lateral ventricles. Although slow growing and rarely large enough to create neurologi-
cal symptoms, they can occasionally cause seizures, headaches, visual field defects, or
gait disturbances.1 However, there are no references in the literature to epidymal cysts related to psychotonic symptoms. Those
documented include confusion, disturbances of memory, and mental deterioration,1 mental-
tal slowness and poor concentration, and irritability and a personality disturbance
improved by the extirpation of a cyst exerting
a mass effect in the right sylvian fissure.1
Some reports have indicated that the psychiatric
symptoms related to the cysts in the
medial temporal lobes, especially in schizophrenia patients,
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**MATTERS ARISING**

Painless Horner’s syndrome in cluster headache

Dissociation between autonomic dysfunction
and pain during cluster headache, with the
painless Horner’s syndrome preceding
the headache attacks by a considerable interval
(see Pearfield1), cannot be resolved 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 associ-
ated 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 defini-
tive diagnosis of the underlying cause of
Horner’s syndrome is often fruitless.2
A theoretical possibility of viral infection, espe-
cially that of herpes simplex virus—often found in the trigeminal, superior cervical sympathetic, and cranial parasympathetic
ganglia—has been advanced; painful peri-
odic activation of the virus may often occur
without skin lesions.3 In a systemic influenza-like illness with accompanying
viral vestibular neuritis,4 it is entirely con-
ceivable that a fortuitous concurrent self
limited right sided viral superior cervical sympathetic ganglionitis was also associated
with the Horner’s syndrome. Virolological studies might lend insight in other-
wise 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.1
Influences other than sympatheticparasympathetic also
operate in producing the ptosis and miosis
of cluster headache. Eyelid oedema in cluster
headache and chronic paroxysmal hemi-
crania indicate that clinically significant
ocular adnexal inflammation develops in both
conditions, probably through anti-
cholinergic ophthalamic nerve discharge. Eyelid
oedema may close the eye mechanically—a
form of pseudo or apparent ptosis.5 Such
non-paralytic purely mechanical ptosis may
occur in cluster headache without miosis.6
Episodic inflammatory oedema of the
upper eyelid in cluster headache (or chronic paroxysmal hemicrania) would seem itself to
periodically exacerbate mechanical ptosis, as was manifested in this patient. Furthermore,
intracranial pressure rises have been shown in
both cluster headache (statistically insignificant) and chronic paroxysmal hemicr-
nia; a biologically remarkable 298.
Intracranial pressure was, however, shown on the symptomatic side in one
subject with cluster headache.7 Intracranial
prostaglandin or substance P release causes miosis and increase in intracranial pressure,7 constituting a form of ocularosynthetic reflex. Mechanistic stimulation of the ophthalmic nerve through variations of intracranial pres-
sure, both with and without stellate gal-
glioneuroma, causes miosis through an antidromic reflex.8 Additionally, the inter-
pertation of pupillary dilatation lag—tardy dilatation being generally accepted—must
denote ocular sympathetic deficit—must be guarded in conditions associated with pain
because pain associated central sympathetic tone is not always normal and requires a well developed degree of central sympathetic tone.9 Importantly, the miosis of Horner’s syndrome is never maximal and is usualy slight.4 Conversely, pain or emo-
tional stimulation may be sufficient to
cause a pupillary dilatation through the psychosen-
tory reflex as well as neurogenic sympathetic lid retraction. These synergistic as well as
antagonistic influences, besides residual ocu-
lar sympathetic deficit that outlasts the pain,9 dissociate pupillary miosis/dilatation
lag and ptosis from each other as well as from the severity of individual cluster
headache attacks. Intriguingly, in chronic paroxysmal hemicrania, this miosis
occurs without miosis.10 The development of ptosis with or without miosis in both cluster
headache and chronic paroxysmal hemicrana-
ia cannot be similarly discerned, consequently to reflect pure ocular sympathetic deficiency.

The lack of salivation in disorders charac-
terised by lacrimation and nasal congestion/ rhinorrhea as well as the inconsistency of perioperative findings and postoperative results in connection with procedures directed on parasympathetic structures remain unexplained.11 Diffuse antidromic trigeminal nerve excitation12 also cannot explain the lack of salivation. The develop-
ment of headaches associated with compo-
ents of both migraine and cluster headache after gasserian ganglial 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 trigeminal trigeminal—activity within the nerve discharge through fluctuations of intracranial pressure lacrimation and nasal congestion/rhinorrhea in both cluster headache and chronic paroxysmal hemicrana-
ia may represent a response to a sympathetic peripheral/local orthodromic-antidromic reflex driven phenomena. This concept obviates the need to invoke a theoretically unacceptable “selective” cranial parasympa-
thetic barrage. Lacrimation and nasal con-
genarion/rhinorrhea are not features of glaucoma in general. The rapidity of rise of intracranial pressure in cluster headache and chronic paroxysmal hemicrania is usually less than 30 seconds in chronic paroxysmal hemicrania—might be critical to the triggering of an aberrant antidromic ophthalamic division trigeminal discharge that results in “auto-
nomous” manifestations.

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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 Gupta suggests otherwise. Unfortunately, follow up information on my patient is not available.

I must confess to unwillingness to invoke a viral infection in parasympathetic 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. However, by contrast with Sellar, we would argue that conventional carotid 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 . . . 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 be 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 being assessed and/or treated by non-invasive techniques such as ultrasound.

We therefore need to know the risks of angiography for the group of patients with moderate risk, could even stenoses 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 in 72 hours postangiography, with 2% death or serious disability. 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%. From their paper it is possible to calculate the postangiographic stroke rate in those patients with > 50% stenosis. The figures are those of 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 quotes sensitivities for ultrasound of the order of 81% to 85%. This is in agreement with a recent meta-analysis of non-invasive techniques which reported sensitivities of 86-86% and specificities of 94%-94% for carotid ultrasound and MRA in detecting 70% stenosis or more.4 When interpreting sensitivity and specificity data it is important to realise that values less than 100% are inevitable, regardless 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 was 12% between consultants in our unit was 8-10% and that clinically important disagreements were reported in up to 6% of angiograms.5 Comparison of other modalities with angiography will inevitably result in sensitivities and specificities considerably less than 100%, simply due to this variability in reporting.

Distinguishing occlusions from very tight stenoses can be reliably achieved with non-invasive tests. Colour duplex, in combination with continuous wave examination, has improved the ability of ultrasound to distinguish very tight stenoses from occlusions and, provided the correct 2D sequence is employed, MRA is reliably 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, routine 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, 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 training of ultrasonographers, we would strongly endorse the suggestion that prospective audit be maintained locally for any centre offering screening ultrasound and counselling 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. Aortic 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.

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 the more invasive 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 then we are not dissatisfied to reveal to expose a patient to the risks of invasive angiography.

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