have occasionally monitored what appeared to be "psychogenic" attacks, but these clearly differed from nocturnal paroxysmal dystonia in being less stereotyped, lacking true dystonic posturing and truly abnormal motor patterns and in particular arising when the patient had already woken up. Nocturnal paroxysmal dystonia attacks on the contrary arise directly from sleep, and forerunning vegetative changes actually occur during sleep (figs 1, 2³). Polysomnography with audiovisual monitoring of the attacks is essential in this respect, and it is indeed difficult to imagine a conversion disorder (of which we could not find previous examples in the literature) arising when the patient is still asleep.

Besides polysomnography, several clinical features make us very reluctant to regard short-lasting nocturnal paroxysmal dystonia as an example of conversion behaviour. Nearly all of our cases were referred to us after long peregrinations with a label of "hysteria" or psychogenic disease, and some had undergone fruitless psychotherapy, relaxation techniques and psychopharmacology for many years. True conversion traits and "secondary" gain were absent in most patients, and the neurotic traits observed in some seemed the consequence rather than the cause of the disorder. Finally, no conversion behaviour responds promptly and selectively to carbamazepine, nocturnal paroxysmal dystonia relapses after withdrawal and again subsides after retreatment with this drug. The associated tonic-clonic or apparently partial sensory-motor seizures reminiscent of epilepsy in some patients instead led us to hypothesise an epileptic origin,² but the definite aetiology still escapes us.

Polysomnography has revealed an entirely new world of nocturnal phenomena. Not only is motor control during sleep still poorly defined in normal people, but we are just beginning to classify abnormal sleep-related motor events. The risk of lumping together different classes of disorders (and nocturnal paroxysmal dystonia itself comprises different entities) can only be avoided by careful monitoring of the ictal phenomena. In any case, regardless of clinical or laboratory features, we remain cautious in labelling as "psychogenic" any paroxysmal event which takes place during sleep.

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Non tumoral aqueduct stenosis and normal pressure hydrocephalus in the elderly

Sir: Drs Vanneste and Hyman, in their recent article¹ discussed the diagnosis of aqueduct stenosis. I agree with the authors' opinion that the metrizamide (CT) cisternographic features, showing a normal configuration of the basal cistern, filling of the fourth ventricle and no contrast in the third ventricle, indicate aqueduct stenosis (or obstruction). In addition, I accept that an additional metrizamide (CT) ventriculography is not always necessary to make a diagnosis of aqueduct stenosis.

I, however, cannot fully agree with the statement cited from the literature that plain CT appearances with the normal-sized 4th ventricle in contrast to markedly enlarged 3rd and lateral ventricles indicate aqueduct stenosis. As I have pointed out,² it is not infrequent in patients with communicating hydrocephalus to have a nearly normal-sized 4th ventricle. For this reason, it is not adequate to make a diagnosis of aqueduct stenosis on the basis of plain CT features showing an enlargement of the 3rd and both lateral ventricles, with a nearly normal-sized 4th ventricle.

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Vanneste and Hyman reply:

Aoki rightly states that hydrocephalus in combination with a normal fourth ventricle

on plain CT brain scan does not necessarily indicate aqueduct stenosis. We are not aware of articles or handbooks suggesting the contrary and in our report there is no trace of such an assertion. When closely read it will be clear that our article only refers to the fact that marked ventricularomegaly with a normal fourth ventricle is suggestive of aqueduct stenosis.

We performed cisternography in all patients with this potential diagnosis, as it is well-known that communicating normal pressure hydrocephalus may present with a normal fourth ventricle on plain CT scan.¹⁻³

Aoki apparently misinterpreted the first line of our discussion, transforming a diagnostic probability into a statement of fact. By advising that additional investigations should be performed when CT scanning suggests aqueduct stenosis, he seems to hold the same views on the question as we do.

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Intravascular coagulation in surgery for large acoustic neuromas

Sir: The observation of a haemorrhagic diathesis after excision of large acoustic neuromas in three patients by Mattock and Crookard¹ in a series of 67 consecutive patients is an intriguing one. Indeed, it is tempting to incriminate disseminated intravascular coagulation (DIC) as the underlying mechanism, although firm laboratory evidence to confirm this hypothesis was not provided. First, thrombin time and partial thromboplastin time tend to lengthen, and not to be shortened, during DIC. Moreover, additional laboratory information, including not only increase in fibrin degradation products (FDP), but also positive ethanol gelation tests, decrease in fibrinogen concentration and decreases in coagulation