anatomical variety of the anterior inferior cerebellar artery.

During an investigation of the posterior cranial fossa, we became aware of an abnormal relation between a vessel and a nerve, which is not described in the current textbooks and encyclopedias. Although there is much literature reporting a close relation between the symptom "facial tic" and vessel variety[3 4]; our variety—that is, with both transfixion of the facial nerve and an arterial loop around the same nerve—has not been described in the specialist literature, nor has it been mentioned in the most recent review of variants.

Several authors have provided illustrations of a loop formed by the anterior inferior cerebellar artery, but without elaborating further on the topographic relation between the artery and the internal acoustic meatus or the seventh and eighth cranial nerves. It has been asserted that this vessel seldom appears on radiographs.

Typical hemifacial spasm is caused by vessels on the anterocaudal side of the nerve, whereas atypical hemifacial spasm is caused by vessels on the posterior rostral side.[5]

The relevant aspect of this article lies in its emphasis on the connection between the neurological symptoms and this anatomical variety of a nerve.

The deceased had begun to experience intermittent symptoms of varying intensity in his face at the age of 49. These symptoms took the form of uncontrollable twitching at the right corner of his mouth, ipsilateral hearing impairment, retroauricular cramps, and retroauricular sensory impairment. According to the case history, the deceased had undergone a full otorhinolaryngological examination and pure tone speech audiometry during his lifetime. Thus, it was possible to diagnose the perceptive unilateral acoustic hypoacousia on the right. Early auditory evoked potential studies had also been performed showing an increase in latency and a decrease in amplitude without any deterioration of morphology of the waves. It had not been possible to use MR for the diagnosis as he had a pacemaker in place. None of these symptoms responded to therapy with botulinum toxin.

In our case the compression of the facial nerve at several points could have led to irritation of the region supplied by the posterior auricular nerve and in this way to the symptoms described above.

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CORRESPONDENCE

Charles Bonnet syndrome: an example of cortical dissociation syndrome affecting vision?

This letter is a response to that by Abhijit Chaudhuri.[1] I thank Chaudhuri for his kind comments, but will address his surprise that I did not comment on the Charles Bonnet syndrome in my 1999 paper.[2]

Chaudhuri and his associates cannot be really criticised for using the eponym "Charles Bonnet syndrome" for the "triad of visual hallucinations, visual sensory deprivation, and preserved cognitive status" as there is support in the literature for the use of the eponym in this way. But I favour the use of the eponym, if it is to be used at all, only in patients with eye disease. This was what Charles Bonnet described,[3] and the way the term was initially used by de Morsier in 1936.[4] I highly recommend the paper by ffytche and Howard[5] in which the authors provide an excellent summary of Charles Bonnet syndrome on pages 1253–1254. I agree with the way that they accept the use of the term, and their reason for doing so. It is, thus, a matter of personal opinion as to how the eponym should be used. It was never used in any of the references in my 1999 paper, but I did not choose the references with this reason in mind.

Is there any reason to use the eponym at all? It may remind ophthalmologists that visual hallucinations can occur with ocular disease and do not necessarily suggest a neurological lesion. But, as the eponym has now acquired two different meanings I think that it leads to more confusion than clarification. Chaudhuri's communication is a good example. I would thus suggest that it no longer be used. I did not use the term because I did not, and do not, think that the subject of my report (myself) has the Charles Bonnet syndrome. Nor, presumably, did the reviewers of my paper and the Editor of this *Journal*.

Finally, I must tell Chaudhuri that "advanced age" hardly begins at 60 years.

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Effects of topiramate on cognition

This letter concerns the recently published study by Thompson *et al*,[1] reporting the authors' findings on cognitive effects with topiramate. Firstly, I want to correct the authors' mischaracterisation of a review paper of mine. The authors state that "the literature on antiepileptic drugs ... emphasised positive psychotropic effects," referencing only a 1998 review in which I discussed cognitive and psychotropic effects of antiepileptic drugs. Although I mention some positive effects, I also discussed negative effects, including studies from my own centre that have shown significant negative psychotropic and cognitive drug effects.

