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 for up to forty-eight hours. On one occasion there was loss of consciousness for a few seconds and dizziness. There was no nausea, photophobia or other symptoms and no family history of headache. Full examination, X-rays of skull and cervical spine and C.T. scan were entirely normal. Again the

patient was advised to stop weight-lifting and was asymptomatic three months later.

It has been suggested that headaches occurring with exertion, such as in footballers, runners and weight-lifters, and that occurring at high altitude and with sexual intercourse, have a common pathogenesis, Massey (1982). In most cases there is no structural lesion. However, in a minority there is a posterior fossa abnormality, Symonds (1956). Subarachnoid haemorrhage must also be excluded by the clinical features. In these two cases the pathogenesis appeared to differ despite the common precipitant, Case One having the characteristics of a strain injury and Case Two appearing to have a vascular basis.

Yours faithfully,

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References

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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 finals 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 $\dot{V}O_2$ max but is achieved adequately between 25% and 65% max $\dot{V}O_2$. 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 splanchnic 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