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. However, there are few 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. Some reports have indicated that psychiatric symptoms related to other intracranial cysts, such as arachnoidid 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, ideas of observation) which resolved postoperatively. The improvement of his symptoms continued for more than six months, suggesting that it was not a non-specific effect of the operation. Several previous reports have indicated that cysts in the temporal region are associated with psychiatric symptoms. However, it is difficult to account for the relation between the location of the cyst in this patient and his psychiatric symptoms, including depersonalisation and ideas of observation, which were similar to those seen in schizophrenia. Previous reports documenting enlargement of the lateral ventricle, especially the left posterior horn, in schizophrenic patients, may explain why the brain region surrounding his cyst was associated with those symptoms, and why the release of the region from compression by cyst resection resulted in his improvement. Further reports and discussion will be needed to evaluate possible mechanisms of ependymal cyst induced psychiatric symp-toms, the relation between the region of the cyst and the type of psychiatric symptoms, and how neurosurgical interventions affect these symptoms.

In conclusion, we suggest that an ependymal cyst without associated neurological symptoms may cause psychiatric symptoms. Thus a physician’s careful attention to psychiatric symptoms in patients with an intracranial cyst is essential, including an evaluation of the indications for surgery.

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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 Pearlfeld’), 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 35 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. 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 per-iodic activation of the virus may often occur without skin lesions. In a systemic influenza-like illness with accompanying viral vestibular neuritis, it is entirely conceivable that a fortuitous concurrent self limited right sided viral superior cervical sympathetic ganglionitis was also associated. Virological 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, but been seen in only about 15% of patients. Influences other than sympathoparalytic also operate in producing the miosis and miosis of cluster headache. Eyelid oedema in cluster headache and chronic paroxysmal hemicrania indicates that clinically significant ocular adnexal inflammation develops in both conditions, probably through antidromic ophthalmic nerve discharge. Eyelid oedema may close the eye mechanically—a form of pseudo or apparent ptosis. Such non-paralytic purely mechanical miosis may occur in cluster headache without miosis. Islet cephalic inflammatory oedema of the upper eyelid in cluster headache (or chronic paroxysmal hemicrania) would seem itself to periodically exacerbate mechanical miosis, as was manifest in this patient. Furthermore, intracranial pressure rises have been shown in both cluster headache (statistically insignificant) and chronic paroxysmal hemicrania; a biologically remarkable 299% increase in intracranial pressure was, however, shown on the symptomatic side in one subject with cluster headache. Intracranial prostanoid or substance P release causes miosis and increase in intracranial pressure, constituting a form of oculosympathetic reflex. Mechanical stimulation of the sympathetic nerve through variations of intracranial pressure, both with and without stellate ganglionectomy, causes miosis through an antidromic reflex.4 Additionally, the interpretation of pupillary dilatation lag–tardy dilatation being generally attributed to note oculo sympathetic deficit—must be guarded in conditions associated with pain because pain associated central sympathetic tone due to allodynia requires a well developed degree of central sympathetic tone. Importantly, the miosis of Horner’s syndrome is never maximal and is usually slight. Conversely, pain or emotional stress can also induce pupillary dilatation 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, 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, Horner’s syndrome occurs without miosis. The development of ptosis with or without miosis in both cluster headache and chronic paroxysmal hemicrania cannot be simplistically construed to reflect pure oculo sympathetic deficiency.

The lack of salivation in disorders characterised by lacrimation and nasal congestion/ rhinorrhea as well as the inconsistency of peripheral/local sympathetic effects in patients with cluster headache and chronic paroxysmal hemicrania are not easily explained. Diffuse antidromic trigeminal nerve excitation4 also cannot explain the lack of salivation. The development of headaches associated with components of both migraine and cluster headache after gasserian ganglial ablation15 attenuates the possible role of activation of the nucleus salivatorius. Lacrimal (ocular adnexal) gland and nasal innervation is associated with the branches of the opthalmic nerve. Given an intracranial source of generation of trigeminal—facial sympathetic preganglionic neurons, 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/rhinorrhea are not features of glaucoma in general. The rapidity of rise of intracranial pressure in cluster headache and chronic paroxysmal hemicrania is less than 30 seconds in chronic paroxysmal hemicrania—might be critical to the triggering of an aberrant antidromic ophthalmic division trigeminal discharge that results in “automatic” manifestations.

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3. Graham JR. Cluster headache: the relation to...

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 I observed 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 ingenuous 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 cerebral vascular disease when we perform invasive, intra-arterial contrast angiography? Secondly, do non-invasive techniques reliably identify appropriate patients? (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 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 referred out for non-invasive techniques such as ultrasound.
We therefore need to know the risks of angiography for the group of patients with moderate to severe 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 4% stroke mortality and 3% temporary-TIA's in the 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 rate 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 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 83-86% and specificities of 94-99% 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 in the classification of comorbidity 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 between lesions from very tight stenoses (which 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 stenosis from 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, ultrafast 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, non-invasive techniques could abandon 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 ultrasound operators, which would strongly endorse the suggestion that prospective audit be maintained locally for any centre offering screening ultrasound and carotid endarterectomy.2
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. Sellar states that the signal void on 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.6
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 operation 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 high risk stenosis we think it is justifiable to expose a patient to the risks of invasive angiography.

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