lateral ventricles. Although slow growing and rarely large enough to create neurological symptoms, they can occasionally cause seizures, headaches, visual field defects, or gait disturbances.1 However, there are no references in the literature to ependymal cysts related to psychotic symptoms. Those documented include confusion, disturbances of memory, and mental deterioration, mental slowness and poor concentration, irritability and a personality disturbance improved by the extirpation of a cyst exerting a mass effect in the right sylvian fissure.1 Several reports have indicated that psychotic symptoms related to other intracranial cysts, such as arachnoid2 or colloid cysts, have disappeared after extirpation of the cyst. However, almost all of the mentioned cases were accompanied by some neurological symptoms, and these cysts showed pronounced mass effects. The present case is the first report of an ependymal cyst in a patient without neurological symptoms, but with severe psychiatric symptoms (depressed mood, agitation, depersonalisation, and ideas of suicide), especially in the context of the cervical sympathetic paralytic paradigm. Such an inverse temporal pattern between autonomic features and pain in cluster headache suggests another unrelated disease. Importantly, attacks of cluster headache that occurred between the ages of 25 to 33 years in this patient were not associated with Horner's syndrome, and, the index pain attacks were identical; it would be useful to know if any subsequent cluster headache attacks are so associated with the usual temporal profile. The quest for a definitive diagnosis of the underlying cause of Horner's syndrome is often fruitless.2 A theoretical possibility of viral infection, especially that of herpes simplex virus—often found in the trigeminal, superior cervical sympathetic, and cranial parasympathetic ganglia—has been advanced; painful periodic activation of the virus may often occur without skin lesions.3 In a systemic influenza-like illness with accompanying viral vestibular neurinits,4 it is entirely conceivable that a fortuitous concurrent self-limited right sided viral superior cervical sympathetic ganginitis was also associated. Virological studies might lend insight in otherwise inexplicable Horner's syndrome.

A pupillometric pattern consistent with a sympathetic third neuron pattern seems to be unusual in cluster headache, having been seen in only about 15% of patients.4 Influenza other than sympathoparalytic also operate in producing the ptosis and miosis of cluster headache. Eyelid oedema in cluster headache and chronic paroxysmal hemicrania indicate that clinically significant oculocutaneous inflammation develops in both conditions, probably through antidromic ophthalmic nerve discharge. Eyelid and nasal oedema may close the eye mechanically—a form of pseudo or apparent ptosis.5 Such non-paralytic purely mechanical miosis may occur in cluster headache without miosis.6 Focal episodic inflammatory oedema of the upper eyelid in cluster headache (or chronic paroxysmal hemicrania) would seem itself to periodically exacerbate mechanical ptosis, as was manifest in this patient. Furthermore, intraocular pressure rises have been shown in both cluster headache (statistically insignificant) and chronic paroxysmal hemicrania; a biologically remarkable 298% increase in intraocular pressure was, however, shown on the symptomatic side in one subject with cluster headache.7 Intraocular prostaglandin or substance P release causes miosis and increase in intraocular pressure,7 consisting of a form of oculoreflexive reflex. Mechanical stimulation of the ophthalmic nerve through variations in intraocular pressure, both with and without stellate ganglioneuroma, causes miosis through an antidromic reflex.8 Additionally, the interpretation of pupillary dilation lag—tardy dilatation being generally accepted—are the note ocular sympathetic deficit—must be guarded in conditions associated with pain because pain associated central sympathetic tone is likely to require a well developed degree of central sympathetic tone.9 Importantly, the miosis of Horner's syndrome is never maximal and is usually slight.10 Conversely, pain or emotional stress—such as pupillary dilation through the psychosensory reflex as well as neurogenic sympathetic lid retraction. These synergistic as well as antagonistic influences, besides residual ocular sympathetic deficit that outlasts the pain,11 dissociate pupillary miosis/dilation lag and ptosis from each other as well as from the severity of individual cluster headache attacks. Intriguingly, in chronic paroxysmal hemicrania, the trigeminal nerve occurs without miosis. The development of ptosis with or without miosis in both cluster headache and chronic paroxysmal hemicrania carcinoma cannot be simply explained in relation to pure oculomotor paralysis.

The lack of salivation in disorders characterised by lacrimation and nasal congestion/rhinorrhoea as well as the inconsistency of perioperative findings and postoperative results in connection with procedures directed on parasympathetic structures remain unexplained.12 Diffuse antidromic trigeminal nerve excitation12 also cannot explain the lack of salivation. The development of headaches associated with components of both migraine and cluster headache after gasserian ganglion ablation13 attenuates the possible role of activation of the nucleus salivatorius.14 Lacrimal (ocular adnexal) gland and nasal innervation is associated with the branches of the ophthalmic nerve. Given an intracranial source of generation of sympathetic trigeminal—sympathetic cervical—for the nasal nerve discharge through fluctuations of intraocular pressure lacrimation and nasal congestion/rhinorrhoea in both cluster headache and chronic paroxysmal hemicrania when represented as sympathetic peripheral/local orthodromic-antidromic reflex driven phenomena. This concept obviates the need to invoke a theoretically unacceptable "selective" cranial parasympathetic barrage. Lacrimation and nasal congestion/rhinorrhoea are not features of glaucoma in general. The rapidity of rise of intraocular pressure in cluster headache and chronic paroxysmal hemicrania is probably 30 seconds in chronic paroxysmal hemicrania—might be critical to the triggering of an aberrant antidromic ophthalmic division trigeminal discharge that results in "autonomic" manifestations.

