should receive thiamine supplements as, indeed, should any patient with prolonged vomiting from any cause. If confusion, ataxia, or ocular signs (usually a sixth nerve palsy, gaze palsy, or nystagmus) develop parenteral thiamine (at least 50 mg daily) should be given until a normal diet is reinstated. Blood should be taken to measure red cell transketolase activity or the thiamine pyrophosphate effect, but administration of thiamine must not await the result of this assay.

Not all this patient’s problems could be explained on the basis of Wernicke’s encephalopathy. Bulbar dysfunction and pyramidal signs are not features of Wernicke’s encephalopathy. Reflexes are usually absent rather than exaggerated. The clinical consequences of central pontine myelinolysis vary considerably but pseudobulbar palsy and pyramidal tract signs along with depressed levels of consciousness are common manifestations.2 14 Wernicke’s encephalopathy and central pontine myelinolysis occur together more often than can be explained by chance.2 15 There are two other reports of the conditions coexisting during pregnancy.2 16

There is still controversy over the pathogenesis of central pontine myelinolysis, but it is widely regarded as being the result of rapid correction of hyponatraemia.2 16 17 Our patient’s serum sodium concentration never fell below 126 mmol/l, though electrolyte values were measured only weekly. No attempt was made to correct the hyponatraemia, and hypertonic saline was never given. In the two other cases of combined Wernicke’s encephalopathy and central pontine myelinolysis in pregnancy the lowest serum sodium concentration recorded was also 126 mmol/l.17 Possibly, therefore, thiamine deficiency somehow makes the myelin sheaths of the central pons more sensitive to changes in serum sodium value. Certainly we agree with the advice of other authors that chronic hyponatraemia should be corrected slowly, but we also emphasise the need for thiamine supplementation if there is any possibility of deficiency of this vitamin.

Correction

Any Questions

Owing to an editorial error the name of one of the experts who answered the question on the “golden hour” in major trauma (22 August, p 459) was omitted. The expert is Dr Fiona Gibson, registrar in accident and emergency medicine, Peterborough.

Would the ingestion of such tablets be hazardous? The amount of material derived from cattle is presumably small. The oral route of challenge is inefficient for the experimental transmission of bovine spongiform encephalopathy to other species. A course of tablets would, at worst, represent possible sequential doses by an inefficient route if, in the first instance, potentially infected raw brain or spleen was involved in the preparation. While normal cooking temperatures do not definitely inactivate the agent causing bovine spongiform encephalopathy, they can reduce the infectivity. The agent does not occur at high concentration in muscle (meat). Eating properly cooked beef (or lamb for that matter) is safe for humans, but I would advise against eating bovine (or ovine) brain or spleen because these tissues are recognised as likely replication sites. It seems rational to extend this exclusion to untreated extracts of bovine brain or spleen, even if the possible hazard cannot be quantified. It would be fair, of course, to determine exactly the details of the source of the material concerned.—J G COLLEE, retired professor of medical microbiology, Edinburgh