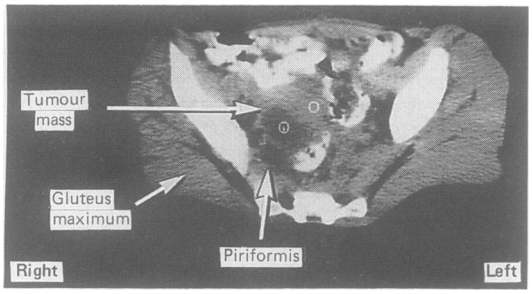

Figure 1 CT scan of the pelvis at the third sacral level. A tumour mass (endometrioma) lies in the region of the right sciatic notch.

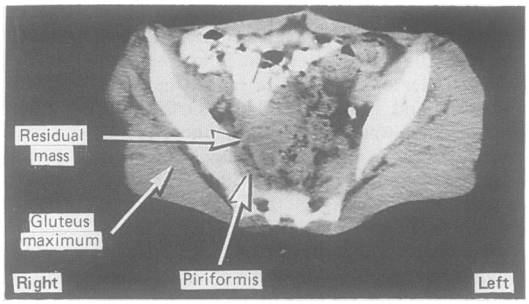

Figure 2 CT scan taken at the same level as fig 1. The endometrioma has largely resolved following danazol therapy.

seen 24 months after presentation she was asymptomatic and had a normal neurological examination.

Sciatic nerve endometriosis, though rare, may be presumptively diagnosed by a history of cyclic (catamenial) pain in a sciatic nerve distribution. Less frequently endometrial neural pain may be non-cyclical. Alternatively motor symptoms may predominate with painless foot drop occurring at each menses or as in our case, leg weakness may develop slowly and insidiously over many months.

Endometriosis may be suspected from a history of secondary dysmenorrhoea, dyspareunia or infertility. An unequivocal diagnosis can only be made by direct visualisation of the disease at the time of laparoscopy or laparotomy. Where the pelvis is found to be free of endometriosis, peritoneal "pockets" may be the only clue to the site of retroperitoneal implants of endometriosis on the sciatic nerve.[2] Ectopic uterine mucosa, once implanted on peripheral nerve, aggressively invades the epineurium and perineurium.[4] Physiological withdrawal of oestrogens and progesterone causes intraneural endometriomata to "menstruate" into adjacent tissue spaces and results in intrafascicular haemorrhage and dense fibrosis.[4]

Electromyographic studies in pelvic endometriosis have indicated that axonal degeneration is the dominant nerve fibre pathology.[4] Pelvic CT examination is often distinctive, showing a solid mass at the level of the sciatic notch which may enhance. Because of its frequency in menstruating women, endometriosis is probably the commonest cause of non-traumatic sciatic nerve compression at the level of the sciatic notch.

Medical treatment of endometriosis followed the observation that this disorder generally improved during the amenorrhoea of pregnancy or the menopause. Danazol, now considered the medication of choice, suppresses all endogenous stimuli to the endometrium and allows spontaneous atrophy of the disease.

With peripheral nerve involvement in endometriosis, danazol (400–800 mg daily) should be continued until there is neurological recovery or a sustained plateau of improvement. There must be long term neurological surveillance following cessation of danazol therapy since trials have shown recurrence rates as high as 39% within 37 months.[5]

While previous reports have suggested that surgical castration is the treatment of choice for peripheral nerve involvement in endometriosis, such treatment has never resulted in a reversal of foot drop.[3] Although the excision of endometrial implants from the sciatic nerve has often been effective in reducing sciatica, this procedure may aggravate or produce sciatic nerve weakness.[2] The complete and sustained recovery of crural weakness with danazol in this patient suggests that this should be the primary treatment for this complication of endometriosis, particularly when the patient is young or desires continuation of her reproductive function.

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1 Donaldson JO. Neurology of pregnancy. In: Walton JN, ed. *Major problems in neurology*, Vol 7. Philadelphia: WB Saunders, 1978: 23–25.
2 Torkelson SJ, Lee RA, Hildahl DB. Endometriosis of the sciatic nerve: a report of two cases and a review of the literature. *Obstet Gynaecol* 1988;71:473–7.
3 Hibbard J, Schreiber JR. Footdrop due to sciatic nerve endometriosis. *Am J Obstet Gynaecol* 1984;149:800–1.
4 Baker GS, Parsons WR, Welch JS. Endometriosis within the sheath of the sciatic nerve: report of two patients with progressive paralysis. *J Neurosurg* 1966;25:652–5.
5 Cohen MR. Laparoscopic diagnosis and pseudomenopause treatment of endometriosis with danazol. *Clin Obstet Gynaecol* 1980; 23:904–15.

Should clinicians be given hard copies of duplex carotid ultrasound images?

