probable multiple system atrophy\(^1\) was made in 10 (eight men, two women) and of idio-
pathic Parkinson’s disease in 14 (12 men, two women) patients. Median age was 57 (range 41–73) years in the multiple system atrophy group and 56 (range 35–76) years in the idopathic 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\(^3\) 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 polyphasicy (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 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 polyphasicy 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 polyphasicy below 50% and fibre density below 2 (figure). On the other hand no patient with idiopathic Parkinson’s disease had MUP polyphasicy above 60% and fibre density above 3. Thus MUP polyphasicy 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 simi-
lar.

Our neuropsychological 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 idio-
pathic 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 atro-
phy showed a distinctly more abnormal pat-
tern, particularly much increased polyphasicy of MUPS on concentric needle EMG and fibre density on single fibre EMG, compared with patients with idio-
pathic Parkinson’s disease.

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

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1 Quinn N. Multiple system atrophy: the nature of the beast. J Neurol Neurosurg Psychiatry 1989;49(suppl):78-89.
4 Pramstaller PF, Wenning GK, Smith SJM, Beck KO, Quinn NP, Fowler CJ. Nerve con-

Ependymal cyst and psychiatric symp-
toms

Many reports have documented the relation between intracranial cysts and psychiatric symp-
toms.\(^1\)\(^2\) Ependymal cysts are rare, non-
vasive, benign tumours.\(^4\) We report a patient with an ependymal cyst and psychi-
atric symptoms which improved with cyst resection.

MRI (axial view: T2 weighted image) (A) showing an ependymal cyst in the posterior horn of left lateral ventricle before surgery; (B) postoperatively, the cyst appears smaller.

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, agita-
tion, depersonalisation, ideas of observation, and suicidal ideation, without any distur-
bances of consciousness, appeared. These symptoms were sometimes so severe that he was unable to work. The patient was first diagnosed with depression at a psychiatric clinic, and was given a 20 mg dose of methylphenidate hydrochloride, which failed to alleviate his symptoms. 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 examina-
tion. 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 normal intelligence quotient (IQ) 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 symp-
ptomatically effective in treating his psychiatric symptoms, and 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 communi-
cation 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 sub-
sequently 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. Ep En dopymal 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 numerous references in the literature to ependymal cysts related to psychotic symptoms. These document 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 psychotic symptoms related to other intracranial cysts, such as arachnoid 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, we are unable 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 symptoms, 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 dysfunc-
tion 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 associ-
ted 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 peri-
odic activation of the virus may often occur without skin lesions. In a systemic influenza-like illness with accompanying viral vestibular neuronitis, it is entirely conceivable that a fortuitous concurrent self limited right sided viral superior cervical sympathetic ganglionitis was also associated. Virolological 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 headaches, having been seen in only about 15% of patients.1 Influences 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 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 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 manifest in this patient. Furthermore, intraocular pressure rises have been shown in both cluster headache (statistically insignificant) and chronic paroxysmal hemicrania, a biologically remarkable 298.7 increase in intraocular pressure was, however, shown on the symptomatic side in one subject with cluster headache.6 Intracocular prostaglandin or substance P release causes miosis and increase in intraocular pressure, constituting a form of ocularosympathetic reflex. Mechanical stimulation of the ophthalmic nerve through variations in intraocular pressure, both with and without stellate gan-


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