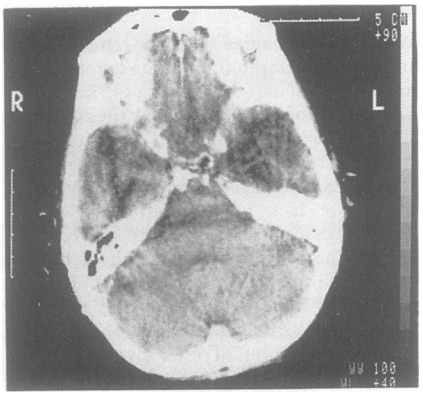

Figure CT scan revealing enlargement of the temporal horn of the left ventricle with temporal lobe atrophy on the same side.

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 angiography[1] 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-