Duplex carotid sonography (DCS) is an accepted, non invasive method for assessing stenotic lesions of the extra-cranial carotid circulation.[1] Duplex scanning uses a combination of high resolution imaging and Doppler ultra sound that detects wall abnormalities and flow changes. Diagnostic accuracy for assessing the cervical carotid artery is comparable to intravenous digital subtraction angiography,[2] but a major problem is that the interpretation of findings are greatly operator dependent; inter-observer variability in the interpretation is therefore an important drawback of this method.[3] Duplex scanning provides real-time images but hard copies of these images on paper or film can be provided-at a cost- for interpretation by others. We have tested whether clinicians can interpret these images correctly, and therefore indirectly, whether clinicians should be routinely provided with hard copy images of duplex studies.

Duplex images of 37 cervical carotid arteries of varying degrees of stenosis from normal to complete occlusion from 21 patients with TIAs or minor strokes were selected for the study. Four clinicians participated, three of whom were clinical neurologists and one a vascular surgeon. One neurologist and the vascular surgeon had some experience and the other two neurologists had no experience of duplex imaging. Participants were provided with one duplex image per artery and were asked to: 1) Identify the ICA and draw a sketch of it; 2) Say how easy the image was to interpret using a scale of easy, difficult and uninterpretable. 3) Assess the degree of diameter stenosis of the ICA into one of the following categories: normal, 1–24%, 25–49%, 50–74%, 75–99% stenosis or occluded.

These assessments were carried out "blind" to the ultrasonographers report and the other clinicians' assessments.

The findings of the ultrasonographer (A) were taken as the "gold standard" for each artery. Confidence intervals were calculated using the standard method for the confidence interval of a proportion.[4]

The clinician (B) found 22 images easy to interpret and the other 15 images difficult to interpret. Clinician (C) found 21 images easy to interpret, 14 difficult to interpret and two images uninterpretable. Clinician (D) found 14 images easy to interpret, 18 difficult and five uninterpretable. Clinician (E) found only four images easy to interpret, 23 difficult and 10 uninterpretable.

The number of arteries incorrectly identified by each clinician was: Clinician (B)—1 artery (3%, 95% CI 0–8), (C)—8 arteries (22%, 95% CI 9–35), (D)—9 arteries (24%, 95% CI 10–38) and (E)—11 arteries (30%, 95% CI 15–44).

The number of arteries in which the clinician agreed with the ultrasonographers assessment are given in the table. The Kappa statistic to assess inter-observer agreement was not calculated because of the high proportion of "uninterpretable" images for some clinicians.

Inter-observer variation in interpreting duplex images of cervical carotid arteries has been the focus of attention in several studies[3] and the variability between trained ultrasonographers seems to be acceptable. This small study assesses the accuracy of interpretation between untrained clinicians. Clinicians with

Table Agreement with ultrasonographer's assessment of stenosis in 37 arteries

Clinician	No arteries correctly identified	Agree on stenosis n (%, 95% CI)*	Disagree by 1 category of stenosis**	Disagree by 2 categories of stenosis	Uninterpretable
B	36	18 (50, 33–67)	15	2	1
C	29	11 (38, 21–58)	15	2	1
D	28	13 (46, 28–66)	12	1	2
E	26	11 (42, 23–63)	9	1	5

*the proportion of correctly identified arteries in which the clinicians' assessment of stenosis was in the same category as the ultrasonographer and 95% confidence intervals. **categories of stenosis: Normal, 1–24%, 25–49%, 50–74%, 75–99% and occluded.

no experience in DCS had difficulty in identifying the ICA correctly in a significant proportion of cases. Amongst correctly identified arteries there was considerable inter-observer variation in the interpretation of the images and the untrained clinicians made errors in interpreting abnormalities in 50–62% of the arteries even when they identified the arteries correctly. It is likely that similar problems may occur in other departments and hospitals, so until clinicians have a greater experience of DCS, hard copies of images should not be routinely provided. It appears that correct interpretation of duplex images require extra training.[5]

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1 Hankey GJ, Warlow CP. Symptomatic carotid ischaemic events: safest and most cost effective way of selecting patients for angiography before carotid endarterectomy. *BMJ* 1990; **300**:1485-91.
2 Taylor KJW. Clinical application of Carotid Doppler ultrasound In: Taylor KJW, Burns PN, Wells PNT, eds. *Clinical application of Doppler ultrasound.* Raven Press: New York, 120-61.
3 O'Leary DH, Bryan FA, Goodison MW, *et al.* Measurement variability of carotid atherosclerosis: real-time (B-mode) ultrasonography and angiography. *Stroke* 1987;**18**:1011-7.
4 Gardner MJ, Altman DG. Calculating confidence intervals for proportions and their differences. In: Gardner MJ, Altman DG, eds. *Statistics with confidence—confidence intervals and statistical guidelines.* London: BMJ Publications 1989:28-33.
5 Nelson TR, Pretorius DH. The Doppler signal: where does it come from and what does it mean. *AJR* 1988:**151**:439-47.

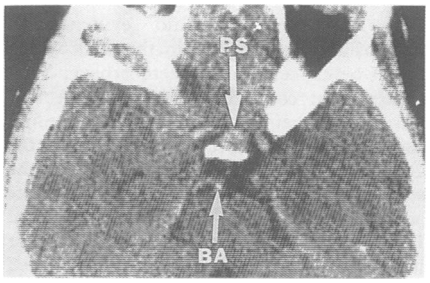

Figure 1 CT findings in case 2 at presentation. BA = basilar artery, PS = pituitary stalk.

