tachycardia and electrocardiogram confirmed atrial fibrillation with a ventricular rate of 170–180 beats/minute. Blood pressure remained stable. The dysrhythmia persisted and required treatment with amiodarone for 3 days before reverting to sinus rhythm. Serial electrocardiograms, and measurement of cardiac enzymes, urea and electrolytes were normal. There were no long-term sequelae.

Ventricular tachycardia associated with hyperkalaemia has been reported after ibuprofen overdose,³ although tachycardia and hypotension are more consistent features.² This is apparently the first report of atrial fibrillation occurring after an overdose of ibuprofen in a previously healthy individual. The presence of mild mitral regurgitation, and a possible hidden accessory pathway (as suggested by the short PR interval) may, however, have been predisposing factors in this case.

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Cerebellar haemangioblastoma in an octogenarian

Sir,

Sporadic cerebellar haemangioblastoma usually affects middle-aged individuals.¹ We recently excised this tumour in an 87 year old woman with subsequent rehabilitation to independence. The mean age at which this tumour presents is rising because a larger proportion of cases are occurring in older patients.² It is believed that during foetal development, a hamartoma forms in the cerebellum from the choroid plexus in the 4th ventricle. Cystic transformation then gives rise to the tumour.³ Why should such a transformation have occurred in our patient so long after the formation of a congenital lesion? Maher has recently provided an answer that requires an understanding of tumour suppresser genes.² Patients with Von Hippel–Lindau syndrome are heterozygous for a recessive tumour suppresser gene and require a single mutation for the formation of familial cerebellar haemangioblastoma. The sporadic tumour only occurs when two spontaneous mutations are acquired during life. This could statistically occur at any age after birth.

Although rare, cerebellar haemangioblastoma should be considered in the differential diagnosis of non-specific symptoms associated with acute cognitive impairment in old patients like ours. The usual physical signs of nystagmus, ataxia and papilloedema may not be elicited if patients are uncooperative in which case a computed tomographic scan of the head is necessary to exclude the diagnosis. A cystic low density mass is seen usually with an enhancing mural nodule. Cerebellar haemangioblastoma is more likely if papilloedema can be demonstrated as it occurs in up to 90% of cases.⁴ When senile miosis and cataract prevent adequate direct fundoscopy, referral to an ophthalmologist for examination by more sophisticated equipment could be valuable. With advances in neuroanaesthesia and a reduction in operative mortality,⁵ elderly patients should not be excluded from major surgery.

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Buerger's disease in Western India

Sir,

The predominant peripheral vascular disease seen in our busy surgical service is Buerger's disease (thromboangiitis obliterans). In 1988–1991 we treated 62 cases of Buerger's disease, all males below 42 years age (mean age 38 years). A total of 51 cases had peripheral gangrene with intractable pain, while the remaining 11 had non-healing ulcers with severe pain requiring narcotic analgesics in increasing doses. Lumbar sympathectomy (unilateral in 52, bilateral in 10 cases) was performed in all patients. Sympathectomy provided only temporary relief of the symptoms, 59 cases ended up in below-knee amputation, while the remaining two patients had minor amputations of the toes. The mean time interval between the onset of symptoms and the amputation was 22 months, while the mean time interval between sympathectomy and amputation was 6.4 months.

Invariably all patients affected by Buerger's disease come from the lowest socioeconomic strata of the Indian society, smoke bidis (home made cigarettes with raw tobacco) and suffer from varying degrees of malnutrition.

The rapid train of events ending in amputation leads us to believe that patients in Western India suffer from a particularly virulent type of Buerger's disease not amenable to sympathectomy. Recently, Fiessinger and Schafer¹ conducted a placebo-controlled prospective trial comparing either oral aspirin and a 6 hour daily placebo infusion, or a placebo tablet identical to aspirin and a 6 hour infusion of Iloprost. They showed that intravenous Iloprost for 21–28 days was significantly more effective than low-dose aspirin or placebo for the relief of rest pain and healing of ulcers. The findings of this multi-centre trial are exciting, but may not be applicable to patients suffering from Buerger's in Western India, as Iloprost has to be given by infusion daily for up to a month and is expensive. Fiessinger followed the patients for only 6 months which is too short a period for the appearance of complications of this disease. However, these results are encouraging and perhaps Iloprost may have a role in salvaging limbs after failure of lumbar sympathectomy.

Percutaneous transluminal angioplasty is of value in selected cases of atherosclerotic peripheral vascular disease² but in Buerger's disease, because of multiple blocks in small arteries, angioplasty is of no use. Laser thermal angioplasty is being tried in total peripheral artery occlusions,³ but this procedure is as yet experimental. Omental revascularization of the extremities as described by Casten and Alday⁴ is being tried in our unit for those patients who have failed to respond to sympathectomy and are in imminent danger of losing their limbs. Long-term results are awaited but in the short term this procedure seems to decrease pain and increase collaterals as seen in post-omental revascularization angiograms.

The absence of proven alternative therapies in Buerger's disease is dismal news for these patients. More research needs to be done and new drug/surgical therapies need to be developed for this disease.

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Depression and chronic clonidine therapy

Sir,

Clonidine is an α_2 adrenoreceptor agonist used in the treatment of hypertension. Central adverse effects such as sedation are the commonest problem. Depression is a known though uncommon side effect of clonidine, and usually presents when the drug is initially introduced. Its prevalence is about 1%¹. We recently encountered a case of depression in an elderly patient who had no past psychiatric history and had been on clonidine for 18 years. The history of depression was short and it improved dramatically once the clonidine was withdrawn. Our experience suggests that clonidine therapy may indeed be a contributing factor in a depressive illness and it may do so many years after commencing the drug.

An 87 year old woman presented as an emergency with a 2 day history of lethargy and immobility. Her husband died recently. Drugs on admission included clonidine 0.3 mg twice a day. Blood pressure was well controlled. She said she felt low in her mood and was tearful. Her appetite was poor and sleep disturbed. It was decided to gradually withdraw the clonidine as it was felt that it may be contributing to her depression. A psychiatric opinion confirmed the diagnosis of mild depression. As soon as the clonidine was withdrawn her mood improved, her appetite returned and she started to mobilize. Her bowel habit which had been constipated also became regular once again.

Recent reviews on the use of clonidine in the treatment of hypertension indicate that depression is not a major problem.² A few studies on clonidine have reported depression as a side effect³ and others have not.⁴ Nevertheless, depression is the commonest psychiatric adverse effect of clonidine reported to the Committee on Safety of Medicine (CSM, personal communication). Clonidine has been shown to produce behavioural depressive effects in laboratory animals, and this has been suggested to be a suitable animal model for depression.⁵ The biochemical basis for clonidine-induced behavioural changes in animals is thought to be due to a decrease in noradrenaline release in the central nervous system.

Though depression is not universally accepted to be an adverse effect of clonidine, it seems likely that it may be in view of its other central adverse effects. In our case the sequence of events strongly suggested that clonidine was contributing to the depression, though one cannot be certain. The other point of note is that depression may result many years after the drug is commenced. We would like to recommend that in any patient with depression on clonidine therapy, withdrawal of the drug may be beneficial. It may be that the potential of clonidine to cause depression is underestimated as it is an uncommon adverse effect in an infrequently used drug.

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