LETTERS TO THE EDITOR

Sternal fracture in a female army officer cadet

EDITOR,—We were interested to read the recent case report of a manubrium sterni stress fracture that had followed a period of strenuous abdominal exercises.1 We describe a similar injury, in which an acute sternal fracture was sustained in the absence of any direct trauma.

A 23 year old female army officer cadet presented during her seventh week of training. While performing “reverse triceps dips”—a gymnastic circuit exercise in which the upper body is raised and lowered by the arms with the hands placed on a low bench behind one’s back and the feet on the floor in front (fig 1)—she experienced a sudden, central chest pain associated with an audible crack. A radiograph taken the same day (fig 2) showed an acute transverse fracture through the upper sternum. Further inquiry, clinical examination, and investigations (including CT and DEXA scans) failed to identify any evidence of localised or systemic bone disease. It was 15 months before the patient was symptom-free and returned to training. The mechanism of this injury (being “intrinsically” or “non-impact”) and the absence of any identifiable underlying cause, make it most unusual, though remarkably similar to that reported by Robertsen et al.1 Interestingly, both fractures occurred as acute events, with no preceding chest discomfort to suggest that a stress fracture might be developing. As both these injuries occurred in otherwise healthy individuals, the possibility of an acute sternal fracture should be considered in anyone developing acute anterior chest pain and tenderness after repeated, strenuous upper body gymnasium exercises.

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Exerci$$s$$ induced leg pain

EDITOR,—We read with interest the paper “Exercise induced leg pain—chronic compartment syndrome”,2 highlighting the need for functional activities to raise intracompartmental pressure. When reporting such cases it is critical to emphasise the major differences that exist between investigators in their choice of pressure recording systems, the exercise protocols, and, most importantly, the intramuscular pressures measured and the pressure criteria for confirming the diagnosis of chronic compartment syndrome.

We would dispute the un referenced statement that “Chronic compartment syndrome is the most common cause of exercise induced leg pain”. The true incidence of CCS in an exercising population is difficult to assess as the literature is incomplete for population based studies. However Styf3 found that even in a group of patients referred for investigation of chronic exercise induced pain in the anterior compartment of the lower leg, and therefore in whom medial tibial stress syndrome was excluded, CCS was only diagnosed in 27% of patients. If medial tibial stress syndrome were included it would form the dominant subgroup.

We would not regard the rise in pressure using the step aerobic routine described as dramatic or diagnostically significant. The pressure changes after exercise are not clearly presented. There are no figures given in the text. Readers are left to interpret the data for themselves from graphs where the axes are not labelled. Furthermore, it is extremely difficult to calculate mean pressures from such pulsatile recordings. No mean pressure during exercise data are given, but the difference between the two protocols seems to be merely the difference between 38 mmHg and 42 mmHg.

We do not feel there is a standard “normal diagnosis of CCS”. The authors correctly highlight the debate over the appropriate pressure parameters but do not point out that even among those authors who support the use of mean muscle pressure during exercise threshold, pressures range from 50 mmHg to in excess of 85 mmHg.1

The authors do not comment on the intensity of the step aerobic routine relative to the “standard” protocol or whether either of the exercise protocols provoked the patient’s symptoms. A number of investigators make this a cornerstone of their exercise protocols.

The potential benefits of a sports specific exercise protocol need further evaluation.

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Turf-toe: super league toe

EDITOR,—It has been over 20 years since sports medicine literature first described turf-toe syndrome, a plantar capsule ligament sprain of the first metatarsophalangeal (MTP) joint.1 In the United States an increased prevalence of the condition has been primarily related to the expanded use of artificial playing surfaces at the expense of natural grass in such sports as American Football, baseball, and soccer. It has further been linked to the specialist footwear worn to compete on the surface, which is of softer material and a lighter weight than traditionally worn on grass.

In the first season of a summer rugby league we encountered three incidences of capsular injury to the first MTP joint. Technically speaking they cannot be described as turf-toe, as all of the injuries took place while playing on grass, but there are certain similarities between the two. Firstly, there was a change in playing surface from softer winter grounds to harder summer surfaces. Secondly, the players’ boots changed from leather with longer aluminum studs to softer material with moulded studs. Although the overall prevalence may be small, the associated factors seem to be an alteration in the playing surface, and a search for footwear to provide more traction.

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