Secondly, I provide some perspective on the report of Thompson *et al* of clinically significant cognitive declines in 18 patients treated with topiramate as adjunctive therapy. The authors correctly conclude that "caution is warranted in the interpretation of the findings due to methodological limitations of the study design." Because their study was retrospective and observational, it is susceptible to considerable subject selection bias. For example, five of the 18 patients were specifically included in the topiramate sample because they reported cognitive effects.

The only way to minimise effects that may bias study conclusions is to conduct a prospective randomised controlled study. Two such studies have recently compared topiramate and valproate as add on therapy to carbamazepine, using comprehensive neuropsychological batteries to objectively measure drug effects.

At the end of 3 months of maintenance therapy, only one of 17 (6%) variables in one study[2] and only two of 30 variables (7%) in the other[3] were significantly worse for topiramate compared with valproate. For the three variables with statistically significant differences, the mean differences in change scores were modest. Analysis of individual data showed that scores were unchanged or even improved in most patients receiving topiramate and valproate. Statistically significant differences could be accounted for by a minority of patients receiving topiramate in whom scores deteriorated>1 SD from baseline. I suggest that the patients reported by Thompson *et al* likely represent a similar subgroup of patients.

Physicians should be aware that a subgroup of patients treated with topiramate may experience clinically significant cognitive effects. When these effects occur, they are generally apparent to the patient or family members and can therefore be monitored with routine clinical evaluations. Alternatively, a brief cognitive test (for example, a verbal fluency test or symbol digit modalities test) should easily detect changes of the magnitude reported. In a subgroup of patients, topiramate may need to be discontinued if cognitive effects do not resolve over time with slowed titration or dosage reduction.

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The authors reply:

We write in response to the letter of Meador. We concur that his review article discusses both positive and negative psychotropic effects of antiepileptic medication.

Meador comments on the biased nature of our patient sample. However, we think that we adequately emphasised that our study was not randomised or controlled but carried out in a clinical environment. We pointed out that some of the patients had been referred due to cognitive complaints. Furthermore we acknowledged studies which reported no cognitive effects of the drug including the valproate and topiramate study and drew attention to the conflicting findings. We agree that prospective randomised controlled trials are the way to minimise selection bias. However, they are not the be all and end all. Bone marrow aplasia with felbamate and visual field constriction with vigabatrin treatment were not found in randomised controlled trials but by the careful clinical study of patients. This is an analogous situation. Randomised controlled trials are not without their own biases as most will be sponsored by the pharmaceutical industry and it would be naïve to conclude that this does not influence the presentation of the results.

We agree with Meador that the adverse effects of topiramate reported are likely to occur in a minority of patients treated with the drug. However, we think that this may represent a clinically significant number of patients, particularly in those attending tertiary referral centres. Negative effects, however small the numbers, are worthy of reporting and of further exploration. The question our findings raise is not does topiramate have adverse effects but rather why does it have adverse effects in some people?

We agree with Meador's final point and indeed this was one of the intended take home messages of our paper. This is why we chose to submit to a journal with a broad readership who would have much less experience with topiramate. We hoped that our paper would draw attention to a group of patients who should be prioritised for neuropsychological monitoring and highlight the type of measures that could be employed showing that an extensive assessment is not necessary.

We, however, do not think that the cognitive changes experienced would be obviously attributed to topiramate treatment. Most patients in the study were not referred because of cognitive complaints. Six were being seen as part of their presurgical assessment and indeed were not considered good candidates due to their neuropsychological test profiles. For some the cognitive complaints did not occur in association with the introduction of topiramate or with any change in dosage and did not seem to develop until they had been on the drug for several months. For such patients, particularly those with left hemispheric pathology, increases in word finding problems and other verbal difficulties are more likely to be attributed to the underlying pathology and ongoing seizures than to a drug effect. Topiramate is a useful antiepileptic drug but it may lead to adverse cognitive changes and we need to be alert to this.

