can not only show "some abnormality that encourages angiographic examination" but can also diagnose dissections involving the pretransverse, C6–C5 and C5–C4 intertransverse segments of the VA.[5] The diagnosis is based on the association of a localised increase in arterial diameter with haemodynamic signs of stenosis or occlusion and/or decreased pulsatility and intravascular echoes at the same level. Furthermore, ultrasonic examination is an excellent tool for the follow up of dissection.

6) Among other diagnostic procedures, the authors did not mention thin-section contrast-enhanced dynamic CT scan and MRI. By virtue of its sensitivity to both blood flow and thrombus formation, its multiplanar imaging capability, and its noninvasiveness, MRI (and soon MR angiography) is becoming the imaging modality of choice for the evaluation of suspected carotid or vertebral dissection (fig). At present, however, MRI does not assist in distinguishing between intraluminal and intramural thrombus and therefore does not allow the diagnosis of occlusive forms of vertebral dissection.

7) The relation of trauma to dissection is a complex issue. Hinse *et al*[1] considered their patient 4 as an example of traumatic (chiropractic manipulation) dissection. We recently reported[6] the case of a woman with a 3 week history of cervical pain who developed iscaemia in the basilar artery territory following cervical manipulation. Necropsy revealed 2 VA dissections, a recent one probably due to cervical manipulation and a second one, a few weeks old, accounting for the initial cervical pain. This case demonstrates that cervical pain that precedes and motivates chiropractic manipulation may be the first symptom of a hitherto unrecognised spontaneous (or traumatic) dissection and illustrates the difficulty in classifying with certainty whether dissection is spontaneous or traumatic.

Apart from trauma and fibromuscular dysplasia, other conditions implicated as risk factors for dissection include migraine, oral contraceptives, and chronic high blood pressure. In a case control study,[7] we found a significant positive association of dissection with migraine and current oral contraceptive use but not with hypertension. However, the mechanisms leading to this association remains speculative.

8) Finally, we agree that anticoagulants are not harmful in extracranial VA dissection and may even be of benefit although no conclusion can be drawn from the comparison of nonrandomised treatment groups.

JEAN-LOUIS MAS
Hôpital Ste-Anne,
75674 Paris Cedex 14,
France

M G BOUSSER
P J TOUBOUL
Hôpital Saint-Antoine,
75571 Paris Cedex 12,
France

1 Hinse P, Thie A, Lachenmayer L. Dissection of the extracranial vertebral artery: report of four cases and review of the literature. *J Neurol Neurosurg Psychiatry* 1991;**54**:863–9.
2 Mas JL, Bousser MG, Hasboun D, Laplane D. Extracranial vertebral artery dissections: a review of 13 cases. *Stroke* 1987;**18**:1037–47.
3 Mokri B, Houser OW, Sandok BA, Piepgras DG. Spontaneous dissections of the vertebral arteries. *Neurology* 1988;**38**:880–5.
4 Hart RG. Vertebral artery dissection. *Neurology* 1988;**38**:987–9.
5 Touboul PJ, Mas JL, Bousser MG, Laplane D. Duplex scanning in extracranial vertebral artery dissection. *Stroke* 1987;**18**:116–21.
6 Mas JL, Henin D, Bousser MG, Chain F, Hauw JJ. Dissecting aneurysm of the vertebral artery and cervical manipulation: a case report with autopsy. *Neurology* 1989;**39**:512–5.
7 D'Anglejan-Chatillon J, Ribeiro V, Mas JL, Youl BD, Bousser MG. Migraine—A risk factor for dissections of cervical arteries. *Headache* 1989;**29**:560–1.

Hinse and Thie reply:

We thank Dr Mas and colleagues for their interest in our recent paper,[1] and we appreciate the opportunity to comment on a few of the issues raised by them.

Our paper did not deal with the incidence of vertebral artery (VA) dissection which remains unknown. Better diagnosis and systematic study will hopefully shed more light on incidence of this condition in the future. In our analysis, we have included only case reports providing sufficient detail of the individual patient, but not summarised series, thus the results of Mokri *et al*[2] were not considered. We apologise for not including the well documented patients by Mas *et al*.[3]

The question of internal carotid artery (ICA) dissection was not the subject of our paper. We agree that angiographic visualisation of all four brain-supplying vessels should be attempted in acute VA dissection in order not to miss concomitant asymptomatic ICA dissection. This point is of particular importance in spontaneous VA dissection: in 5 of 29 reviewed patients concomitant ICA dissection was documented, but not in 28 patients with traumatic VA dissection.

