

ascertain the aetiology. It is true that serology may miss some cases of cysticercosis but our results are not grossly dissimilar. They could demonstrate definite cysticercosis in seven out of 15 cases (46%) while in our series serology was positive in 31%. The number in both studies is relatively small and much should not be made of difference in percentages. In our opinion persistent lesions should not be compared with disappearing lesions as the two may be entirely different.

Our hypothesis that contrast enhancement is due to a recent seizure is based on well documented evidence in the literature that seizures lead to transient breakdown in blood-brain barrier. This is supported by observation in at least five patients in whom the lesion disappeared, reappearing after a flurry of seizures to disappear again. We are unable to accept the argument that this could all be due to technical factors as suggested by Drs Rajshankar and Abraham. Since lesions due to other causes are known to show similar CT morphology,³ it is not wise to state that cysticercosis is the only underlying cause of "disappearing lesions". Larger studies using different strategies are required to answer the question and one such study has been initiated in our department.

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The lacunar hypothesis

The paper by Drs Anzalone and Landi¹ is an interesting contribution to the debate about the validity of the "lacunar hypothesis" that links a small number of clinical syndromes to occlusion of a single perforating artery by specific vasculopathies.² The significant number of non-ischaemic lesions in their series certainly seems to justify the early scanning of these patients, but in the context of cerebrovascular disease it is important that the result is not interpreted as a failure of the lacunar hypothesis.

Although the authors do not state the number of CT scans which showed an appropriately sited small deep infarct, our experience using a similar scanner suggests that it is unlikely to have been more than about 50%.³ This lack of a definite clinico-radiological correlation in a significant number of cases, combined with isolated case reports of patients with lacunar syndromes and more extensive (non-lacunar) areas of infarction, has meant that concern is still expressed about how often non-lacunar infarction may present in this way. This is despite recent reports which have shown that the majority of patients who present with a lacunar syndrome and a negative CT scan have an appropriate small deep infarct on MRI.⁴

It is possible that many of the patients who are reported to have had a lacunar syndrome from extensive areas of infarction were examined during the recovery phase and that more extensive clinical deficits, not compatible with a lacunar syndrome, would have been found if the examinations had been performed in the acute phase. The fact that no cases of non-lacunar cerebral infarction were seen on CT scanning in this large prospective study where patients were examined very soon after the onset of their stroke lends support to this view and helps to put the previous case reports into perspective.

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Drs Landi and Anzalone reply:

We were interested to read the comments by Dr Bamford on our paper, which stressed the importance of early CT scanning in patients with very recent onset of a lacunar syndrome.

We agree with Dr Bamford that, in the context of ischaemic cerebrovascular disease, our results should not be interpreted as evidence against the "lacunar hypothesis". Appropriately sited lacunar infarcts were observed in 37 (42.0%) of our ischaemic subjects, and no patient had a larger (> 15 mm diameter) or cortical infarct at CT scan.

However, since non-lacunar infarcts have been reported in a small percentage of patients with clinical evidence of a lacunar syndrome,¹ we attribute their absence in our series to chance. Alternatively, as suggested by Dr Bamford, examination in the acute phase of stroke may have allowed us to exclude those patients with initially more extensive clinical deficits who would later improve and present the clinical features of a lacunar syndrome during the recovery phase. This possibility underscores the importance of early neurological assessment in patients considered for inclusion in studies of lacunar infarct.

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Possible benign intracranial hypertension and essential thrombocythaemia

We read with interest the paper by Esack *et al*¹ on benign intracranial hypertension and essential thrombocythaemia.

The syndrome of isolated intracranial hypertension with normal cerebrospinal fluid and CT scan presented by their patient does indeed suggest benign intracranial hypertension. Such a diagnosis, however, seems

difficult to admit in a patient with essential thrombocythaemia, which has been reported as a possible aetiology of cerebral venous thrombosis,² and who presented with a popliteal vein thrombosis during the course of his neurological disease.

The authors rightly envisaged sinus thrombosis but ruled it out on a single digitalised intravenous angiography. We think that this investigation alone is not sufficient to exclude this diagnosis in their patient. The timing of the angiography in cerebral venous thrombosis is crucial since magnetic resonance imaging (MRI) studies have shown the possibility of rapid repermeation of the vessel.³ The sinus blockage is sometimes incomplete and a greater volume of contrast material may be necessary to evaluate the venous sinuses better.⁴ Collateral circulation in the sinus wall may simulate the normal opacification of the sinus by contrast material.⁴

Though heparin has a proven efficacy in cerebral venous thrombosis⁵ the lack of improvement during anticoagulant treatment in their patient does not rule out this possibility. As for the dramatic improvement one week after starting hydroxyurea, it is compatible with both benign intracranial hypertension and dural sinus thrombosis.

The hypothesis of an intermittent sinus blockage may well be right, and essential thrombocythaemia the cause of intracranial hypertension in their patient. However, dural sinus thrombosis has not been fully excluded. It should be looked for with appropriate techniques (four vessel arteriography or MRI) in patients with essential thrombocythaemia presenting symptoms of intracranial hypertension, be it isolated or associated with epilepsy or focal deficit.

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Plasma serotonin

Recently Anthony and Lance¹ published their interesting results on serotonin in patients with chronic tension headache. However, the title of their paper contains a serious error which is also repeated in the text. From the methods section it can be inferred that the authors used whole blood for their study. They even state that "Plasma serotonin was expressed as ng/10⁹ platelets, since they contain about 98% of serotonin in blood." This means that in this study not plasma, but whole blood serotonin was studied and reported, as is also apparent from the results. Anderson *et al*² recognise three compartments of whole blood serotonin, plasma,