Figure CT scan revealing enlargement of the temporal horn of the left ventricle with temporal lobe atrophy on the same side.

The development of the disease in the patient resembles the description of the patient in the article by Scheltens *et al.*¹ Our patient, however, showed a unilateral lesion in the temporal region of the brain as the most striking feature. Thus conforming with Scheltens' case we assume our patient had a unilateral temporal form of Pick's disease. These temporal lobe variants are probably more frequent than had been suspected.

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- 1 Scheltens Ph, Hazenburg GJ, Lindeboom J, Valk J, Wolters EC. A case of progressive aphasia without dementia: "Temporal" Pick's disease? *J Neurol Neurosurg Psychiatry* 1990;53:79-80.

Scheltens *et al* reply:

We thank Dr Colon *et al* for their comment on our article.¹ Their clinical description of the patient indeed resembles the one given by us. We based our clinical diagnosis of a strictly temporal form of Pick's disease on the age of onset (50 years), the relative sparing of visuospatial abilities and memory function and the bilateral temporal atrophy seen on MRI.

In the case of Colon *et al* the age of onset was 65 years with a 12 year duration of slowly progressive aphasia. However, disorientation in time, place and persons was also prominent and together with the slight apraxia and agnosia, a diagnosis of Alzheimer's disease might be just as likely. In that respect it resembles the case of Pogacar and Williams.²

Unfortunately, in the case of Colon *et al* only CT was performed. MRI, with its higher sensitivity might have shown more lesions in the left hemisphere or lesions in the contralateral temporal lobe.

Since our paper was published we have had the opportunity to examine another patient, aged 63 years, with a severe slowly progressive receptive aphasia for six years, without any precipitating factor. An MRI scan showed extensive bilateral temporal damage.

We agree with Dr Colon and colleagues that temporal variants of primary degenerative cerebral disorders are probably more frequent than had been expected.

Improved neuro-imaging techniques have contributed significantly to this knowledge.

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- 1 Scheltens Ph, Hazenberg GJ, Lindeboom J, Valk J, Wolters E Ch. A case of progressive aphasia without dementia: "temporal" Pick's disease? *J Neurol Neurosurg Psychiatry* 1990;53:79-80.
- 2 Pogacar S, Williams RS. Alzheimer's disease presenting as slowly progressive aphasia. *RI Med J* 1984;67:181-5.

Complications of carotid angiography

We found the survey of complications of carotid angiography¹ interesting, but do not feel it illuminates the authors' goal in evaluating the risks of angiography vis à vis carotid surgery. Most of their patients (about 75%) could not be considered for carotid endarterectomy, so complications in the total cohort studied are largely irrelevant.

Many of the procedures are of historical interest only, since the series encompasses the period from 1977-86, during which time the approach, methodology and materials for angiography have undergone radical changes.

The conclusions do not seem to relate to the data. The authors state that angiographic risks will be reduced by selective, aortic arch procedures, yet in their series, only one patient developed a complication following direct carotid puncture, which argues just the opposite. There are no data to substantiate their statement that patients with least complications should be "younger, systemically well and neurologically stable". The mean age of the ten patients with neurological complications was 58.9/year, a little less than the mean age of the whole series.

Surely their data represent simply a personal audit of a whole spectrum of neurological disorders, in patients aged seven to 76 years, and it would be misleading to extrapolate these results to the special subset of patients being screened for carotid surgery. In a recent report of angiographic complications in 1002 consecutive procedures, permanent neurological sequelae were encountered in only 0.3% of cases, and in 0.7% of those undergoing angiography for cerebrovascular disease.²

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- 1 Complications of cerebral angiography for patients with mild carotid territory ischaemia being considered for carotid endarterectomy. *J Neurol Neurosurg Psychiatry* 1990;53:542-8.
- 2 Dion JE, Gates PC, Fox AJ, Barnett HJM, Blom RJ. Clinical events following neuroangiography: a prospective study. *Stroke* 1987;18:997-1004.

Hankey *et al* reply:

Drs Zhu and Norris have raised several points

about our paper to which we would like to reply. It is true that after cerebral angiography in 382 patients, 74% (282) were not considered to have an "operable" lesion at the carotid bifurcation but it was stated that "carotid ultrasound facilities were not available". If patients could have been screened by duplex carotid ultrasound, as we advocate in our conclusion, then perhaps many or even most of those 74% of patients who did not have an "operable" lesion would have been spared the unnecessary risks and costs of angiography. However, only two of the eight patients, who had a post-angiographic stroke, did not have an "operable" lesion (50-99% stenosis of the symptomatic internal carotid artery) and one of those two had an occluded ICA which can be difficult to diagnose with duplex alone. So, if duplex had been available ideally it could have reduced the number of angiograms being performed (by 74%) to 100 but it would have only reduced the absolute number of post-angiographic strokes from eight to seven with a significant increase in the relative risk of angiography from 2.1% (eight strokes out of 382 angiograms) to 7% (seven strokes out of 100 angiograms).

