This is the first demonstration of paroxysmal neurogenic hypertension triggered by a seizure in a patient with complex partial seizure. The diagnosis of complex partial seizure was supported by the following: focal EEG changes during a subsequent absence seizure; no reproduction of absence symptoms during drug induced paroxysmal hypertension; characteristic hippocampal atrophy on MRI⁴; and complete response to anticonvulsant drugs. Other possible diagnoses including brain stem tumour, pheochromocytoma, and renal artery stenosis were excluded by appropriate imaging and neurohormonal analysis. Pseudoseizures were excluded on the basis of the EEG findings and the rapid response to treatment. Although rapid increases in MNSA and heart rate have been found during panic attacks, paroxysmal hypertension and loss of consciousness are not consistent features.⁵

The paroxysm consisted of simultaneous hypertension and tachycardia associated with sweating and facial pallor during the absence attack. We suggest that this is secondary to a generalised increase in sympathetic activity causing vasoconstriction and increased cardiac output. This is supported by (a) increased MNSA and heart rate despite progressive rise in blood pressure; (b) symptomatic blood pressure overshoot; (c) noradrenaline increased to over seven times the normal tilt levels; (d) prominent low frequency (0.1 Hz) oscillations in blood pressure and MNSA during recovery.² These low frequency oscillations (0.1 Hz) are thought to be secondary to changes in brain stem sympathetic activity separate from the effects of respiration, which are generally of a higher frequency (0.2 Hz). We emphasise that the initial increase in MNSA occurred when blood pressure was increasing and so was not baroreflex mediated as would be expected for respiratory or normal brain stem low frequency oscillations.

We hypothesise that this generalised increase in sympathetic activity is permitted by a transient interruption of baroreflex feedback inhibition during the seizure. We think that this is a unique recording of transient baroreflex failure characterised by a rapid and generalised increase in sympathetic activity, overriding the baroreflex afferents in the brain stem. It has long been suspected that paroxysmal hypertension occurs in complex partial seizures but to date, ambulatory monitoring has only demonstrated changes in heart rate.⁶ Ambulatory beat to beat blood pressure monitoring would allow closer study of this phenomenon and its possible relation to sudden cardiac death in epileptic patients. Finally, this a good example of an episodic medical condition which may be very difficult to diagnose. Occasionally, when an episode is seen fortuitously in the laboratory, we may identify pathophysiology previously suspected but not actually seen.

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- 1 Valbo AB, Hagbath KE, Torebjork HE, *et al.* Somatosensory, proprioceptive and sympathetic activity in human peripheral nerves. *Physiol Rev* 1979;59:919-57.
- 2 Eckberg DL, Sleight P. *Human baroreflexes in health and disease*. Oxford: Oxford University Press, 1992:255-99.
- 3 Jardine DL, Melton IC, Crozier IG, *et al.* The neurohormonal response to head-up tilt and its role in vasovagal syncope. *Am J Cardiology* 1997;79:1302-6.
- 4 Lee DH, Gao F, Rogers JM, *et al.* MR in temporal lobe epilepsy: adults with pathological confirmation. *Am J Neuroradiol* 1998;19:19-27.
- 5 Wilkinson DJC, Thompson JM, Lambert GW, *et al.* Sympathetic activity in patients with panic disorder at rest, under laboratory mental stress, and during panic attacks. *Arch Gen Psychiatry* 1998;55:511-20.
- 6 Devinsky O, Price BH, Cohen SI. Cardiac manifestations of complex partial seizures. *Am J Med* 1986;80:195-202.

Compression by looping and perforation of the facial nerve by the anterior inferior cerebellar artery: a possible cause of facial tic

Hemifacial spasm is a distressing, common, and well defined condition, which is difficult to treat. It is characterised by clonic and tonic contractions of the muscles supplied by the facial nerve, is intermittent, and is usually worsened by fatigue or emotional upsets.

A clinical picture similar to that of hemifacial spasm is also seen in trigeminal neuralgia, another condition for which widely varying pathophysiological bases, including vascular compression, have been proposed.¹

In our case postmortem exploration of the posterior cranial fossa disclosed a strikingly abnormal relation between the anterior inferior cerebellar artery and VIIth cranial nerve. Such abnormal vascular anomalies around the facial nerve are repeatedly reported in the literature, and they seem to be closely correlated with hemifacial spasm.²

We took the cadaver of a man who had died aged 51, from the dissection material held by the First Department of Anatomy at the University of Vienna.

