bicarbonate measurement (strictly total CO_2 as bicarbonate cannot be readily measured) is rarely clinically indicated and is expensive. In addition it deteriorates in transit and routine measurement of venous samples is likely to be misleading.[1] An accurate assessment of a patient's acid–base status requires measurement of pH and PCO_2 and is, therefore, usually performed on arterial samples when clinically indicated.

Regular routine estimation of plasma electrolytes on patients receiving acetazolamide is not practised by most ophthalmology departments and our study found no need for regular potassium estimation. We believe ophthalmologists will demand more evidence-based medicine in the form of prospective studies of the acid–base status in elderly patients before advocating regular arterial samples to assay bicarbonate in ophthalmology clinics.

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Sir,

I read with interest the recent report by Fraser and Horgan of meningococcal meningitis associated with unilateral subretinal and vitreous haemorrhage.[1] This is not the sole documented case as I have also reported on a patient with meningococcal meningitis in whom intraocular haemorrhage occurred.[2] In this case, bilateral intraretinal haemorrhages developed in addition to unilateral petechial iris haemorrhages and sectorial haemorrhagic iris infarction. In contrast to Fraser and Horgan's case, this latter case was quite clearly related to meningococcal septicaemia and disseminated intravascular coagulation. Cutaneous and systemic haemorrhages in meningococcal septicaemia are considered to occur by different mechanisms. While cutaneous purpura is the result of an endotoxin- and immunologically-related occlusive vasculitis (in which bacteria may thrive), disseminated intravascular coagulation is the main cause of the sterile systemic haemorrhages.[3–5] Thus, although initial management of intraocular haemorrhages in meningitis may be conservative, as suggested by Fraser and Horgan, particularly when associated with *Neisseria meningitidis*, a concerted effort must be made to exclude potentially fatal disseminated intravascular coagulation, even in the absence of cutaneous purpura.

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Sir,

We are grateful for Dr Kennedy's interest in our paper[1] and his comments are highly pertinent.

It is undoubtedly very important that disseminated intravascular coagulation is excluded in meningococcal meningitis, and any patient noted to have an intraocular haemorrhage in the acute stages of the disease should be investigated appropriately. However, retinal or vitreous haemorrhage may be difficult to diagnose in the acute situation. The patient often has a decreased level of consciousness and therefore will not complain of a decrease in vision. Examination of the fundi can also be difficult because of photophobia and the problems of dilating a patient who is undergoing neurological observations.

The patient who we reported did not complain of any visual problems until 6 days after her admission, and this seems to be typical of ocular complications in meningitis. Dr Kennedy's case exemplifies the importance of ocular examination in the patient with meningococcal meningitis at the time of presentation – and this examination should continue throughout the course of the disease. Further to this, because ocular complications of meningitis may be as high as 70%,[2] there may well be a case for routine referral to an ophthalmologist for a fuller examination once the acute stage of the disease has passed.

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