It is conceivable that the onset of pneumomediastinum occurred during the episode of asthma on the night before presentation; however, the mild nature of this attack suggests otherwise. This attack was probably a factor in increasing the amount of mediastinal air following the initial leak initiated by the patients unusual exertion.

This case represents the first report of pneumomediastinum occurring as a result of surf lifesaving. Of importance is the associated history of mild chronic asthma and this case also represents the first description of this complication of asthma in an asthmatic athlete. The importance of impeccable control of asthma in the athlete—and in particular the monitoring of lung function before activities involving straining and the Valsalva manoeuvre—and the correct use of prophylactic medication is highlighted.

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Exercise induced leg pain—chronic compartment syndrome. Is the increase in intra-compartment pressure exercise specific?

Nat Padhiar, John B King

Abstract

Intra-compartment pressure studies remain the main investigative method in diagnosing chronic compartment syndrome (CCS). Standard exercise protocols have been used to cause the raise in pressure measured in the laboratories. This case suggests that CCS cannot be excluded without the specific sports activity being used to raise the intracompartmental pressure.

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Key terms: chronic compartment syndrome; intra-compartmental pressure; exercise

Exercise induced leg pain is a common

Figure 1 Transverse section through the left leg showing the four compartments.

problem encountered by young athletes after an increase in activity or at the start of the season.¹ The terms shin splints² or freshers leg³ are often used to describe the condition. These terms are non-specific and are falling out of favour. The pivotal symptom is pain, which occurs on exercise and is relieved by a variable period of rest. The cause of pain will usually fall into one of the following four categories:

- (1) Pain of bony origin, for example focal stress fracture or diffuse micro-stress fractures.⁴⁻⁶
- (2) Pain of osteofascial origin, for example periostitis and medial tibial stress syndrome.^{7,8}

Figure 2 Pressure change following jogging on the spot.

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Figure 3 Pressure change following step aerobic routine.

- (3) Pain of muscular origin, for example chronic compartment syndrome.⁹⁻¹¹
- (4) Pain of nerve compression, for example superficial peroneal nerve compression.¹²

Chronic compartment syndrome is the most common cause, particularly affecting the anterior and deep posterior compartments of the leg (fig 1). The condition is characterised by an increase in intra-compartmental pressure, resulting in pain¹³ which may well be localised.

Case report

A 32 year old Caucasian woman presented with activity related pain affecting the anterior compartment of the right leg. She was a step aerobic instructor with average of six high impact classes a week. Resting and dynamic pressure studies were performed using a slit catheter¹⁴ and a Medex pressure transducer as described by Barnes *et al.*¹⁵

A "standard" exercise protocol (jogging on the spot for 60 seconds with 30 seconds rest) was used and the pressure remained within normal limits (<40 mm Hg)¹⁵ (fig 2). A study using a step aerobic routine showed a dramatic rise in the pressure (>40 mm Hg),¹⁵ the normal diagnosis of CCS (fig 3).

After failed conservative management she underwent superficial fasciotomy of the anterior compartment (fig 4). She returned to her sport 12 weeks after the operation.

Figure 4 Superficial fasciotomy.

Discussion

Chronic compartment syndrome of the leg was first described by Mavor in 1956⁹ and is characterised by exercise induced pain, swelling, and impaired muscle function.¹³ Increase in intra-compartmental pressure on exercise is regarded as diagnostic but there is a variation in parameters used by various authors. French and Price¹⁰ and Wallenstein¹⁶ considered raised intra-compartmental pressure at rest after exercise to be crucial; Puranon and Alavaikko¹¹ cite raised intra-compartmental pressure during exercise; McDermot *et al.*¹⁷ claim muscle contraction pressure to be critical; and Styf *et al.*¹⁸ regarded increase in muscle relaxation pressure to be the important variable.

