Letter to the Editors,

Dear Editors,

WEIGHT-LIFTER'S HEADACHE

Headache precipitated by exertion was first reported by Tinel in 1932. In some cases the underlying mechanism may relate to strain injury or cervical soft tissues, Paulson (1983). In others, reflex vascular changes seem likely, Rooke (1968).

Two cases of headache in weight-lifters are described in whom these mechanisms appear important.

A 23 year old concrete finisher had a one year history of severe headaches of acute onset while doing bench presses (lying supine and pushing weights against gravity). Each lasted 48-72 hours. He described a deep ache beginning in the right forehead and extending to temple, jaw and right side of the neck. There was associated neck stiffness but no nausea or photophobia. General and neurological examinations were normal, with the exception of slight pain on neck rotation. X-rays of the cervical spine were normal. The patient was advised to stop weight-lifting and was asymptomatic three months later.

A 19 year old cabinet maker had a six month history of headaches with acute onset precipitated by weight-lifting (again bench presses) and sexual intercourse. The headaches were occipital, throbbing and lasted 48-72 hours. He described a deep ache beginning in the forehead and extending to temple, jaw and right side of the neck. There was associated neck stiffness but no nausea or photophobia. General and neurological examinations were normal, with the exception of slight pain on neck rotation. X-rays of the cervical spine were normal. The patient was advised to stop weight-lifting and was asymptomatic three months later.

Dear Editors,

A 400 m runner suffering from recurrent post-race collapse, cramps and vomiting was successfully treated by induced alkalosis, therapeutic warm down and bronchodilators.

From 1980 to 1982 the athlete had no real problems, being sick occasionally after some 400 m races. In the summer of 1982 he felt tired all the time and was sick after far more races. He was investigated in hospital and thought to have a possible ulcer though no direct evidence was discovered. Results went steadily down hill over 83/84 and exercise testing showed a very high lactic acid which was confirmed in 1984/5. About 5-10 minutes after a hard 400 m he would develop cramp in his legs that could last for 2-4 days and he would start vomiting with abdominal pains. On one occasion he ran a hard heat on the Saturday, vomited all night and collapsed during the next day's race and was taken to hospital suffering from hypoglycaemia and dehydration. Like many athletes, he would suffer from post-race coughing that could last 3-4 days.

A recent indoor race programme consisted of heats in the late morning, semi-finals some 5-6 hours later in the early evening with the final the following afternoon.

Three major target areas were identified;

1. Lactic Acidosis
2. Post Exercise Coughing
3. Dehydration

Very high lactic acid levels have been recorded after 400 m races, conversation with an East German doctor suggested readings of 20-23 mmol.l⁻¹. Removal of lactic acid is achieved most effectively during exercise eliciting, 60% of VO₂ max but is achieved adequately between 25% and 65% max VO₂. Unfortunately the high lactic acid, hyperventilation and race situation prevent adequate warm down. This therefore encourages peripheral pooling of blood in the leg muscles and inadequate venous return, which may induce faintness, headaches and splanchic ischaemia causing abdominal pain and retching.

Post-exercise coughing is not uncommon and may last several days. Indeed some athletes are unable to sleep lying down on the post-race night. This situation may be compounded at indoor events by air conditioning which can increase dust and potential allergens. Air conditioning at indoor events is notorious for producing dehydration and a vomiting athlete presents problems of inadequate rehydration and glucose replacement.

Attempts to reduce the acidosis were made by administering Tabs Sodium Bicarbonate 325 mg. This was taken at 15 min intervals before the race to a maximum of three. For the heat we tried to raise this to four but the reported side effects of wet flatus started an embarrassing situation. As soon as possible after the race the athlete was taken to the physiotherapy room and laid flat with the legs elevated on a chair. This encouraged venous return, preventing fainting and, as has been seen before, correcting the headache. Both the physiotherapist and myself then cycled a leg each and performed effluage massage for 5-10 minutes. It was noticeable that episodes of leg cycling would increase the ventilatory rate, presumably in response to the increased systemic acidosis returning from the legs.

When the ventilatory rate had slowed, 2 puffs of Duovent...
Dear Editors,

I would like to report a case of bilateral shoulder dislocation.

A healthy 24-year-old male library assistant had been weight training regularly for 2 years. After warming-up exercises, he usually performed 3 sets of 15 bench presses with a 40 Kg barbell. In this lift, the lifter grasps the bar with the hands about a shoulder width apart whilst lying horizontally on a bench (see cover photograph). The bar is then lowered to the chest and pushed back up to the starting position. On the fifteenth lift of the first set both arms suddenly became stiff and painful. He lost control of the weight which fell backwards but was caught by his training partner. Shoulder movements were very painful and tingling was noted over the outer aspect of both shoulders. He then attended the Accident and Emergency Department.

On examination, he was a well-built man and both shoulders appeared to be clinically dislocated. Movement at the gleno-humeral joints was virtually nil. Hypoesthesia was noted in the distribution of the upper lateral cutaneous nerve of the arms bilaterally. There was no vascular abnormality.

X-rays showed bilateral anterior dislocations. No fractures were seen.

Reduction was achieved using Kocher’s manoeuvre after 50 mg of pethidine and 10 mg of Medazolam intravenously. Post-reduction films were satisfactory and reduction maintained using bilateral collars and cuffs and a body bandage. He was allowed home the following day. He was reviewed one week later in outpatients, axillary views were taken and were normal.

Review at 3 weeks after the injury showed no neurological deficit in the right arm, but on the left there was wasting of the posterior half of deltoid with overlying hypoesthesia. The collars and cuffs were maintained for a further 3 weeks. Examination at that time showed the hypoesthesia to be present, but much improved.

Abduction was 90 degrees bilaterally. The bandaging and collars and cuffs were discarded and the patient advised against external rotation. Review at 9 weeks showed a full range of movement with slight blunting of sensation over the left deltoid. The patient failed to attend any further follow up.

Bilateral anterior dislocations of the shoulders (with or without fracture) is rare with only 38 cases reported in papers by Brown (1984), Carew-McColl (1980), Hartney-Velazco et al. (1984), Onabowale et al (1979), Salem (1983) and Yadav (1977) in a literature search. Of these cases, 17 were due to electrocution or seizure and 18 were due to considerable direct violence such as falls, overturning tractors and falling embankments. The remaining 3 were seen in cases with neuromuscular disease, namely myasthenia gravis, cerebral palsy and scapular myopathy. This case seems to be unique in that it involves a healthy male performing an exercise previously within his capability. No report of the bench press being the aetiological factor was found. The position used to perform the bench press is ideal for dislocation, as there is forced extension, abduction and external rotation of the arm which lever the humeral head out of the glenoid. With the current vogue for fitness, it is a surprisingly rare injury.

Yours sincerely,

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