The fact that our series is of "historical interest only" applies to any series of patients. This is a general problem with reporting medical information and generalising the results to future practice. Unlike many other series, however, at least we described what type of investigations were done from which the reader can draw their own conclusion. Besides, 67% of patients were studied with selective carotid angiography, which is still current practice. The only real difference in the management of this cohort of patients from those of today is that they were not screened with duplex carotid ultrasound, as mentioned above.

In our concluding paragraph we stated that "it would seem logical to assume that the complications of cerebral angiography can be reduced if patients are selected carefully for the procedure" and that the selection criteria should include "physiologically younger patients who are systemically well, not uraemic and neurologically stable." This conclusion was prefaced as such to indicate that it was not derived from our data but from what data there are and what we believe is common sense. Apart from the lack of any statistical difference between the ages of the whole cohort and those with neurological complication (as inferred by Drs Zhu and Norris) we were referring to patients who were physiologically younger. We also suggested that "the risk of angiography will be reduced if arch and/or selective cerebral angiography is performed . . . using a transfemoral approach". It is not possible to prove or disprove this claim (which again was not based on our data but on common sense) because there have been no randomised studies comparing the complication rates of cerebral angiography via the transfemoral approach with direct carotid or brachial puncture. The few non-randomised comparisons, for what they are worth, yield no significant difference in permanent neurological complication rates.¹⁻³

The advantages of the transfemoral approach over direct carotid puncture are two fold: 1) it provides greater flexibility by allowing the radiologist to study different arteries without repeated arterial puncture, and 2) the consequences of local complications (such as haematoma, intimal dissection or fragmenta-

tion and embolisation of atherosclerotic plaque) are likely to be less if the femoral artery is involved rather than the common carotid artery.

We agree with Drs Zhu and Norris that our data do represent a personal audit, as does every other such series, but we emphasised that the "results from this study . . . cannot be generalised to all medical centres but only used as an approximate guide. All medical institutions . . . need to monitor their own results." Unlike the series of Dion *et al.*,⁴ quoted by Drs Zhu and Norris, in which 1002 angiographic procedures were performed in patients with a whole spectrum of neurological disorders, our study was confined to patients with symptomatically mild cerebral or ocular ischaemia in the carotid distribution. Out of the 1002 procedures studied by Dion *et al.*, 285 (28.4%) were for "TIA/stroke" and the permanent neurological complication rate for these patients was 0.7% (95% confidence interval 0.1% to 2.5%); not significantly different from the 1.3% (95% CI: 0.4% to 3.0%) permanent neurological complication rate in our study.

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- 1 Huckman MS, Shenk GI, Neems RL, Tinor T. Transfemoral cerebral angiography versus direct percutaneous carotid and brachial arteriography: a comparison of complication rates. *Neuroradiology* 1979;132:93-7.
- 2 Olivecrona H. Complications of cerebral angiography. *Neuroradiology* 1977;14:175-81.
- 3 Hass WK, Fields WS, North RR, Kricheff II, Chase NE, Bauer RB. Joint study of extracranial arterial occlusion. II. arteriography, techniques, sites and complications. *JAMA* 1968;203:961-8.
- 4 Dion JE, Gates PC, Fox AJ, Barnett HJM, Blom RJ. Clinical events following neuroangiography: a prospective study. *Stroke* 1987;18:997-1004.

Diaphragmatic weakness in hereditary motor and sensory neuropathy

We were interested to read a short report on diaphragmatic weakness in hereditary motor and sensory neuropathy. Whilst the case reports were highly suggestive of diaphragm weakness, we were disappointed that the authors relied on clinical and indirect assessments rather than on quantitative measurements of diaphragm and respiratory muscle strength. The techniques for quantifying respiratory muscle function are now well described² and can be used routinely in appropriate respiratory physiology laboratories. The measurements, which include those of maximum expiratory and inspiratory mouth pressures, of oesophageal and transdiaphragmatic pressures during maximal inspiratory manoeuvres such as an inspiratory sniff and during phrenic nerve stimulation, and of phrenic nerve conduction time, can be carried out with minimal discomfort to the patient in less than one hour.

Diaphragm weakness has already been reported in hereditary motor and sensory neuropathy,^{3,4} and we were surprised to find no mention of these reports in the paper by Hardie *et al.*¹ In our paper we reported two cases in which diaphragm dysfunction was proved and quantified using a full range of appropriate tests. In a third report,⁵ a patient

who had died from the condition was found at necropsy to have identical neuropathic changes in the phrenic nerves as in the other peripheral nerves.