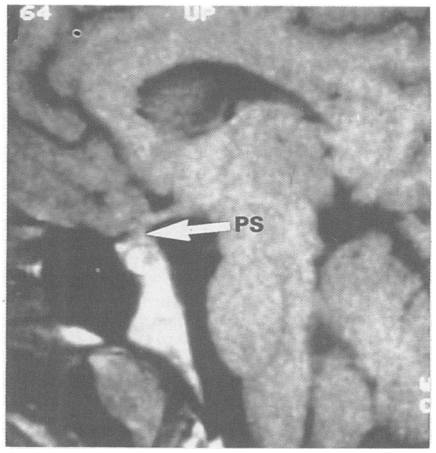

Figure 2 MRI findings in case 2, three months after presentation.

Hyperventilation tetany: effect of carbamazepine

Schaaf and Payne first reported the use of diphenylhydantoin to treat tetany in 1966.[1] We decided to try anticonvulsant therapy with carbamazepine in hyperventilation tetany. Carbamazepine was administered to 211 women and 77 men aged 18–42 (mean 28) in a dose of 600 mg daily for three months. Chvostek's sign was found in 70%, carpopedal spasms and paraesthesiae occurred in 28% and nonspecific features such as headache, fatigue and anxiety in 72%.

Electromyography was performed on all patients after two minutes of hyperventilation and all showed repetitive double, triple and multiple discharges typical of tetany.

Carbamazepine significantly improved many of these features and Chvostek's sign disappeared in 31%.

A general reluctance to use anticonvulsant medication to treat hyperventilation tetany is probably due to the belief that tetany is due to ionic imbalance. Our results have shown that the stabilising effects of carbamazepine on neurons, probably implemented at the level of the brainstem reticular formation which is responsible for the integration of tetanic activity, was the reason for the improvement. The effect on the limbic system was shown by the decrease in anxiety.

Our experience suggests that carbamazepine may be useful in the treatment of hyperventilation tetany.

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1 Schaaf M, Payne Ch. Effect of diphenylhydantoin and phenobarbital on overt and latent tetany. *New Engl J Med* 1966;**22**: 1228–33.

Reversible pituitary stalk enlargement in cranial diabetes insipidus

Infiltrative disease or neoplastic involvement of the pituitary stalk results in the CT appearance of stalk thickening[1] often accompanied clinically by diabetes insipidus. Some patients, however, with cranial diabetes insipidus of no identifiable cause may also exhibit stalk enlargement.[2] The natural history of this radiological abnormality in so-called idiopathic cranial diabetes insipidus has not been previously reported. We report on the follow up of two patients with cranial diabetes insipidus whose CT findings at initial presentation revealed gross stalk enlargement.

CT scan showed gross thickening of the pituitary stalk in a 14 year old boy who presented three months after onset of symptoms. Water deprivation test with parallel assessment of thirst sensation confirmed the diagnosis of diabetes insipidus with concomitant adipsia. Treatment comprised water restriction (2 l/day) and intranasal desmopressin. Three years later a repeat CT scan showed no abnormality of the stalk. Serial CT and MRI over an eight year follow up have excluded a mass lesion.

In a second case a 24 year old female presented with a two week history of polyuria and polydipsia of sudden onset. Water deprivation testing confirmed cranial diabetes insipidus. Anterior pituitary function was intact. CT findings in this patient at initial presentation are shown in fig 1. The normal pituitary stalk (PS) is smaller than or equal to the size of the basilar artery[3] (BA), but as illustrated in fig 2, was considerably enlarged in this patient.

MRI performed after three months of treatment with intranasal desmopressin confirmed that the stalk was still thickened. One year after presentation, however, repeat CT showed that the stalk was then of normal size (fig 3).

Structural involvement of the pituitary stalk in a number of pathological states such as histiocytosis, sarcoidosis, primary and secondary neoplasms may result in diabetes insipidus. Our patients with diabetes insipidus had gross stalk enlargement at presentation but resolution of the abnormality without treatment other than desmopressin in these cases confirms that stalk enlargement may occur in the absence of any progressive infiltrating or neoplastic process.

The MRI findings in case 2 (fig 2) three months after presentation are noteworthy. There was no abnormalities of the hypothalamus. Stalk thickening was still obvious, though less marked than at presentation suggesting a slowly resolving process. The fact that the lesion was still present at this time excludes the possibility of ischaemic infarction of the stalk since the effects of infarction

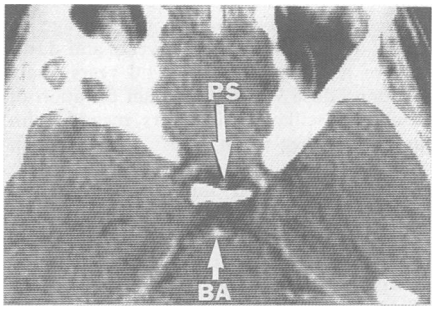

Figure 3 CT findings in case 2, one year after presentation.