Letters

in sera are low or undetectable.² Phillips *et al* found rises in CK-BB correlated with the degree of head injury.² The source of CK-BB which is responsible for these increased levels is as yet unclear.

In a previous study we noted a rise in CK-BB in amateur boxers after three rounds of 3 minutes. The rise appeared to correlate with the number of direct blows to the head.³ The present study set out to look at another sport involving some head trauma, and its effect on CK-BB. This is again compared with a non traumatic but vigorous sport—track racing cycling.

Twenty eight rugby players and 16 track cyclists agreed to cooperate in the study. Blood was taken before and 15 minutes after two rugby matches, each of which lasted 80 minutes. Blood was taken from the 16 cyclists before and up to 30 minutes after a 40 mile (64 km) race—these values were taken from our previous study. None of the subjects had experienced any trauma in the previous 48 hours and the two groups were of the same age range, 16–25 years. The blood was spun and refrigerated within 2 hours to -20°C , where it was stored for a maximum of 5 days before being transferred to -70°C . At this temperature the enzyme is stable. All samples were allocated random numbers, and were analysed by radio-immunoassay⁴ which has an intra-assay variation of 4.5% and an interassay variation of 10.5% (2).

The baseline values of CK-BB and rises in levels were compared in cyclists and rugby players. Two-way analysis of variance of the log CK-BB values were performed with one grouping factor (cyclists vs rugby players) and one repeated measures factor (before vs after activity). The increase in CK-BB was significantly greater in rugby players than in cyclists ($F = 10.87$, $df = 1, 42$, $p < 0.002$). Although the mean final levels of both groups are similar, the rugby players had a greater rise of the enzyme because they had significantly lower baseline values ($t = 3.47$, $df = 42$, $p < 0.001$). However, the baseline values of neither cyclists nor rugby players differed significantly from established normal values in male blood donors, that is $1.36 \pm 0.88 \mu\text{g/l}$.²

We analysed CK-BB levels of the rugby players according to position of play, dividing them into forwards and backs. There were 15 forwards and 13 backs. Baseline values were not different. Both groups showed significant increases, but this was greater in the forwards than the backs ($F = 5.34$, $df = 1, 26$, $p < 0.03$). The only players in whom there were no significant rises

were the wings (5 wings whose mean values fell from 1.08 to 0.74 after the matches). One of the highest rises in the rugby players was a back who reported a heavy blow to the head (his rise was $2.5 \mu\text{g/l}$).

We have thus shown that there is rise in CK-BB levels in rugby players after an 80 minute match. The rise is greater in forwards than in backs. The lowest rises are found in players on the wing, a position which probably involves the least trauma. The rises in CK-BB could thus be due to trauma sustained during the match. The organ which is the source of the rise is uncertain, as CK-BB has been shown to rise after head injury, and has also been postulated to come from the chest or bowel after injury. In a previous study we showed a rise in young amateur boxers after 9 minutes in the ring and the rise correlated with blows to the head. Thus the rise seen in rugby players may be due to trauma to the head, although other sources cannot be ruled out.

The significance of the finding that baseline values in cyclists differed from that in rugby players is not clear, and is not accounted for by the interassay variation. They were all, however, within the normal range. In our previous study boxers had higher baseline levels, although these were at the upper end of the normal range. Further studies will be required to determine the source and significance of the rises in CK-BB in these sports.

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² Phillips JP, Jones HM, Hitchcock R, Adama N, Thompson RJ. Radioimmunoassay of serum creatine kinase-BB as index of brain damage after head injury. *Br Med J* 1980;281:777–9.

³ Brayne CE, Dow L, Calloway SP, Thompson RJ. Blood creatine kinase isoenzyme BB in boxers. *Lancet* 1982;2:1308–9.

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Table

	Before	After
Cyclists	1.88 $\pm$ 0.83	2.17 $\pm$ 0.94 $\mu\text{g/l}$
Rugby	0.97 $\pm$ 0.85	2.24 $\pm$ 1.01 $\mu\text{g/l}$
Backs	1.00 $\pm$ 0.82	1.74 $\pm$ 1.15 $\mu\text{g/l}$
Forwards	0.95 $\pm$ 0.93	2.58 $\pm$ 0.73 $\mu\text{g/l}$

Grand-mal as the major presenting symptom of infectious mononucleosis

Sir: Infectious mononucleosis is a common acute systemic viral infection which usually affects young adults. The main manifestations of the disease are fever, exudative pharyngo-tonsillitis, generalised lymphadenopathy and splenomegaly. Neurological complications in infectious mononucleosis are uncommon—less than 1% in the large series.^{1–3} Patients who had tonic-clonic seizures as the major presenting manifestation of infectious mononucleosis have rarely been described in the medical literature. We report a further such case.