Exercise protocol has also varied with individual researchers. Styf *et al.*¹⁸ used the foot and shoe attached to a device loaded with 4-6 kg and linked it to an ergometer. Patients were asked to dorsiflex both feet once a second for five minutes. Logan *et al.*¹⁹ and Rorabeck *et al.*²⁰ used a treadmill with varying speeds to exercise their subjects. Allen and Barnes¹ used jogging on the spot for 60 seconds with 30 seconds rest, which was repeated three times and results averaged.

CONCLUSION

Despite variations in detail, an exercise protocol is generally used in an attempt to demonstrate pressure changes in the compartment affected by CCS. Our case suggests that changes in pressure may only come about when a specific activity is used, and adherence to a set protocol may not reveal some cases.

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Late deterioration after decompression illness affecting the spinal cord

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Abstract

A former amateur diver presented with a progressive paraparesis. Thirteen years previously he had developed acute spinal cord dysfunction immediately after dry hyperbaric exposure. He had completely recovered motor function in the intervening period. No alternative reason for the later decline emerged from detailed investigation.

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Key terms: decompression illness; late paraparesis

Dysbarism (decompression illness) is one of the most common identifiable causes of nontraumatic ischaemic myelopathy.¹ Histological examination of the spinal cord of divers who have suffered dysbarism can reveal widespread damage, with hyalinised blood vessels and demyelination, even in those who have subsequently recovered. There may also be degeneration of nerve fibres in the dorsal and lateral spinal columns, with gliosis and white matter atrophy.²⁻⁶

Case report

A 37 year old Caucasian man presented with weakness and spasticity of his legs, worsening since the onset six months earlier.

Thirteen years previously he had suffered a spinal cord decompression illness. At no other time had he had any significant disease or diving accident. The decompression illness was experienced following a closely monitored hyperbaric exposure to a maximum of 6.5 atmospheres. He shared the compression chamber with four other divers, none of whom was adversely affected. The chamber pressure was controlled from outside following the standard national guidelines then available (1978).

Thirty minutes after recompression he experienced paraesthesia in his right buttock. After one hour he had developed weakness of both legs (MRC grade 3/5). He managed to return to the chamber for therapeutic recompression within 2½ hours, which was at 6 atmospheres

for 5½ hours. During this period his decompression illness deteriorated, most notably in that he developed urinary retention, which required catheterisation. He also had reduced pin prick, light touch, and temperature sensation below his umbilicus, with pain around his lumbar spine. Vibration and proprioception sense were unaffected; his legs remained as weak though he now showed hyperreflexia. After transportation to a Royal Navy base he had further recompression. The following day he had a third recompression but on this occasion breathing oxygen enriched air. Over these two days there was a steady recovery to normality, with the exception of a slight deficit in light touch and proprioception below his umbilicus and a bandlike sensation around his waist. Negative investigation in the weeks following this were: CSF and urine microscopy, blood urea and electrolytes, glucose, full blood count, VDRL, chest x ray, electromyography, visual evoked responses, and whole spine myelography.

Without further dives he presented 13 years later with spastic paraparesis developing over six months. His minor sensory impairment was unchanged. Bladder, bowel, and sexual function are unaffected. He has never taken regular medication, drank alcohol moderately, has no relevant family history, but had recently become so incapacitated that he was unable to continue as a building contractor. All leg muscles have MRC grade 4/5 power with increased tone, global hyperreflexia, and extensor planter reflexes. The previous investigations remained normal, as did his HTLV 1, CSF electrophoresis for IgG, and magnetic resonance imaging (MRI; T2 weighted spin echo) of his brain.

MRI of his cervical and thoracic spine (T1 weighted spin echo), scanned in sagittal and axial section, showed focal narrowing of the cord between T2 and T7, with expansion at either end to normal calibre. In the narrow section there is increased epidural fat, indicating that this is a long standing lesion (figure).

Over the four years following this latter deterioration, his neurological condition worsened only subjectively. Presently he requires walking sticks but otherwise reports no new changes.

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