than the 3 × 10 group. 3 × 2 was significantly greater than 2 × 2, but not significantly greater than 1 × 2. The data are all from Dr Berger’s table 4 (p 176).

Dr Berger claimed that comparisons between his nine groups nullified his previous study (p 373). I did not make any comparisons between Dr Berger’s groups. However, I did accurately note in my retrospective exactly what Dr Berger reported in his study. Dr Berger’s subsequent admission that he erroneously made the comparisons among the nine groups in table 4 of his study (p 373) nullifies his previous claim.
Dr Berger claimed that when he compared the main effects among one, two, or three sets, and among two, six, or 10 repetitions, there were six repetitions resulting in the greatest improvement in strength (p 373). In his Results section, Dr Berger stated that analysis of covariance was used to test for a significant interaction between sets and repetitions—that is, whether one, two, or three sets might be better in combination with two, six, or 10 repetitions. The F ratio was not significant at any period of training (p 178). Dr Berger also reported in Conclusion 6 that training with one, two, or three sets was not more effective in improving strength than other combinations of sets and repititions (p 181). These antithetical statements were reported in my retrospect. Dr Berger failed to address them in his response or in our personal correspondence (1998). Dr Berger noted that he spoke with me about my misuse of statistics and that he suggested that I consult a statistician (p 373). I followed his advice and consulted two statisticians before submitting my retrospective. Their opinion was that Dr Berger should not have made the 36 individual comparisons that he reported because the multiple comparisons increased the chance of a type I error, which could have led to rejection of the null hypothesis when the null hypothesis was actually true. Dr Berger stated that he still unequivocally supports multiple sets over a single set for optimising strength (p 373). Dr Berger is perfectly entitled to his opinion, but it has nothing to do with supporting his belief with scientific evidence. As noted in my retrospect, Dr Berger expressed his opinion about the superiority of multiple sets in our personal correspondence (1998), and in at least five publications. The only study he cited to substantiate his opinion was his 1962 study. Dr Berger neglected to cite his own follow up studies that failed to support his opinion. Dr Berger suggested that I conduct research in the hope of gaining support for my position, although he does not define my position (p 373). In fact, my position is simply that if someone in the scientific community makes a claim about anything, they should be held accountable to support that claim with peer reviewed scientific evidence. As noted in the flow chart (p 320) of my retrospective, most of the physiologists who recommend multiple sets of each exercise cite each other’s opinions, reviews and books, cite nothing at all, or they cite Dr Berger’s study. Because I am not making a claim about the superiority of one strength training protocol over another, I am under no obligation to conduct research. I am simply challenging the widely held belief that multiple sets are superior to a single set, and in turn, challenging the apparent genesis of that belief—Dr Berger’s study.

References

Science versus opinion
Dr Berger recently responded to my article in the British Journal of Sports Medicine entitled: Berger in retrospect: effect of varied weight training programs on strength, which is a critical analysis of one of Dr Berger’s early studies. In his response, Dr Berger presented no scientific evidence to support his opinion on single versus multiple sets. He cited references that were irrelevant to the topic, and challenged the reported statistical analyses. I have responded to each of Dr Berger’s claims in a brief review which differentiates the scientific evidence from Dr Berger’s opinion.

References
4 Carpinelli RN. Science versus opinion. www.bjsm.bmj.com/journals/cgi/letters.
and prognosis of sporting injuries. Therapeutic image guided intervention using fluoroscopy, CT and ultrasound will also be demonstrated.

13 RCR category one CME credits have been awarded.

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BASEM Conference 2004
14–17 October 2004, Belfast, UK
Main themes: Overuse Sports Injuries and Muscle Injuries. Keynote speakers include: Chris Bradshaw, Medical Director, Olympic Park Medical Centre, Melbourne and Kim Bennell, Assistant Professor, School of Physiotherapy, Melbourne University.
Further details: Email: fionnuala.sayers@greenpark.n-i.nhs.uk

2nd International Ankle Symposium
15–16 October 2004, Newark, DE, USA
The meeting will examine ankle instability and other related ankle pathologies from a multidisciplinary perspective. Attendees will include clinicians and scholars from the disciplines of orthopedics, podiatry, physical therapy, athletic training, biomechanics, and sports medicine. This conference aims to build on the success of the inaugural symposium held in Ulm, Germany in December 2000. Emphasis will be on oral and poster presentations of original research along with selected plenary presentations.
The deadline for abstract submissions is July 1 2004.
Further details: Jay Hertel, PhD, ATC, FACSM, Conference Co-Chair, Penn State University, email: jnh3@psu.edu, or Thomas Kaminiski, PhD, ATC, FACSM, Conference Co-Chair and Host, University of Delaware, email: kaminiski@udel.edu. Website: http://www.udel.edu/ias/

1st World Congress on Sports Injury Prevention
23–25 June 2005, Oslo, Norway
This congress will provide the world’s leading sports medicine experts with an opportunity to present their work to an international audience made up of physicians, therapists, scientists, and coaches. The congress will present scientific information on sports injury epidemiology, risk factors, injury mechanisms and injury prevention methods with a multidisciplinary perspective. Panel discussions will conclude symposia in key areas providing recommendations to address the prevention issue in relation to particular injuries and sports.
Further details: Oslo Sports Trauma Research Centre and Department of Sports Medicine, University of Sport and Physical Education, Sognsvænken 220, 0806 Oslo, Norway. Email: 2005congress@nih.no; website: www.ostrc.no