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 Rajshekhar 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 Landi[1] 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 thrombosis[5] 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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1 Esack A, Thompson G, Burmester H. Benign intracranial hypertension and essential thrombocythaemia. *J Neurol Neurosurg Psychiatry* 1989;**52**:914–22.
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Plasma serotonin

Recently Anthony and Lance[1] 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/10^9 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,

platelet and cellular. Ortiz *et al*[3] recently confirmed the existence of two distinct pools of serotonin in human blood, plasma and platelet. There is now a general agreement that more than 99% of the serotonin present in blood is contained within the thrombocytes and that the real plasma serotonin content in normal individuals is less than 1% of that in whole blood.

Anderson *et al* rightly emphasise that the plasma fraction is by definition platelet- and cell-free. Apparently it may be concluded that it is incorrect or even impossible to express "plasma" serotonin per platelet! This is not merely a problem of semantics. We must assume that the separate pools of serotonin in blood have different functions and are influenced by different mechanisms. It is correct to measure serotonin in whole blood and regard the result as a measure for platelet serotonin.[2] However, using "plasma" serotonin for total blood or platelet serotonin is misleading and may give reason to serious misinterpretations.

The title and the results of the article by Anthony and Lance should therefore be changed accordingly. This would, of course, not diminish the significance of their results.

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1 Anthony M, Lance WJ. Plasma serotonin in patients with chronic tension headaches. *J Neurol Neurosurg Psychiatry* 1989;52:182–4.
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BOOK REVIEWS

A Task Force Report of the American Psychiatric Assoc. Treatments of Psychiatric Disorders. (Vols 1–4; Pp 3068. Price: £195.00 H/b.) Cambridge University Press. ISBN 0-89042-201-X.

This is truly a mammoth compendium; running to over 3000 pages, packaged in three volumes plus an index. Its very size prompts the question: "Have such large scale undertakings, like the mammoth, outlived their usefulness?"

According to the compilers of these volumes, they represent: "a description of clinically useful clinical approaches for the treatment of mental disorders . . . reflecting a combination of cumulative scientific knowledge." But this up-beat claim is followed by a number of caveats: Firstly, it is not for the novice: "Proper use of this document requires specialised training." Nor should it be viewed as a simple cook-book of therapeutic recipes into which the uninitiated can delve: "Sound use requires a clinician's judgement based on a knowledge of a patient." Nor does it (perish the thought) "Represent the Official Policy of the American Psychiatric Association". Finally, "This Report is not intended to be construed as or to serve as a standard for psychiatric care." (A statement presumably included to exorcise the spectre of any possible legal confrontation.)

What is it then? It is a compilation of 263 chapters covering a diverse range of clinical topics ranging alphabetically from abortion to zetgeibers, and developmentally from infantile autism to senile dementia. The seemingly interminable list of names of over 400 contributors reminded me of the regimental rolls of honour seen on war-memorials.

In each of the sections the scope of the chapters ranges more widely than the general title of this undertaking would suggest. As Klerman, in his preface to the section on affective disorders, points out, this work is not intended to be a manual (it could hardly be so—in the literal sense at least—weighing, as it does, over 6 kg). Nor is it a series of individual idiosyncratic views on management. Each section has been carefully welded together under a distinguished chairperson to form the corporate view of a panel of experienced psychiatrists. Not only is this publication comprehensive, it is as up-to-date as one could expect. Of course, there are blemishes. One wonders, for example, about the wisdom of recommending the combination of an MAOI with a tricyclic antidepressant with only a caution against using imipramine in this context. I also question the statement that secondary amines (such as desipramine) are better for patients with seizure disorder than the parent compound. Similarly the table listing the preferred antidepressants for specific medical disorders which might co-exist with depression is more categorical than present evidence allows; as for example in stating that the newer antidepressants are better in secondary depression resulting from organic brain syndrome. Such tables are reminiscent of the cook-book approach which the work in general, successfully avoids.