A 20-year-old army officer was admitted for evaluation of a tonic-clonic seizure which occurred 3 days earlier. The attack lasted for about 20 minutes and was accompanied by temporary loss of consciousness. He became alert before admission. No neurological abnormalities were noted then and computed tomography of the brain was normal. A week before this attack, the patient suffered from mild fever and sore throat which disappeared without any treatment. Past and family history were nonrevealing. Physical examination disclosed a young man in a good physical condition. He had a temperature of 38°C . The tonsils were hyperaemic and enlarged, covered with patchy exudates. He had cervical lymphadenopathy and mild hepatosplenomegaly. There was no neck stiffness and neurological examination was normal. Chest radiogram and the electrocardiogram revealed no abnormalities. Laboratory tests gave results as follows: Hb—14.8 g/dl, WBC 12000/cmm with 65% lym-

phomonocytic cells and marked atypia. Platelet count 188000/cmm. Alkaline phosphatase 353 u/l (N < 100), SGOT 119 u/l (N < 40), LDH 542 u/l (N < 250), total bilirubin 0.5 mg/dl. Mononucleosis slide test was strongly positive. Paul-Bunnell was positive twice in a titre of 1:112. VDRL and Weil-Felix tests were negative. Initial EEG showed diffuse disturbance with theta waves and some bursts of high amplitude slow waves, compatible with encephalitis. The patient recovered within a few days. The fever subsided and liver function tests returned to their normal levels. The seizures did not recur; however, in a repeat EEG, although no longer a slow-high activity could be detected, two episodes of short bursts of spike and wave pattern were recorded on hyperventilation. In view of the obvious diagnosis and the quick recovery, spinal tap was not performed. The patient was discharged with instructions to take phenytoin 300 mg/day.

Although the incidence of neurological manifestations in infectious mononucleosis ranges from 0.37% to more than 20%, the largest series showed less than 1%.¹⁻³ The main types of neurological complications are: meningo-encephalitis, encephalitis, peripheral neuritis, Guillain-Barré syndrome and spinal cord disease.¹⁻³ The neurological involvement may precede or follow the common manifestations of fever, lymphadenopathy and splenomegaly by as much as several weeks. In a few cases of infectious mononucleosis the only clinical signs that appeared were related to the nervous system. The pathological changes in the brain consist of inflammatory lesions with dense perivascular cuffing and diffuse infiltration of the parenchyma mainly with typical mononuclear cells.⁴

Infectious mononucleosis is the cause of up to 5% of all cases of acute viral encephalitis.^{5,6} It is not yet clear whether the encephalitis of infectious mononucleosis is due to direct invasion of the brain or represents a remote effect of the viral infection. The occurrence of seizures during the course of infectious mononucleosis encephalitis is uncommon, and in fact only less than 20 cases have been recorded so far in the English literature.^{1,2,4,7,8} Only in a few of these cases were the seizures the major presenting sign of infectious mononucleosis, as it was in our case. The EEG usually demonstrated increased activity of slow waves, predominantly theta waves, appearing either in paroxysms or in a more continuous pattern.² The disease is usually self limited. The exact prognosis of the neurological complications is difficult to

ascertain. Nevertheless, some reviews indicate a mortality rate of 8% and residual neurological deficits in 12% of the patients.⁹ We advised the patient to take phenytoin, a therapeutic regime which was also recommended by others.⁷ The possibility that the seizures which appear during the course of infectious mononucleosis actually express a quiescent tendency of epilepsy that was activated by the viral disease cannot be ruled out. The focal spike and wave pattern which was demonstrated in EEG after the clinical recovery of our patient may hint at that possibility. On the other hand, there could be of course a focal lesion induced by the virus itself. In another encephalitic case presented by Ruutu *et al*, a transient localised lesion was demonstrated in CT scan.¹⁰

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CT scan evidence of postero-lateral thalamic infarction in pure sensory stroke

Sir: Pathological studies have shown that the posterolateral thalamic nucleus is the most frequent site of infarction in pure sensory stroke.¹ The CT scan is commonly normal in these patients,² and none of the few reported cases with positive CT findings had a single hypodense lesion limited to the thalamus.³ In the following case of pure sensory stroke, CT scan revealed a small infarction in a site believed to be that of posterolateral thalamic nucleus.

A hypertensive right-handed man, aged 51 years, with no previous history of cerebrovascular disease, experienced the sudden onset of numbness and paraesthesias involving the whole left side of the body. Neurological examination revealed distal hypaesthesia of the left limbs without motor or visual impairment, dysphasia or dyspraxia; tendon reflexes were increased on the left side. A CT scan showed a small hypodense lesion in the lateral part of the right thalamus, 5 mm in diameter (fig). Cerebral angiography showed tortuous intracerebral vessels without significant stenosis. Symptoms gradually disappeared within four days. A second CT scan, performed three weeks later, was unchanged.

Fig. CT scan showing small lacune in the right posterolateral thalamus.