Table 2 Significant differences found in stroke and control groups between patients with acute and chronic diseases

	Stroke patients N = 30		Controls N = 30	
	D	PA	D	PA
Acute N = 17	0.99 ± 0.45	0.45 ± 0.39	0.69 ± 0.55	0.3 ± 0.3
Chronic N = 13	1.57 ± 0.78*	0.93 ± 0.6*	1.01 ± 0.72*	0.6 ± 0.42*

Student t test *p < 0.05 (two tailed)

insomnia and sleeping disorders. As for the obsessive-compulsive disturbances, whose values are rather high and hence indicate a fairly characteristic pattern, it should be noted that while the scale mainly measures clearly obsessive-compulsive symptoms such as the inability to get rid of undesired thoughts, words or ideas, the need to check and double check what is done, etc, it also contains questions aimed to evaluate more general cognitive difficulties and could hence be influenced, in the stroke group, by organic symptoms due to the lesion to the CNS. Lastly, we draw attention to the fact that with the passing of time phobic type symptoms often tend to appear in stroke patients.

In conclusion, our data confirm depression as the main form of psychological distress appearing after stroke, but also demonstrate the existence of other response patterns. Since some of these patterns appear to vary in time, longitudinal studies would be of great help in increasing our understanding of the problem.

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Toxic shock syndrome presenting as cerebral infarct

Sir: Neuropsychological sequelae such as impaired memory, calculation and poorly sustained concentration have been described after the toxic shock syndrome.¹ We report a man whose presentation was with a cerebral infarct.

A 38-year-old taxi driver presented with 12 hours of severe diarrhoea, vomiting and myalgia. Immediately prior to admission he suddenly collapsed in his kitchen. On arrival he was semi-conscious and shocked with a pyrexia 40.0°C, pulse 110 and a systolic blood pressure of 80 mm Hg. He had purposeful movement to pain of the left side of his body but not his right, and the right plantar was extensor. All cultures including blood and lumbar puncture were negative. He was treated with intravenous ampicillin, flucloxacillin, gentamicin and steroids. Within 24 hours he developed respiratory failure with bilateral interstitial pulmonary infiltrates and required ventilation for 8 days. His platelet count fell to $69 \times 10^9/l$ and there was a rise in titre of fibrin degradation products. His urea and creatinine both rose to three times normal. After 36 hours his left leg became cold with loss of left femoral and all distal pulses. These returned within 6 hours of full heparinisation. By the third day he had developed a fine erythematous macular rash, and a pointing scrotal abscess with inguinal lymphadenopathy was noticed. 10 ml of pus from the abscess showed no growth but a skin swab grew a coliform and a non-toxin-producing strain of *Staphylococcus aureus*. Once off the ventilator he was found to have a mixed motor and sensory dysphasia with a right-sided hemiplegia. A CT brain scan showed a left temporoparietal infarct in the middle cerebral artery territory. A repeat scan after one month was unchanged. On the 13th day his

soles and palms desquamated. During the illness his antistaphylolysin titres and anti-nuclease titres rose four fold. Normal investigations included serum amylase, viral, mycoplasma, legionella and antistreptolysin-O titres. He went home after 6 weeks.

Although we failed to isolate a *Staphylococcus aureus* able to produce exotoxin F we feel the clinical picture fulfills the case definition of toxic shock syndrome.² The abscess was sterile as he had received 2 days of effective anti-staphylococcal treatment prior to drainage. We believe this is the second case of scrotal infection causing the syndrome in this country.³ It is not clear why this man should have developed a cerebral infarct. There was no clinical evidence of endocarditis nor blood culture.⁴ Suggestions for mechanism of neurological damage have included direct toxic, altering permeability of the blood brain barrier or an immunologically mediated vasculitis.¹ Large vessel spasm in this case might explain the cerebral infarct and the transient loss of leg pulses. The toxic shock syndrome may still have surprising presentations and should be considered in any septic ill patient, not just in menstruating women.

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Creatine kinase BB isoenzyme in rugby football players

Sir: Creatine kinase isoenzyme BB (CK-BB) has been found in high concentrations in the brain. It is found in lesser concentrations in the gut.¹ Normally, concentrations

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.

We thank Dr RJ Thompson for arranging the CK-BB assays, Dr P Fonagy for his work on the statistics and the sportsmen for participating. SPC is a Wellcome Research Fellow.

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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-