can also not only show "some abnormality that encourages angiographic examination" but can also diagnose dissections involving the transverse, C6-C7 and C5-C4 intertransverse segments of the VA. 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 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 'digital' 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. Hinsé et al. considered their patients an example of traumatic (chiro-

practic) manipulation) dissection. We recently repeated this case of a woman with a 3 week history of cervical pain who developed sciac-

calgia and basilar headache following cervical manipulation. Neurocopy revealed two 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 unrecognized spontane-

ous (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,1 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 nonrandomized treatment groups.

Hinsé and Thié reply: We thank the authors 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 sys-
tematic 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 visual-

isation 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 reports of acute 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 ap-
ppearance of tapering occlusion is highly suggest-
tive of dissection. 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 experience1 and the work of Touboul et al.4 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 recom-
mended 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.

Late onset globul 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 globul cell leukodystrophy (GLD) distinct from the infantile and late infantile type. 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.3 If so, this posterior white matter involvement on MRI would seem to be very useful to distinguish GLD from other cerebral white matter dis-

cases.

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Aseptic meningitis associated with high dose intravenous immunoglobulin therapy

We read with great interest the report by Watson et al.4 and would like to draw the authors attention to a similar case we published last year.5

Our patient was a seven year old boy with idiopathic thrombocytopenic purpura who had well controlled 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 patient reported by Watson, whereas Kato's patient developed the aseptic meningitis two days after a five day course of intravenous immune globulin therapy.6

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 10 mg/kg of body weight infused over a 1 hour period.

References

3. Shapiro EG, Lockman LA, Krivit W. Correla-
From these three reports, we conclude: 1) Intravenous immune globulin can induce aseptic meningitis in children as well as in adults: the strong temporal association (on day three in our patient and in Watson’s patients) and the repetitive occurrence in the same patient (on two occasions in both our patient and in our patient) are both strong arguments in favour of this. It should no longer be a hypothesis.

2) Several preparations of intravenous immune globulin can induce aseptic meningitis. Up till now it has been reported with four different preparations: with immunoglobulin prepared with polyethylene glycol and with a sulphonated preparation,\(^1\) with a formulation prepared by cold ethanol fractionation (Sandoglobulin)\(^2\) and with a low PH formulation prepared with ethylene glycol (Intragam).\(^3\)

As with many other iatrogenic diseases, the aseptic meningitis in itself is rather benign and resolves rapidly with cessation of the therapy. But not recognising this complication as such might result in potentially dangerous and/or unnecessary examinations (lumbar puncture in a thrombocytopenic patient;\(^4\) CT scan)

The possible occurrence of aseptic meningitis following immune globulin infusion should thus be known by all physicians and should be monitored by the pharmaceutical firms.

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The brain in schizophrenia

The excellent editorial by Ron and Harvey\(^5\) notes that “to have forgotten that schizophrenia is a brain disease will go down as one of the great aberrations of twentieth century medicine”.\(^5\)

We have therefore like to report on our experiences on the side effects of IgV.

In \(54\) patients 0.4 g/kg of IgV (Gammagard\(^6\), Hyland Division, Baxter) was infused during five consecutive days, a total of \(370\) infusions. These infusions were all performed during the acute phase of the Guillain-Barré syndrome in patients of all ages. During the infusion period patients were scored every day and other questions and complications were included in the datasheets. A treatment form was also filled out, and this included questions about any unusual event during treatment.

Five time-related events occurred during these 370 infusions; blood pressure dropped but did not need any treatment (2), dyspnoea treated with a diuretic (1), temperature increase treated with clemastine (1) and transient macroscopic haematuria (1). The treatment course was not interrupted in any of the cases. The last event may have been coincidental, but the others may have been caused by the IgV infusions. They were, however, mild and transient. Aseptic meningitis was not observed in any of the patients. We conclude that the incidence of side effects of high dose IgVs is low and that IgV may be safely applied in neurological patients.

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With great interest we read the article from Spampinato et al\(^6\) and we would like to comment on some aspects of the article.

We performed single photon emission computed tomography (SPECT) using \(^99\)Tc-HM-PAO as a tracer in 51 clinically demented and 6 non-demented patients with Parkinson’s disease (PD). HM-PAO uptake was measured in the frontal, parietal and temporoparietal cortex and was expressed as cortical/ocarital ratio. None of our patients had a reduction of HM-PAO uptake of more than 20%, which seems to be in concordance with Spampinato et al.\(^6\) In contrast to Spampinato et al, however, we found no difference between the demented and non-demented PD patients. We investigated whether there was a relation between SPECT-scan data and neuropsychological tests in our group as a whole (n = 11). Using the Spearman rank correlation test, we found no significant correlations between neuro-psychological performance and SPECT data on any test.

One of the main problems in our research was the estimation of dementia in PD. Of the 5 patients who fulfilled the criteria for dementia as described in the DSM-III-R, all lacked typical cortical features. All of our patients were able to undergo the neuro-psychological tests. In the study by Spampinato et al**, diagnosis of dementia was based on neurological assessment, according to which the PD patients were divided into two groups of 15 patients. However, five of their patients could not be tested, probably due to severe dementia. It was not stated by the investigators whether their demented PD patients were free from cortical features.

In our opinion, one of the possible explanations for the differences in results is that our patients had no cortical dysfunction and therefore no Alzheimer-like pathology. Further elucidation of dementia as seen in PD seems mandatory.

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