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Postoperative hearing loss due to venous congestion at the inferior colliculus, or cochlear dysfunction?

I read with interest the article by Strauss *et al*[1] on postoperative contralateral hearing loss which developed on the third day after microvascular decompression for trigeminal neuralgia. They attributed the symptoms to venous congestion at the ipsilateral inferior colliculus after dissection of the pontotrigeminal vein, which was documented by MRI. Symptoms resolved partially after intravenous rheologic medication for a total of 19 days. The authors' explanation for the delayed postoperative hypacusis, however, merits further discussion. Strauss *et al*[1] provided preoperative and postoperative recordings of brain stem auditory evoked potentials: postoperatively, after stimulation on the operated side, ipsilateral waves I through V, and contralateral waves II through V are all clearly identifiable, contrasting with stimulation on the non-operated side, depicting only a small wave V bilaterally, but no other components. This pattern suggests a left sided lesion involving the generator of wave I—presumably the auditory nerve near the cochlea—and is also consistent with the patient's pancochlear hearing loss.[2–4] By contrast, brain stem lesions—unless damaging the cochlear nucleus— are usually not associated with pure tone hearing loss, but rather with abnormal auditory localisation or interaural time discrimination,[2 5] as auditory impulses are conveyed bilaterally in the brain stem.[2–4 6] Furthermore, a brain stem lesion causing profound hearing loss is likely to produce also contralateral wave IV/V abnormalities after stimulation on the non-affected side, but even the severest brain stem lesions, such as in evolving brain death, do not affect wave I.[3] The vascular supply of the mesencephalic brain stem differs from that of the inner ear, the first being fed by mesencephalic arteries via the posterior cerebral or superior cerebellar artery, and drained through the superior petrosal vein; the second being supplied by the more caudally originating labyrinthine artery via anterior—or occasionally posterior—inferior cerebellar artery, and drained by the labyrinthine vein through the posterior part of the superior petrosal or transverse sinus, and the internal jugular vein.[6] Although the patient's hearing may have been partially affected by the documented mesencephalic lesion, hearing loss may in fact be more likely caused by concomitant cochlear dysfunction. An ischaemic lesion seems probable, presumably postoperative vasospasm, or—less likely—unrelated embolism. In either situation, rheologic treatment may have been beneficial, as well as in venous congestion of the inferior colliculus. Concomitant cochlear dysfunction should have been considered as a cause of hearing loss in this patient, or ruled out by further examination.

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Strauss replies:

We appreciate Kofler's comment on our paper and his interest in this unusual and still poorly understood clinical picture. We agree that the brain stem auditory potentials (BAEPs) after contralateral stimulation do not clearly point to a lesion of the ipsilateral colliculus; however, to our knowledge the neurophysiology of auditory pathways within the brain stem is not yet fully understood. For example, in our series of more than 300 cases of acoustic neurinoma monitoring we have made the observation that the contralateral wave V is much more pronounced compared with the ipsilateral wave V. The advantage of this case report is the preoperative and postoperative radiological and clinical documentation. The delayed onset of symptoms several days after the surgical procedure, the lack of effect of calcium blocker therapy—actually the patient's pure tone audiogram and speech discrimination deteriorated under nimodipine treatment—and the hearing improvement after heparinisation do not suggest vasospasm as the underlying pathophysiological mechanism. The literature on this rare yet important phenomenon of contralateral hearing loss after cerebellopontine angle surgery is purely speculative. By contrast this case report follows a straight course, which started at surgery with dissection of the pontotrigeminal vein, followed by a delayed contralateral hearing loss, and ended with a lesion of the ipsilateral colliculus. This lesion was not documented on preoperative MRI. Taking these findings into consideration, together with the neurophysiological findings of BAEPs in a still not fully understood auditory pathway within the brain stem, the "isolated vasospasm theory" seems unlikely.

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