Finally, I was pleased to see that the editors have preferred the user-friendly Harvard System of references with the authors' names given in the text and the references listed in alphabetical order, to the numerical citations of the Vancouver System.

All psychiatric libraries should possess this major work. I am sure it will be consulted frequently—until, that is, it becomes out of date—like the mammoth.

TREVOR SILVERSTONE

Recent Achievements in Restorative Neurology Vol. 3: **Altered Sensation and Pain.** Edited by M R DIMITRIJEVIC, P D WALL, U LINDBLOM. (Pp 212; Price: £82.50/US $132.00/DM 237.0.) Basel: Karger, 1990. ISBN 3-8055-5036-7.

Many conference and workshop proceedings are simply not worth publishing. Emanating from a workshop held in Houston in November 1988, this book, however, proves that an important and interesting subject, authoritative contributors, well-written articles and skilful editing can make a first-rate book.

The title of the monograph does not give the reader a very clear idea of what it contains, and it is necessary to peruse the contents carefully. The book is divided into three sections: clinical aspects of pain and altered sensation, procedures for control of pain and altered sensation, and underlying mechanisms of pain and altered sensation. Every reader will of course pick out different chapters which are of particular interest, and the range of subjects dealt with is extensive. Of contributions which the reviewer found of particular interest, Berić's chapter on altered sensation and pain after spinal cord injury points out, amongst other things, the slow evolution of pain syndromes that can occur after cord injury. Sindou and Jeanmonod give a masterly review of surgical procedures that can be carried out for relief of pain.

Particularly illuminating is their report of the benefit of microsurgical dorsal root entry zone lesions in patients with cancer pain, and post-mortem findings in four patients. A frank view of the limitation of neurosurgery for treatment of chronic pain comes from Siegfried in a chapter entitled "Neurosurgical Procedures Abandoned in the Management of Pain", with statements such as ". . . there is no neurosurgery for pain without neurological side effects . . .", and . . . "the use of destructive lesions in the management of chronic pain as was done earlier is no more considered today, except in some very well defined and rare conditions . . .".

Ochoa's superlative chapter, "Neuropathic Pains, from Within: Personal Experiences, Experiments, and Reflections on Mythology", has perhaps a grandiose title, but is of considerable interest, at his own expense. One can only sympathise with the author who in addition to dental pain, has suffered pain from iatrogenic nerve injury, referred pain from ulnar nerve entrapment, root pain leading to two laminectomies, and a plantar neuroma requiring surgery. These experiences, however, have enabled him to study various pains at first hand. He also summarises his views on causalgia and reflex sympathetic dystrophy which elsewhere might be attributed to sympathetic nervous system involvement; he argues that this is a myth; abnormal warmth in "causalgic" states is due to antidromc vasodilatation, and cold is due to somatosympathetic reflex vasoconstriction.

Another unusual chapter is by P D Wall who recounts the divergent views of Brown-Séquard and Mott. The fascinating controversy concerned whether there was increased ipsilateral and decreased contralateral sensation, or vice versa, following hemisection of the spinal cord. It took Denny-Brown to settle the argument: that contralateral decrease in sensation was only observed with partial lesions, and by converting a partial to a complete hemisection, a reversal of signs occurred. This historical and neglected controversy is used as a start for a further theoretical consideration of how morphological and functional restoration of function of a damaged nervous system could occur.

Other interesting chapters include the contribution by Yaksh, who reports that intrathecal strychnine in rats induces allodynia in response to tactile but not thermal stimuli; a cautious report by Coggeshall in which he reviews evidence for small fibre primary afferent input to the dorsal column nuclei; and a contribution from Sir John Eccles on how pain is generated in the neocortex, in which the psyche, the self, the soul and the liaison brain are included in an information flow diagram for brain-mind interaction.

This book can be highly recommended to all those interested in pain and sensory mechanisms. It is well produced, up-to-date, and apart from one chapter for which references may be obtained from the authors, is well referenced.

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