It is important to distinguish between diffuse respiratory muscle weakness and isolated diaphragm dysfunction. Isolated diaphragm paralysis has not been shown to cause significant nocturnal hypoventilation or respiratory failure, as long as the patient does not sleep supine, or does not have other significant lung or chest wall disease.⁶ The majority of patients in the paper by Newsom Davis quoted by Hardie⁷ had diffuse neuromuscular disease, which would be expected to affect all the respiratory muscles as well as the diaphragm. It is interesting, however, that in the reported cases where respiratory muscle dysfunction has been quantified, hereditary motor and sensory neuropathy appears to predominantly affect the diaphragm, perhaps because of the length of the phrenic nerves. Only one of the patients described by Hardie *et al.* had developed respiratory failure and she had smoked heavily. Use of discriminating tests of respiratory muscle function would have elucidated whether there was involvement of the other respiratory muscles or co-existing lung disease due to smoking.

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- 1 Hardie R, Harding AE, Hirsch N, Gelder C, Macrae AD, Thomas PK. Diaphragmatic weakness in hereditary motor and sensory neuropathy. *J Neurol Neurosurg Psychiatry* 1990;53:348-50.
- 2 Laroche CM, Moxham J, Green M. Respiratory muscle weakness and fatigue. *QJMed* 1989;71(265):373-97.
- 3 Chan CK, Mohsenin V, Loke J, *et al.* Diaphragmatic dysfunction in siblings with hereditary motor and sensory neuropathy (Charcot-Marie-Tooth disease). *Chest* 1987;91(4):567-70.
- 4 Laroche CM, Carroll N, Moxham J, *et al.* Diaphragm weakness in Charcot-Marie-Tooth disease. *Thorax* 1988;43:478-9.
- 5 Gilchrist D, Chan CK, Deck JHN. Phrenic involvement in Charcot-Marie-Tooth disease. *Chest* 1989;96(5):1197-9.
- 6 Laroche CM, Carroll N, Moxham J, Green M. Clinical significance of severe isolated diaphragm weakness. *Am Rev Respir Dis* 1988;138(4):862-6.
- 7 Newsom Davis J, Goldman M, Loh L, Casson M. Diaphragm function and hypoventilation. *QJMed* 1976;45(177):87-100.

Hardie, *et al* reply:

The primary intention of our paper was to bring the possible development of diaphragmatic weakness in hereditary motor and sensory neuropathy (HMSN) to the attention of neurologists, to whom patients with these disorders are largely referred. We were of course aware of the other case reports on this subject, and indeed quoted the paper of Chan *et al.* In the report of Laroche *et al.* there were no clinical or electrodiagnostic data provided to substantiate the diagnosis of HMSN, and one of the patients had prominent urgency of micturition and defaecation, which would be most unusual. The paper of Gilchrist *et al.* was published after ours was accepted.

We also pointed out that involvement of the diaphragm in HMSN could be related to the length of the phrenic nerve, without proposing that diaphragmatic weakness was isolated. The relative importance of diaphragmatic involvement in our cases was illustrated by the nocturnal hypoventilation demonstrated in case 1, and the respiratory distress induced in the patients when lying supine, a position

assumed by most of us at some time during sleep.

Whilst we agree that quantitative analysis of respiratory muscle weakness is important, there has to be a strong suspicion that these muscles are involved before the patient is referred to an "appropriate respiratory physiology laboratory". Simple bedside tests of respiratory function, as described in our six cases, are useful in this context. Nocturnal oxygen saturation studies are also essential to exclude coexisting obstructive sleep apnoea.

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BOOK REVIEWS

Fits and Faints. By J B P STEPHENSON. (Pp 202 illustrated; Price: £21.00). Oxford: Blackwell Scientific Publications Ltd, 1990. ISBN 0 632 02811 4.

This book is about 'fits' and 'faints' and not primarily about epilepsy. It discusses the many ways in which non-epileptic attacks may sound like epilepsy. It is mainly about paediatric practice although this does not detract from its overall interest. Dr Stephenson emphasises Jeavons data which suggests that 20%-30% of patients are erroneously labelled epileptic. None of us like the term 'known epileptic'; the dangers of a supposed positive family history of epilepsy are also stressed.

Most neurologists must have had doubts about their diagnosis of epilepsy in a few patients during follow-up. This may occur even after a confident initial diagnosis was made based on the patient's and witness's descriptions, cardiovascular and neurological examination, as well as EEG and ECG data. All are aware of the anoxic fit. However, my horizons about anoxic seizures have been widened as a result of reading this book. Dr Stephenson is to be congratulated on pursuing the art of history taking even further. The book is essentially clinical: it includes over 140 case histories which makes it fun for clinicians to decide what they would have diagnosed, although at times I think there may be unnecessary repetition.

Many neurologists will test for carotid hypersensitivity, especially in the elderly; few, I suspect, will use ocular compression with ECG and EEG monitoring in the assessment of anoxic seizures. The author clearly feels this is a useful test and it needs to be assessed further.

This book will be enjoyed by all neurologists who see blackouts!

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