probable multiple system atrophy\(^1\) was made in 10 (eight men, two women) and of idiopathic Parkinson’s disease in 14 (12 men, two women) patients. Median age was 5\(^{2}\) (range 41–73) years in the multiple system atrophy group and 56 (range 35–76) years in the idiopathic Parkinson’s disease group. Median disease duration was 3–5 (range 1–11) years in the multiple system atrophy group and 4–5 (range 1–10) years in the idiopathic Parkinson’s disease group. A concentric needle EMG and a single fibre EMG\(^2\) of the external anal sphincter were performed. In concentric needle EMG 10 to 20 different motor unit potentials (MUPs) were recorded. Mean MUP duration, mean MUP amplitude, and MUP polyphasicity (percentage of polyphasic MUPs) were expressed for each patient. Mean muscle fibre density\(^3\) per insertion site was determined by single fibre EMG. A non-parametric (Mann-Whitney) test was used to analyse the results.

The difference in the mean MUP duration was close to significance (6.7 ms and 5.1 ms, \(P = 0.09\)), being longer in the multiple system atrophy group, whereas there was no difference in the mean MUP amplitude (0.50 mV and 0.45 mV, \(P = 0.79\)). The MUP polyphasicity was significantly higher in patients with multiple system atrophy (68% and 40%, \(P = 0.0002\), as was fibre density (3.3 and 2.1, \(P = 0.0003\)). No patient with multiple system atrophy had MUP polyphasicity below 50% and fibre density below 2 (figure). On the other hand no patient with idiopathic Parkinson’s disease had MUP polyphasicity above 60% and fibre density above 3. Thus MUP polyphasicity of 60% and/or a fibre density of 3 could be a “cut off” level to distinguish patients with multiple system atrophy from those with idiopathic Parkinson’s disease. In this respect, the sensitivity and specificity of concentric needle EMG would be 80% and 93%, respectively, and for single fibre EMG these estimates would be 80% and 100%. The diagnostic value of the two tests is similar.

Our neurophysiological data were obtained from small groups of patients, but they had similar ages, male to female ratios, and durations of disease. Whether the results from patients with idiopathic Parkinson’s disease deviate from normal or not will have to be ascertained in future studies. Importantly, however, the results from the patients with multiple system atrophy showed a distinctly more abnormal pattern, particularly much increased polyphasicity of MUPs on concentric needle EMG and fibre density on single fibre EMG, compared with patients with idiopathic Parkinson’s disease.

In conclusion, a polyphasicity of 60% or above and/or fibre density of 3-0 or above in the anal sphincter in a patient with parkinsonian symptoms should raise the possibility of multiple system atrophy.

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Ependymal cyst and psychiatric symptoms

Many reports have documented the relation between intracranial cysts and psychiatric symptoms.\(^1\)\(^2\) Ependymal cysts are rare, non-invasive, benign tumours.\(^3\) We report a patient with an ependymal cyst and psychiatric symptoms which improved with cyst resection.

A 22 year old right handed blue collar worker, with no personal or family history of psychiatric disorders, had had general fatigue, poor concentration, and insomnia since the age of 16. Gradually, psychiatric symptoms such as depressed mood, agitation, depersonalisation, ideas of observation, and suicidal ideation, without any disturbances of consciousness, appeared. These symptoms were partially alleviated. At the age of 21 his pronounced agitation caused him to extinguish a cigarette on his own hand, so he was admitted to the psychiatric ward at our hospital. A neurological examination, EEG, and SSF were normal. Brain MRI showed a cystic tumour (6 x 4 cm) in the posterior horn of his left lateral ventricle (figure A). This proved to be an ependymal cyst on subsequent pathological examination. We determined that the cyst did not require resection, because of the absence of abnormal neurological findings. The Wechsler adult intelligence scale-revised (WAIS-R) yielded a verbal intelligence quotient (IQ) of 67, a practical IQ (PIQ) of 70, and a total IQ of 63. His score on Bender’s gestalt test was 18 points. Major tranquilizers and mianserin (60 mg) were stopped, but symptoms were partialy alleviated and severe, so he was discharged after 99 days in hospital. However, he soon experienced a relapse, and in accordance with his wishes, cyst resection was performed. Using an endoscope, the wall and contents of the cyst were partially removed, and communication was established between the cyst and the body of the lateral ventricle anteriorly; this resulted in a reduction in cyst size (figure B). His psychiatric symptoms subsequently improved and had almost resolved within three months of the operation. His psychological test scores also greatly improved (WAIS-R total IQ 96 (VIQ 92, PIQ 105) and Bender’s gestalt test 10 points). Postoperatively, he has worked as a blue collar worker for the past six months without a relapse to his previous psychiatric symptoms.