A square 2 cm×2 cm was drilled in the centre of the skull cap. formalin:water (1:5; 30 ml) was injected subdurally. The cadaver was left in the cold room for 24 hours. Then a mixture of phenol:formalin:water (3:1:10) was introduced into the femoral artery about 5 cm below the inguinal ligament through a 3 mm diameter cannula. Before preparation the cadaver remained in this phenol:formalin:water mixture for about 6 to 8 months.

The vascular anomaly reported was noted when the brain was moved carefully backwards. The brain, which was otherwise normal, was then dissected out of the cranial cavity. The external radius of the anterior inferior cerebellar artery was determined by a digital gauge.

During the exploration of the posterior cranial fossa of the cadaver, we encountered an unusual course of the right anterior inferior cerebellar artery (1.20 mm diameter), which arose from the lower third of the basilar artery and passed through the facial nerve 1.5 cm from the cerebellopontine angle, forming a ring around the nerve about 0.5 cm proximal to the point of penetration and compressing it at several points (fig 1).

The anterior inferior cerebellar artery first ran vertically downwards closely following the basilar and the right vertebral arteries. Then it took a horizontal course until it made a V shaped angle before penetrating the seventh cranial nerve. After it had passed through the facial nerve, with about one third of the fibres above it and two thirds below, its course made a ring around the seventh cranial nerve and disappeared between the cerebellum and the pons into the deep tissue. Along its course a branch went off to the choroid plexus of the fourth ventricle.

The cause and treatment of hemifacial spasm remain controversial. Whereas some theories postulate a cerebral or brain stem mechanism for its origin, others suggest that the causative lesion is within the facial nerve, either within the posterior fossa or at a more distal location.

Our unusual finding lends support to the neurovascular compression theory to explain the aetiology of this disorder as we would like to point out the unusual nature of this

Figure 1 Inferior surface of the brain. A=Anterior inferior cerebellar artery; F=facial nerve (seventh cranial nerve); C=cerebellum; P=pons; V=vestibulocochlear nerve (eighth cranial nerve).

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 antero-caudal 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 hypoacusia 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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- 1 Kitt CA, Gruber K, Davis M, *et al*. Trigeminal neuralgia: opportunities for research and treatment. *Pain* 2000;85:3-7.
- 2 Fukuda M, Kameyama S, Honda Y, *et al*. Hemifacial spasm resulting from facial nerve compression near the internal acoustic meatus. *Neurol Med Chir* 1997;37:771-4.
- 3 Nagaseki Y, Omata T, Ueno T, *et al*. Prediction of vertebral artery compression in patients with

hemifacial spasm using oblique sagittal MR imaging. *Acta Neurochir (Wien)* 1998;140:565-71.

- 4 Ryu H, Yamamoto S, Sugiyama K, *et al*. Hemifacial spasm caused by vascular compression of the distal portion of the facial nerve. *J Neurosurg* 1998;88:605-9.
- 5 Jannetta PJ. Typical or atypical hemifacial spasm. *J Neurosurg* 1998;89:346-7.

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 flytche and Howard⁵ 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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- 1 Chaudhuri A. Charles Bonnet syndrome: an example of cortical dissociation syndrome affecting vision [letter]? *J Neurol Neurosurg Psychiatry* 2000;69:704-5.
- 2 Cole M. When the left brain is not right the right brain may be left: report of personal experience of occipital hemianopia. *J Neurol Neurosurg Psychiatry* 1999;67:169-73.
- 3 Flournoy T. Le cas de Charles Bonnet. Hallucinations visuelles chez un vieillard opéré de la cataracte. *Archives de Psychologie (Geneve)* 1902;1:1-23.
- 4 de Morsier G. Les automatismes visuels. (Hallucinations visuelles retro-chiasmiques). *Schweiz Med Wochenschr* 1936;66:700-3.

- 5 flytche DH, Howard RJ. The perceptual consequences of visual loss: "positive" pathologies of vision. *Brain* 1999;122:1247-60.

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 study² and only two of 30 variables (7%) in the other³ 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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