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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 ischaemic 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 al*[5] 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 foot[8] 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 myofacial compartments are commonly reported in elite and recreational athletes,[10] military personnel,[11 12] and non-athletes alike,[13] and that CECS is included in the differential diagnosis.

As a tertiary referral centre for exertional leg pain, we have conducted large numbers (c.100/year) of intracompartmental pressure measurements, often with subsequent referral for fasciotomy.[6] While short-term outcome following fasciotomy reflected published data[14 15] we have found long-term outcome (>12 months) to be disappointing, using objective measures.[16] Both the previously reported groups used athletes or adolescents as subjects and may differ in that the 'return to play' criteria were less objective, which may explain the differences in outcome.

Biomechanical factors have been shown to improve running economy.[17] In particular stride length,[18] ground contact time, vertical oscillation and lower extremity angles all have an effect on running efficiency. Despite this, recreational athletes and military recruits rarely receive training in running technique, either with verbal cues, video analysis or feedback as running is assumed to be a natural skill that man has acquired over several millennia.[19]

During walking gait, tibialis anterior dorsiflexes the ankle concentrically to provide foot clearance during swing phase, and isometrically (with lengthening of the tendon)[20] to control the lowering of the forefoot during the first part of stance; this is assisted by the long-toe extensors (extensor hallucis longus, extensor digitorum longus) and peroneus tertius. During running gait, both the tibialis anterior and gastrocnemius have a high degree of preactivation prior to foot strike.[21] Tibialis anterior activity decreases more rapidly during running-induced metabolic fatigue, compared with the gastrocnemius.[22]

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.[23] However, Kirby and McDermott[24] have confirmed reduction in anterior compartment pressures with forefoot running and Diebal[25] 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

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described. Tibialis posterior lies within the deep posterior compartment. It is thought to assist in restabilising the foot at midstance after maximal pronation has occurred.[20] Provided the foot has an effective windlass mechanism the load on tibialis posterior should be minimal.[26] 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[27] supports this conclusion with a suggestion of forefoot running as a causative factor in the development of posteromedial 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,[28] 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.[29] and the presence of calcitonin gene-related peptide and substance P in free nerve endings in fascia[30] are an identifiable cause of fascial pain in its own right.

Muscle overuse syndromes are not new. They are well described in the literature,[31 32] 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,[33] and electromyogram intensity of the tibialis anterior at heel strike is higher when wearing shoes compared with running barefoot,[32] 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,[34 35] 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 conditioning programme of the lower-limb kinetic chain.

We have applied the same reasoning to other exertional lower-limb problems such as medial tibial stress syndrome (MTSS) and deep-posterior CECS. By viewing these conditions with the new paradigm of BOS we have sought to address biomechanical deficiencies in order to reduce the load on the tissues and structures thought to be responsible for the pain experienced in these exertional lower-limb conditions. In the case of MTSS, this reduction in load may be sufficient to promote repair while still allowing for continuation of sport-specific training.

We have developed a one-week inpatient 'running re-education' programme where patients with BOS undergo initial assessment with the Provocation Challenge Test (PCT) and assessment of two-dimensional (2D) running kinematics. The PCT is conducted on a treadmill wearing high combat boots carrying initially 15 kg load. Subjects start at 6.5 km/h for 5 min, then the incline is increased to 5% at 6.5 km/h for a further 5 min, following which the subject then removes the Bergen and the speed is increased to 11 km/h for the final 5 min period. Subjects continue until they cannot carry on due to pain and then the score is the time sustained. Patients undergo a programme of running coaching, dynamic core and gluteal strengthening, podiatric input and hip, knee ankle triple flexion alignment improvement, supported by delivery of an education package. The inpatient course is followed by a 3-month 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.

Contributor AFM, AR, JF and DH contributed to both the development concepts of the redefined diagnosis, the clinical care of the patients who were responsible for underpinning this work and in the writing of the textual submission. AFM wrote the initial draft manuscript and JF, AR and DH critiqued and re-edited the piece making substantial individual contributions. AFM is the Guarantor of the piece.

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