Current evidence suggests that the long-term effects of increased physical activity are beneficial (Shephard, 1974). Unfortunately, exercise can sometimes have serious orthopaedic and cardiac complications in the apparently fit and healthy but cerebrovascular complications have been rarely reported.

We report here a series of cases presenting to a regional neurosurgical unit with serious cerebrovascular complications from exercise who presented within a period of six months.

CASE 1
A 45 year old previously fit man developed a sudden, severe headache associated with neck pain while playing squash. On admission to hospital he had moderate neck stiffness but no focal neurological signs. Lumbar puncture revealed uniformly bloodstained cerebrospinal fluid. CT scan and four vessel cerebral angiography were normal. Recovery was uneventful.

CASE 2
A 43 year old man developed a sudden, severe headache while running in an orienteering competition. He continued to compete for a further 90 minutes before the worsening headache and the onset of vomiting forced him to seek medical help. He had moderate neck stiffness and no neurological deficit when admitted to hospital. Lumbar puncture showed uniformly bloodstained CSF, but CT scan and cerebral angiography were normal. Recovery was uneventful.

CASE 3
A 34 year old regular jogger developed neurological symptoms while running up a steep hill, having already covered eight miles of a planned ten mile run. Headache was followed by dysarthria and weakness of his left side. On examination he was alert with mild neck stiffness and had a grade III pyramidal weakness of his left side. He also had some impairment of pin-prick and joint position sense on the left. Lumbar puncture revealed uniformly bloodstained CSF. CT scan showed a fresh haematoma in the thalamic region of the right hemisphere, but a right carotid arteriogram was entirely normal. He went on to make a good recovery.

CASE 4
A 32 year old fit man suddenly developed a left hemiplegia while playing squash. He had a slight headache but admitted to seeing “flashing white lights” before his
eyes some minutes before his deficit appeared. On examination he was alert and had no neck stiffness. He had a left homonymous hemianopia with grade I weakness of his left upper limb and grade II weakness of his left lower limb. He also had a marked hemisensory loss on the left. CT scan revealed a diffuse low density throughout the right hemisphere with no enhancement after contrast, suggesting massive hemispheric infarction. Shortly after admission his right pupil became dilated and his level of consciousness deteriorated. Despite intravenous Mannitol and elective ventilation he continued to deteriorate and died.

CASE 5

A 17 year old Army cook developed a sudden weakness of his left side while on a training run. On examination he was alert but disorientated with no neck stiffness. He had a left homonymous hemianopia and a grade II weakness of his left-sided limbs. CT scan showed a diffuse low density in the right hemisphere suggesting hemispheric infarction. The day following admission his right pupil became dilated and his level of consciousness deteriorated. He continued to deteriorate and, despite Mannitol and elective ventilation, he died.

COMMENT

In each of the cases above the patient was a relatively young man who did not smoke and who took exercise regularly, at least three times a week, and of reasonable severity. None of the patients was obese and none had investigation including Hb, PCV, urea, electrolytes, ESR, liver function, VDRL, chest X-ray and ECG were normal in all cases and clinical examination (other than neurological) was normal.

There are numerous reports of orthopaedic and traumatic complications of exercise particularly while jogging, such as those of Corrigan and Fitch in 1972, and Corrigan in 1980. Deaths during exercise are widely reported, for instance by Opie, 1975 and by Thompson et al, 1979, and are mainly attributable to ischaemic heart disease. In 1980 spontaneous intracranial haemorrhage while jogging was reported by Lynch, and cerebral infarction has been described in a jogger by Kelly and Roussak, again in 1980, and more recently in a young marathon runner (Phillips et al, 1983).

Transient hypertension occurring during exercise may have been a factor in three of our cases. It is certainly not unusual for spontaneous intracranial haemorrhage to occur from an intracranial aneurysm or A-V malformation at times when blood pressure might be raised, such as during unaccustomed exertion or during sexual intercourse, but none of our cases had a demonstrable vascular abnormality.

The two cases of cerebral infarction reported here were thought, on the basis of the extensive CT scan appearances, to be due to spontaneous internal carotid artery occlusion. Non-penetrating trauma to the carotid arteries in the neck is a recognised cause of occlusion and sometimes the trauma is not obvious clinically, as Milligan and Anderson described in 1980. It is regrettable that no post-mortem data is available to try and elucidate the mechanisms involved.

Our five cases presented within six months to the Wessex Neurological Centre which serves a population of 2.6 million. Hence these complications may not be as uncommon as the isolated reports in the literature have previously suggested.

REFERENCES