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References

Peatfield replies:
I wholly agree with Gupta that independent mechanisms for the headache and Horner’s syndrome are required to explain the sequence of events in my patient. It is certainly possible the Horner’s syndrome is entirely coincidental although the correlation we observe suggests otherwise. Unfortunately, follow up information on my patient is not available.

I must confess to unwillingness to invoke a viral infection in paranysmaphagic ganglia to explain the pathogenesis of typical cluster headache, which even in this patient proved to be a spontaneously reversible periodic syndrome responding to corticosteroids among a wide variety of pharmacologically active agents.

Dr Gupta’s second suggestion, that a purely mechanical process can be related to a rise in intracranial pressure, is certainly ingenious but I find it difficult to accept that a sudden rise in intracranial pressure sufficient to set up a trigeminal nerve mediated axon reflex would not first be painful.

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Non-invasive carotid imaging
We agree with most of the comments made by Sellar in his comprehensive review of imaging techniques for displaying blood vessels of the head and neck.1 However, by contrast with Sellar, we would argue that conventional invasive angiography is no longer an essential component of the preoperative work up for prophylactic internal carotid endarterectomy; the combination of ultrasound and magnetic resonance angiography (MRA) is satisfactory for the vast majority of patients. In coming to this conclusion, we considered two key issues. Firstly, to what risk do we expose patients with symptomatic carotid occlusive disease when we perform invasive, intra-arterial contrast angiography? Secondly, do non-invasive techniques reliably identify appropriate candidates? (using currently available ECST criteria this would be those patients with \( > 70 \)% stenosis of the symptomatic internal carotid artery).

In assessing the risk of angiography, Sellar 2udes suspect(s) that the complication rate remains at around 1% in most centres . . . , but provides little appropriate prospective evidence to support this figure. The risks of conventional angiography clearly vary according to which group of patients are studied. Patients with symptomatic carotid stenosis seem to at greater risk than other groups and several studies have shown that the risk increases with increasing degrees of stenosis. This is particularly important because, in many centres, patients with mild disease are effectively excluded out of non-invasive techniques such as ultrasound. We therefore need to know the risks of angiography for the group of patients with moderate stenosis, as these are the patients who will be exposed to the test. Few studies have looked specifically at this group.

In a prospective study of 200 such patients in our centre, Davies and Humphrey found a 4% stroke rate for 72 hours postangiography, with 2% death or serious disability.2 This figure intuitively seems high. However, it is not dissimilar to other studies, provided the appropriate patients are considered. In a study from Edinburgh, Hankey et al. reported an overall stroke rate of 2% with a permanent deficit rate of 1.3%. From their paper it is possible to calculate the postangiographic stroke rate in those patients with \( > 50 \)% stenosis. The figures are those of an angiographic stroke risk of 5–5% and a permanent deficit risk of 3–1%. The benefits of carotid endarterectomy depend on a low surgical complication rate, as shown by the ECST and NASCET studies. Neither of these studies included the angiographic risk in their analyses. In borderline decisions, the risk of angiography, added to the surgical risk, could outweigh the benefits of endarterectomy.

The accuracy of non-invasive techniques is rightly assessed by comparison with invasive intra-arterial angiography, and Sellar concludes that ultrasound and MRA in detecting \( > 70 \)% stenosis or more is unreliable, respectively of how accurate the method being assessed is. This is because of the variation in reporting that exists, both between different observers and also between the same observer reporting on separate occasions. Experienced neuroradiologists reporting the same conventional angiograms can disagree by considerable amounts. In a recent study we found that the mean magnitude of disagreement is between 52% in our ward was 8–10% and that clinically important disagreements were reported in up to 6% of angiograms.3 Comparison of other modali ties with angiography will inevitably result in sensitivities and specificities considerably less than 100%, simply due to this variability in reporting.

Distinguishing lesions from very tight stenoses and occlusions can be reliably achieved with noninvasive tests. Colour duplex, in combination with continuous wave examination, has improved the ability of ultrasound to distinguish between near occlusion and complete occlusion and, provided the correct 2D sequence is employed, MRA is highly reliable in making this distinction. Indeed, in situations of very slow flow, we have found that 2D MRA sequences can be easier to interpret than conventional angiograms.

As Sellar points out, ultrasound examination of the carotid arteries does require experienced operators if the results are to be relied on. This should not be used as an argument against the technique in general. By the same token the writer is incorrect in abandoning carotid endarterectomy itself, as the operation requires highly experienced surgeons, not available to all centres. The solution is to ensure a more thorough and effective use of ultrasound, which would strongly endorse the suggestion that prospective audit be maintained locally for any centre offering screening ultrasound and considering endarterectomy.

The concern raised by Sellar regarding the signal gaps that appear on magnetic resonance angiograms beyond a tight stenosis has not proved to be a problem in our experience. Angiography is the issue of co-existent carotid siphon disease, which cannot be picked up reliably by duplex ultrasound. Our experience has been that significant siphon disease is a relatively uncommon finding. Furthermore, we are not convinced that its presence should alter the decision to perform endarterectomy, a similar conclusion to studies specifically looking at this question.4

In our unit we now use a combination of ultrasound and MRA for preoperative assessment of patients, and, like others, have found this highly successful. We reserve conventional angiography for those patients in whom the non-invasive investigations disagree significantly (an infrequent occurrence). In a situation where non-invasive methods clearly indicate the presence of a tight stenosis, we do not think it is advisable to expose a patient to the risks of invasive angiography.

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