Mas *et al* correctly state that dissection as a cause of arterial occlusion may be hard to diagnose. However, the angiographic appearance of tapering occlusion is highly suggestive of dissection.[4] In our patients, complete recanalisation of a formerly occluded VA (cases 2 and 3) and visualisation of a small pseudoaneurysm (case 2) made the diagnosis of VA dissection highly probable.

The value of ultrasound method in the diagnosis of VA dissection remains to be determined. Our own experience[1] and the work of Touboul *et al*[5] on three patients examined by duplex scan are too preliminary to allow any firm conclusions. As we have pointed out in our paper, ultrasound methods may be suitable for diagnosis of recanalisation, possibly obviating the need for repeated angiography in some patients. The same holds true for modern neuroimaging methods. Contrast-enhanced CT scan and MRI may corroborate the diagnosis of VA dissection, and are also increasingly recommended for follow up studies. However, both methods do not allow examination of the affected vessels in their entire length. These neuroimaging techniques will have to prove their value in a systematic study against the present "gold standard" (angiography).

The role of many presumably predisposing conditions, in particular migraine, use of oral contraceptives or hypertension, remains totally obscure. It is speculative whether some of these factors might act by merely facilitating the occurrence of stroke after dissection, but not dissection itself. It is also unknown why minor trauma may induce cervical dissections in some patients at any particular time, but why recurrences in these patients are rare.

P HINSE
A THIE
Department of Neurology,
University of Hamburg,
Martinistr 52,
2000 Hamburg 20, Germany

1 Hinse P, Thie A, Lachenmayer L. Dissection of the extracranial vertebral artery: report of four cases and review of the literature. *J Neurol Neurosurg Psychiatry* 1991;**54**:863–9.
2 Mokri B, Houser OW, Sandok BA, Piepgras DG. Spontaneous dissections of the vertebral arteries. *Neurology* 1988;**38**:880–5.
3 Mas JL, Bousser MG, Hasboun D, Laplane D. Extracranial vertebral artery dissections: a review of 13 cases. *Stroke* 1987;**18**:1037–47.
4 Mokri B. Traumatic and spontaneous extracranial internal carotid artery dissections. *J Neurol* 1990;**237**:356–61.
5 Touboul PJ, Mas JL, Bousser MG, Laplane D. Duplex scanning in extracranial vertebral artery dissection. *Stroke* 1987;**18**:116–21.

Late onset globoid cell leukodystrophy

I read with great interest the paper by Grewal *et al*.[1] I would like to add a few comments.

First, the authors suggest that their patient's late onset (at age 14) distinguishes his disease from globoid cell leukodystrophy (GLD) distinct from the infantile and late infantile onset types. This may be true for the first type, but the latter can occur within one family together with a later onset type.[2]

Second, it would be interesting to know whether the white matter hyperintensities on the MRI were diffuse or rather restricted to the occipito-parietal white matter, as described in other late onset GLD.[2 3] If so, this posterior white matter involvement on MRI would seem to be very useful to distinguish GLD from other cerebral white matter diseases.

PETER VERDRU
Neurologie,
Universitaire Ziekenhuizen,
Herestraat 49,
3000 Leuven, Belgium

1 Grewal RP, Petronas N, Barton NW. Late onset globoid cell leukodystrophy. *J Neurol Neurosurg Psychiatry* 1991;**54**:1011–2.
2 Verdru P, Lammens M, Dom R, Van Elsen A, Carton H. Globoid cell leukodystrophy: a family with both late-infantile and adult type. *Neurol* 1991;**41**:1382–4.
3 Shapiro EG, Lockman LA, Krivit W. Correlation of Magnetic Resonance Imaging and Neuropsychological Abnormalities in Late-onset Leukodystrophies. *Ann Neurol* 1991; **30**:466–7.

Aseptic meningitis associated with high dose intravenous immunoglobulin therapy

We read with great interest the report by Watson *et al*[1] and we would like to draw the authors attention to a similar case we published last year.[2]

Our patient was a seven year old boy with idiopathic thrombocytopenic purpura who had well-documented episodes of acute aseptic meningitis on two occasions after the second intravenous dose of immune globulin. On these two occasions, the patient developed aseptic meningitis on day three; quite identical to the two patients reported by Watson, whereas Kato's patient developed the aseptic meningitis two days after a five day course of intravenous immune globulin therapy.[3]

In our patient the immune globulin preparation used was Sandoglobulin IV (Sandoz), which is a formulation prepared by cold ethanol fractionation. It was given at a dose of 0·4 g per kilogram of body weight infused over a 11 hour period.