shown no benefit after oral immunosuppression. Indeed, introduction of a moderately high dose of prednisolone caused a dramatic deterioration, a response that is recognised in CIDP.[3 6] Unlike the modest clinical benefit seen by others after the adminstration of IVIg[3] our patient remains exquisitely sensitive to IVIg.

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Sodium valproate for tinnitus

In 1935 Barany serendipitously discovered the temporary relief of tinnitus after lignocaine injection of nasal turbinates.[1] Since that time, other agents known to suppress the activity of excitable membranes have been tried, including antiarrhythmic and anticonvulsant drugs. Among such drugs, carbamazepine has the best documented efficacy in patients with a positive lignocaine test,[2] but is generally unhelpful in unselected tinnitus populations and often discontinued due to adverse effects.[2 3]

A 53 year old man with viral cardiomyopathy developed severe (60 dB) tinnitus after bilateral temporal lobe strokes. Various treatments including masking and diazepam were unhelpful. Carbamazepine (200 mg nightly) was effective, but was withdrawn due to progressive hyponatraemia (120 mM after two weeks of therapy), followed by the rapid recurrence of tinnitus. Sodium valproate (200 mg twice daily) was also promptly effective in suppressing tinnitus, and was well tolerated until his death due to cardiac arrhythmia one month later.

In part due to its diverse aetiology, pharmacotherapy of tinnitus has met with very limited success.[2 4] Uncontrolled trials in the French[3] and Japanese[5] literature have indicated benefit from sodium valproate in selected patients, but its use seems not to have been described in English apart from a specialist monograph.[2] Tinnitus loudness[5] and sensorineural pathology[3] but not lignocaine response[5] seem to predict response. Valproate may also differ from carbamazepine in that it seems better tolerated in an unselected tinnitus population.[3] Controlled studies of valproate for this common, often debilitating[4] condition seem warranted.

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Audible carotid dissection

Carotid dissection is a common cause of stroke in the young patient and can present with various clinical syndromes or symptoms. These may include stroke or transient ischaemic attack,[1] ipsilateral ptosis, isolated or multiple cranial nerve palsies,[2] carotidynia, hemicrania,[3] scintillating scotomata, pulsatile tinnitus, or subjective bruit.[4] I recently cared for a man who experienced an audible "creaking" sound heard even by his wife in the hours before a right middle carotid artery (MCA) infarct secondary to a carotid dissection. I think that this sound represented the actual dissection.

A forty three year old, right handed lawyer with a presumed viral pharyngitis and severe cough for two weeks duration returned from work at 6 00 pm and began hearing periodic, high frequency, "creaking" sounds in his right ear. These sounds occurred every 1–2 hours lasting a few seconds each time. These sounds were not pulsatile or rhythmic. He had not experienced these sounds previously with his illness. When sitting at the dinner table, his wife too heard these peculiar sounds. On admission she provided a detailed description of these sounds as the patient himself was lethargic. At midnight, he experienced a scintillating scotomata with right retroorbital headache and by 1 30 am was lethargic with a left sided weakness. According to the wife and the patient these sounds had now ceased and did not recur.

On examination he had diminished attention, and mental status was otherwise normal. Neck auscultation was normal and there was no audible creaking sound. Fundi and visual fields were normal. A left lower facial droop was present with otherwise normal cranial nerve function. Left arm plegia and left leg paresis was present with associated hyperreflexia and extensor plantar response. He reported diminished sensation to pin, position, and light touch and extinguished left sided touch on simultaneous bilateral stimulation. Brain MRI at 12 hours showed a T1 hypointense and T2 hyperintense lesion in the anterior MCA distribution, thrombus in the MCA (M-1 segment), with a suggestion of focal narrowing in the upper cervical region of the right internal carotid on MRA. An angiogram confirmed a right internal carotid dissection and MCA thrombus. The patient was anticoagulated and with rehabilitation is ambulatory with partial use of his left arm.

Whereas the clinical picture may be typical of carotid dissection, the clearly audible creaking sound that occurred in the early phase of the illness was unusual. I excluded relating this sound to middle ear congestion, hallucinosis, or aura of migraine or epilepsy as these possibilities would have been subjective phenomena not experienced by the patient's spouse. More commonly, auditory symptoms associated with carotid or vertebral dissection are related to altered vascular haemodynamics. Subjective sudden onset of bruit and pulsatile tinnitus are well described and are relatively frequent symptoms of carotid dissection. When present, these symptoms are constant, occasionally appreciated by auscultation, and usually persist even after presentation to the physician. Again however, to my knowledge they have not been reported as being heard externally. Aortic dissection can have a variable presentation, but I have been unable to find evidence of externally heard sounds in this setting.[5]

I suggest that these audible creakings in the context of a later documented arterial dissection were more likely the early tearing sounds themselves of the carotid dissection evolving over several hours. Such sound could have been heard externally and would have represented a progressively enlarging mechanical tear of a high pressure arterial system. The fact that these sounds were intermittent and recurrent, of limited duration, and unassociated with complaints of bruit or tinnitus suggests that the dissection developed slowly with several short bursts of "mini-dissection" before the devastating stroke. This case offers additional evidence that some carotid dissections may have a time course that if recognised might be amenable to emergency intervention. If a patient reports audible creaking without a clear explanation, an evolving arterial dissection should be considered.

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Gynaecomastia in association with phenytoin and zonisamide in a patient having a CYP2C subfamily mutation

Anticonvulsant drugs can have various side effects on endocrine functions, such as impotence, hirsutism, infertility, and thyroid dysfunction. Gynaecomastia is caused by many types of drugs such as methyldopa, tricyclic antidepressant drugs, isoniazid, and spironolactone,[1] but there have been only a few reports of gynaecomastia caused by anticonvulsant drugs, including phenytoin[2] and zonisamide.[3] We recently encountered a young man with partial seizures, who