Biomechanical overload syndrome: defining a new diagnosis

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Chronic exertional compartment syndrome (CECS) was first described in 1956,1 but little research has been performed since then to confirm the pathological physiology. An assumption is made that elevated subfascial or intramuscular pressure during exercise causes tissue hypoxia and subsequent ischemic pain due to decreased blood flow.2 To date, no conclusive evidence exists to demonstrate cellular hypoxic damage or decreased capillary perfusion.3 Further supposition is made regarding muscle hypertrophy, reduced compartment volume due to a decreased fascial compliance,4 and shorter periods of muscle relaxation as the underlying pathophysiology of CECS.

There are many questions over whether the technique of intracompartmental pressure measurement is reliable. Examination of the widely accepted diagnostic criteria published in the seminal paper by Pedowitz et al5 reveals significant flaws, as the CECS and non-CECS groups were preselected by their differences in intramuscular pressure. We have also demonstrated significant overlap of the published diagnostic criteria for CECS with the published normative data.6 Furthermore, intramuscular pressure measurement varies considerably with the depth of the catheter tip, the means of measurement and the mode of exercise. It is also important that the criteria presented are only applicable to the anterior compartment. CECS is also reported as being diagnosed in the deep posterior and peroneal compartments of the leg,7 the foot8 and the forearm,9 despite diagnostic pressure criteria never having been established in these other myofascial compartments. What is undeniable however is that exertional lower-limb symptoms localised to the anterior compartment as pain develops. It follows that fatigue combined with poor running biomechanics may cause the dorsiflexors to become rapidly overloaded. If the load on the dorsiflexors is further increased by extrinsic factors such as load-carrying, heavy footwear, gradient and increased training load, a gradual onset of exertional symptoms may result. Tightness, cramping pain and engorged muscles are all commonly described symptoms of those referred with anterior CECS. Eccentric contractions of the anterior leg compartment have, in the short term, been associated with an increase in running-induced metabolic fatigue, compared with the gastrocnemius.10

We have consistently observed, in military personnel referred with anterior compartment pain, prolonged ankle dorsiflexion and reduced heel lift during swing phase with excessive dorsiflexion at heel strike, reduced ankle plantarflexion at toe-off and persistent ankle dorsiflexion and toe extension at mid-stance. Within minutes of initiating running, the patient develops an audible ‘slapping’ of the foot at heel strike. These observations are consistent with repeated and prolonged inner range tibialis anterior contraction, which may therefore result in early onset of fatigue and the development of cramp-like symptoms. Perhaps this is why many patients express the desire to passively stretch the anterior compartment as pain develops. It follows that fatigue combined with poor running biomechanics may cause the dorsiflexors to become rapidly overloaded. If the load on the dorsiflexors is further increased by extrinsic factors such as load-carrying, heavy footwear, gradient and increased training load, a gradual onset of exertional symptoms may result. Tightness, cramping pain and engorged muscles are all commonly described symptoms of those referred with anterior CECS. Eccentric contractions of the anterior leg compartment have, in the short term, been associated with an increase in intracompartmental pressure; however, there is currently no evidence of a direct association between this rise in compartment pressure and the pain and reduced muscle function described in chronic anterior compartment syndrome.11 However, Kirby and McDermott12 have confirmed reduction in anterior compartment pressures with forefoot running and Diebal13 showed improvements in pain and function with changing from a heel strike to forefoot strike in patients with CECS.

The same principles can be applied to other compartments of the leg in which CECS has been
described. Tibialis posterior lies within the deep posterior compartment. It is thought to assist in restabilising the foot at mid-stance after maximal pronation has occurred. Provided the foot has an effective windlass mechanism the load on tibialis posterior should be minimal. If, however, altered biomechanical factors reduce the effectiveness of the windlass mechanism there may be excessive eccentric load on this muscle and a deep posterior pain may result. A single case report supports this conclusion with a suggestion of forefoot running as a causative factor in the development of postero-medial shin pain.

The mechanism of pain and muscle engorgement may be related to abnormal firing of α motor neurons due to miscommunication with the muscle spindle and the Golgi tendon organ. Local muscle fatigue has been shown to be responsible for increased muscle spindle and decreased Golgi tendon organ afferent activity, but as yet this has not been demonstrated conclusively. It also cannot be discounted that exercise-related leg pain may be fascial in origin. Irregularity of the fascial collagen has been observed in subjects with long-term symptom duration and the presence of calctonin gene-related peptide and substance P in free nerve endings in fascia are identifiable causes of fascial pain in its own right.

Muscle overuse syndromes are not new. They are well described in the literature, and significantly in musicians and office workers (occupational overuse syndrome) and there is a clear synergy with the predisposing factors in repetitive exercise: increasing frequency and the intensity or load of work and practice; and, altered limb biomechanics alongside limited rehabilitative intervention. We believe that in patients with exertional leg pain related to the myofascial compartments we are simply observing a phenomenon seen commonly in other patient groups; that of muscle overload. As the aetiology in these patients is biomechanical we have described their condition as a ‘biomechanical overload syndrome’ (BOS).

Freed from the restrictions of the compartment pressure model we have managed our patients with anterior symptoms by altering their running gait characteristics to reduce the load on the tibialis anterior. As foot strike patterns have been associated with injury rates in runners, and electromyogram intensity of the tibialis anterior at heel strike is higher when wearing shoes compared with running barefoot, it seems logical to promote a mid-foot landing rather than the heel-strike pattern commonly observed. Foot inclination angle at initial contact also decreases as step rate increases, and so an increased cadence of 5–10% was also encouraged. Other gait adjustments were made according to individual assessment, such as reducing the vertical tibial angle at foot strike, promotion of a smooth, gait pattern, promoting a more anterior centre of mass and shortening stride length. Alterations in the patients’ running gait have been supported by an individualised gait rehabilitation programme based around return to running and improved lower-limb conditioning. Follow-up assessment with 2D kinematics at the 3-month stage confirmed that patients had retained their new running form and a 70% success rate in resolution of symptoms was measured using a repeat of the PCT and an individuals employability using the functional activity assessment (FAA) score and Joint Medical Employment Standard (JMES) score. The success in the maintenance of these changes and the resolution of symptoms lead the authors to believe that BOS defines the exertional compartment pain seen in running and that there is clear evidence that intracompartmental pressure measurement should no longer be considered a valid diagnostic tool for CECS.

Further studies to define the kinematic changes in running technique, alongside resolution of symptoms, will be an important step forward in alleviating suffering, reducing surgical intervention and maximising return to sport. Care should be taken in recommending a surgical intervention where the pathophysiological and diagnostic evidence for surgery are not clearly defined.

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