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D Smith et al reply:

We thank Mr Wilden for his interest in this article. Whilst recognising minor problems with some of our definitions, we would like to emphasise that this was a retrospective study which generates a simple hypothesis demanding prospective testing.

We agree that definitions of "early" and "late" surgery have not been agreed upon and that our own definitions, of necessity, are somewhat arbitrary. They do, however, allow the essential differentiation between those patients receiving surgery soon after diagnosis and those in whom surgery is delayed until a change in clinical status occurs. Furthermore, by using an intention to treat analysis, we avoided bias against the effectiveness of surgery as a whole by excluding patients whose surgery was performed because of neurological deterioration.

With regard to the type of surgery—resective surgery refers to procedures which involve debulking of the tumour and non-resective to biopsy or drainage of cysts. As Mr Wilden states, we have not subdivided resective surgery according to whether or not the surgeon considered the resection to be complete or partial. However, this information is difficult to obtain from a retrospective review of case notes and is not likely to be reliable. Furthermore, whilst accepting that this issue may be relevant in a minority of patients, with well circumscribed temporal or frontal lobe tumours, it is well recognised that resections "considered" to be total macroscopically are rarely confirmed on histopathological specimens, and many pragmatic neurosurgeons would concede that total excision of infiltrative gliomas, often involving more than one lobe, is not possible.

Mr Wilden has answered his second query himself. We would, however, like to emphasise that it was never our intention to conduct sub-group analyses, in small numbers of patients, and it is well known that such analyses on retrospective data yield unreliable results that are very difficult to interpret.

His final remarks merit two comments. Firstly, we would like to reiterate that we made every effort not to over-interpret our data and our intention was simply to generate a testable hypotheses and certainly not the development of "definitive management policies". Secondly, we must take issue with Mr Wilden's comments on the need for a national database derived from individual centres inevitably employing different treatment regimens. Data collected in this way would not allow development of satisfactory management policies and the necessary information can only be obtained, as we state clearly in our final paragraph, from a well designed, multi-centre, prospective randomised trial comparing conservative and aggressive management policies.

Extracranial vertebral artery dissection

We would like to make several comments about the recent report by Hinse *et al*[1] on extracranial vertebral artery (VA) dissection:

1) This article is based on 4 personal cases and a review of 53 published cases. However, several published reports, including 2 recent studies[2 3] on a total of 38 cases, are not quoted, which may give the impression that VA dissection is rare. In 1988, Hart[4] estimated that nearly a hundred cases of VA dissections had appeared in the English language literature in the past decade and that between 0·5 and 2·5 cases per year were reported from large referral-based hospitals.

2) We have come to similar conclusions in our own work on the subject[2] regarding symptoms and prognosis of this disorder. The authors, however, do not mention in their discussion that VA dissection may be asymptomatic and possibly discovered when exploring a concomitant carotid artery dissection.[2] They point out that there is a high incidence of bilateral VA dissection but do not mention the possibility of simultaneous occurrence of vertebral and internal carotid artery dissections, which is not infrequent particularly in patients with spontaneous dissection.[2 3] The frequency of multivessel dissection implies that four-vessel exploration (by angiography or other method) should always be attempted if a VA dissection is demonstrated.

3) The diagnosis of dissection relies classically on angiography, which reveals irregular stenosis, pseudoaneurysm, occlusion, or double-lumen. Of these features, the only pathognomonic one (but also the rarest) is the double lumen. Occlusion which can be due to thrombosis of any cause is the least specific. We think that the diagnosis of probable occlusive form of VA dissection can only be made when there is angiographic evidence of dissection in the other VA or in the internal carotid artery. In this respect, the diagnosis of dissection in patients 2 and 3 of Hise *et al*[1] who had VA occlusion should only be regarded as possible.

4) Normalisation or improvement of angiographic abnormalities is frequent in this condition and is an excellent argument in favour of the diagnosis. In one of our cases,[2] marked improvement was observed as early as 7 days after the first angiogram, which stresses the point that the first angiography should be performed as early as possible so that the diagnosis should not be overlooked. We also concluded that control angiography could be performed around the third month, a time by which most dissected vertebral arteries had returned to normal.

5) In our opinion, ultrasound examination

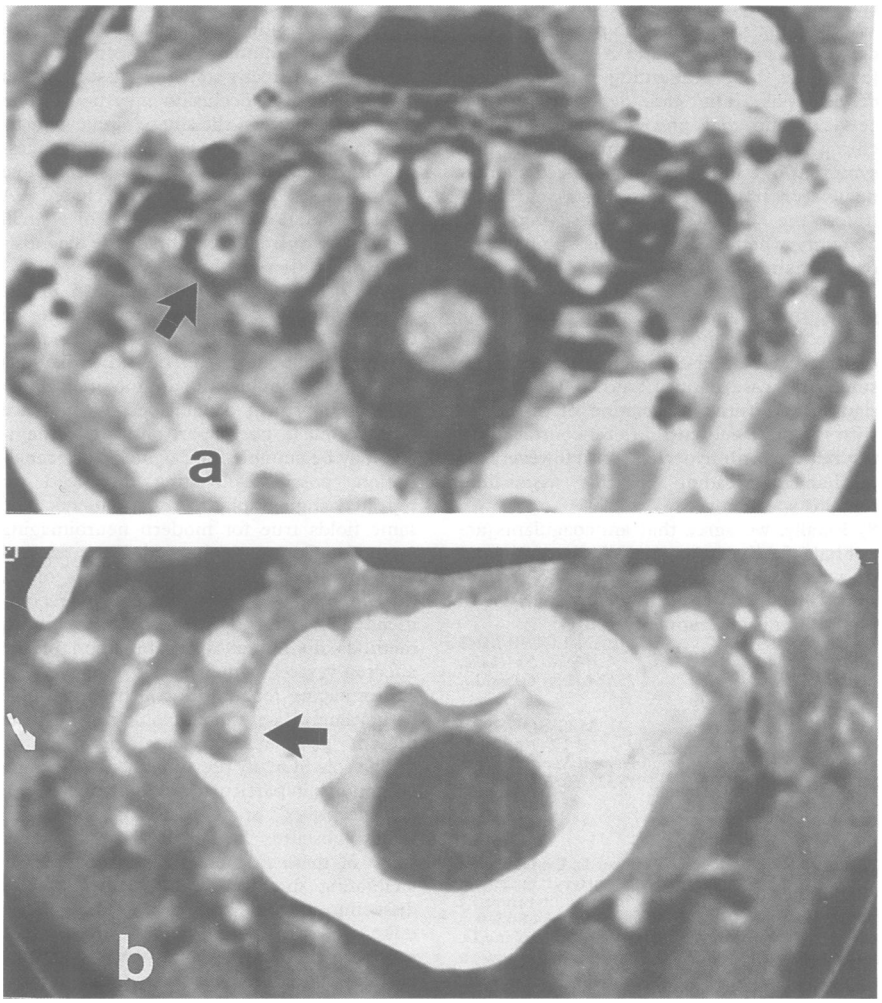

Figure Dissection of the third segment of the right vertebral artery.
a) T1-weighted axial MRI (SE 500/25): Central hyposignal (flow void in the residual lumen of the vertebral artery) surrounded by hypersignal (parietal haematoma).
b) Thin section contrast-enhanced dynamic CT scan: Eccentric hyperdensity (injected residual lumen) surrounded by isodense haematoma and peripheral ring enhancement.