inadequate to cause brain necrosis. Coma can persist for about 2 weeks in cases where there has been respiratory arrest without cardiac arrest in young people and they can recover (reference 4).

In hypoxic lesions without ischaemia there are usually no neuropathological changes (reference 5).

In this case there is no description of a macroscopic infarct of any type and no infarcts are present in the sections. There is some hypoxaemic/ischaemic neuronal damage, that is pink neurones are seen and if this is graded as in the reference from Graham et al (reference 6) it is diffuse and mild. This change can occur within an hour of hypoxic/ischaemic damage. It can also be due to other insults than the original episode when he was assaulted. Hyperthermia itself can also cause hypoxic/ischaemic neuronal damage

As far as I am aware hyperthermia gives rise to fits, but does not usually give rise to active breakdown of muscle fibres (this process is called rhabdomyolysis) on its own without there being a disorder of mitochondria. I also do not know of any specific brain changes that occur in malignant hyperthermia (reference 7).

THE EXACT NATURE OF THE BRAIN DAMAGE

In my opinion he had sustained blows to his head, which resulted in the fractured left sphenoidal wing and the haematoma over the left supra-orbital plate. The latter is more likely to have been due to an occipital injury than to the injury causing the left sphenoidal wing fracture. During these injuries he had acceleration/deceleration damage to his brain resulting in traumatic diffuse axonal injury of Grade II microscopically. Most of the traumatic diffuse axonal injury was in the white matter, the areas were seen as collections of macrophages and all appeared to be of the same age with no large areas of white matter rarefaction around them so they were not getting larger.

There was some evidence of hypoxaemic/ischaemic change with pink neurones in some areas of the cortex also mild loss of neurones was present as well. There was no evidence in the slides or the macroscopic description of the brain from the post mortem report of infarction, so the hypoxaemic/ ischaemic change was mild. This change could well be related to the initial assault, but some may have been due to the hyperthermia he had. Also the amount of white matter damage may have been increased by the hypoxaemia/ischaemia, but I do not think this has increased the damage by more than one third and it is probably much less.

CONCLUSIONS

1. The neuropathology in this case has been hampered by no photographs of the brain and the macroscopic description is brief with no indication if there were any areas of infarction; although mention of