Ependymal cysts are usually seen in the fourth ventricle, but sometimes occur in the
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 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, and 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 arachnoid12 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 epedymal cyst in a patient without neurological symptoms, but with severe psychiatric symptoms (depressed mood, agitation, depersonalisation, and irritability) and a personality disturbance 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 reports1–3 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, with the release of the region from compression by cyst resection resulting in his improvement. Further reports and discussion will be needed to evaluate possible mechanisms of epedymal cyst induced psychiatric sympotms, 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 epedymal 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**

**Painful 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 Pearfield’s), 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 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 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 of herpes simplex virus—often found in the trigeminal, superior cervical sympathetic, and cranial parasympathetic ganglia—has been advanced; painful paroxysmal activation of the virus may often occur without skin lesions.1 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 ganginitis was also associated. Virological studies might lend insight in otherwise inexplicable Horner’s syndrome. A pupillomotor pattern consistent with a sympathetic third neuron pattern seems to be unusual in 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 discharges. Eyelid oedema may close the eye mechanically—a form of pseudo or apparent ptosis.4 Such non-paralytic purely mechanical ptosis may occur in cluster headache without miosis.4 Ictal 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, intracranial pressure rises have been shown in both cluster headache (statistically insignificant) and chronic paroxysmal hemicraniain; a biologically remarkable 29% increase in intracranial pressure was, however, shown on the symptomatic side in one subject with cluster headache.4 Intracranial prostanoglandin or substance P release causes miosis and increase in intracranial pressure,3 constituting a form of oculovertical reflex. Mechanical stimulation of the ophthalmic nerve through variations in intracranial pressure, both with and without stellate ganglioneuroma, causes miosis through an antidromic reflex.4 Additionally, the interpretation of pupillary dilatation lag—tardy dilatation being generally to denote ocular sympathetic deficit—must be guarded in conditions associated with pain because pain associated central sympathetic tone is likely to dilate pupils requires a well developed degree of central sympathetic tone.5 Importantly, the miosis of Horner’s syndrome is never maximal and is usually slight.2 Conversely, pain or emotional states elicit other 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,5 dissociate pupillary miosis/dilatation lag and ptosis from each other as well as from the severity of individual cluster headache attacks. Intriguingly, in chronic paraspinal sympathetic reflex, which occurs without miosis. The development of ptosis with or without miosis in both cluster headache and chronic paroxysmal hemicrania cannot be simply attributed to the central sympathetic outflow and is assumed to reflect pure ocular sympathetic deficiency.

The lack of salivation in disorders characterised by lacrimation and nasal congestion/ rhinorrhoea as well as the inconsistency of periorbital findings and postoperative results in connection with procedures directed on parasympathetic structures remain unexplained.6 Diffuse antidromic trigeminal nerve excitation7 also cannot explain the lack of salivation. The development of headaches associated with components of both migraine and cluster headache after gasserian ganglial ablation8 attenuates the possible role of activation of the nucleus salivatorius.9 Lacrimal (ocular adnexal) gland and nasal innervation is associated with the branches of the ophthalmic nerve. Given an intracranial source of generation of trigeminal trodromic reflex, the nerve discharge through fluctuations of intracranial pressure lacrimation and nasal congestion/rhinorrhoea in both cluster headache and chronic paroxysmal hemicrania may represent the lack of salivation. The 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 congestion/rhinorrhoea 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 “auto-mano” manifestations.

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