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 brainscan 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 Crockard¹ 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

Matters arising

inhibitors (antithrombin III) are nowadays mandatory before the diagnosis of DIC can be made. Finally, the administration of fresh frozen plasma tends to aggravate DIC rather than controlling the haemostatic defect. The observed haemorrhage may therefore result from local surgical factors rather than systemic coagulation defects.

Changes in haemostasis including increase of fibrinolysis and decrease of plasma fibrinogen and blood platelet count, are not uncommon during and immediately after surgery, and were reviewed by Owen and Bowie.² The effects of cranial surgery upon coagulation have been studied less frequently. Coagulation defects have been observed after neurosurgery,³ increase of blood coagulation activity was reported after removal of meningiomas,⁴ and DIC was observed after removal of an oligodendroglioma.⁵

Mattock and Crockard state that a prospective study of coagulation during intracranial surgery is indicated. Such a study was performed recently, although not exclusively in patients with acoustic neuromas.⁶ In this study coagulation abnormalities occurred in the majority of patients, and more often than observed after general surgical procedures. There seemed to be a relationship between the extent of the intracranial procedure and the extent of the coagulation changes. Removal of deeply situated tumours, which necessarily involved more manipulation and damage of brain tissue than surgery of superficially situated tumours or extracerebral surgery, tended to be associated with a larger increase of FDP concentration, and with a decrease in plasma fibrinogen concentration. Damage to brain tissue, brain tumour cells, and blood vessels may lead to the liberation of thromboplastic material into the circulation, and, subsequently, to the conversion of fibrinogen into fibrin. Finally, fibrinolysis may result in an increase of FDPs. It might well be that the introduction of CUSA (Cavitron Ultrasonic Surgical Aspirator) contributes to an increased release of thromboplastic material into the circulation, and thus to an increased occurrence of coagulation changes during and after neurosurgical procedures. The exact nature of the coagulation defect after neurosurgery however, still needs to be defined.

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Mattock and Crockard reply:

We are grateful to Drs Van der Sande and Buller for their comments and helpful criticism.

Our intention was to raise doubt as to whether all bleeding during and after excision of large (diameter greater than 4 mm) acoustic neuromas was due entirely to failure of surgical haemostasis. It is our impression, and that of other neurosurgeons (personal communications), that in some cases bleeding occurs which cannot be readily explained on the basis of operative technique alone.

We fully accept that the supporting coagulation data in our original report were inadequate to make a diagnosis of disseminated intravascular coagulation (DIC). At the time we did not have the facility for more complex measures of haemostatic function. It was for this reason that we posed the question in the title and proposed that a formal systematic study be undertaken.

Happily we now have access to a specialised haemostasis laboratory and have embarked upon a prospective study. To date we have assessed 10 patients with acoustic neuroma during and immediately after surgery, and have included serial assays of platelet count, fibrinogen, prothrombin time, partial thromboplastin time, thrombin time, fibrin degradation products (FDPs), antithrombin III (ATIII) and Factor VII activity.

In contrast to our initial suggestion we have found so far no convincing evidence of

DIC. There are modest falls in platelet count, fibrinogen and ATIII during and after surgery, and only trivial rises in FDPs. However, one of the 10 individuals required re-exploration for intracranial bleeding and although he had no laboratory evidence for DIC he had a Factor VII level at the time of bleeding of only 0.41 IU/ml (normal range 0.5-2.0 IU/ml), which fell to 0.31 IU/ml a few hours later. Despite infusions of fresh frozen plasma the Factor VII level remained around 0.30 IU/ml for the next 12-24 hours.

As Factor VII is not consumed during normal coagulation, a low Factor VII level does not represent intravascular coagulation, but implies binding of the factor to tissue thromboplastin, either in the circulation or locally at the operative site. Such binding and depletion of Factor VII may encourage operative or postoperative haemorrhage. Although we wish to finish our study before formally reporting, it is possible that Factor VII depletion may be a mechanism for bleeding in some patients with acoustic neuroma, and theoretically for other neurosurgical patients.

Apnoea testing to confirm brain death in clinical practice

Sir: We read the paper by Van Donselaar *et al*¹ with great interest and are not surprised to learn of their failure to raise the pCO₂ of their six patients to 7.98 kPa (60 mm Hg) during apnoea testing. Their data indicate that all of their patients were hyperventilated (presumably to reduce intracranial pressure), resulting in a mean start-of-test pCO₂ of 2.8 kPa (21 mm Hg). It has been our experience, and the experience of others,²⁻⁴ that when the apnoea test is begun with the patient's pCO₂ in the hypocapnic range, the pCO₂ often does not reach 7.98 kPa (60 mm Hg) after 10-15 minutes of apnoea. This failure to reach the target pCO₂ value using the "USA" recommendations is due to the fact that these recommendations do not specify a starting pCO₂.⁵

It has been both our practice and recommendation⁶⁻⁸ to adjust the patient's minute ventilation so that the start-of-test pCO₂ is 4.79 kPa (36 mm Hg) or higher. Using this protocol, our patients exceeded the target pCO₂ of 7.98 kPa (60 mm Hg) in 27 of 28 apnoea tests of 10 minute duration. Our one patient failing to reach the target pCO₂ was relatively hypothermic (35.5