Visual loss in cysticercosis: Analysis of 23 patients

To the Editor: I read with great interest the article by Chang and Keane¹ about visual loss in patients with neurocysticercosis. I do not agree with the authors' explanations given for retrochiasmal causes of vision loss. Patients 18, 19, 22, and 23 were reported to have parenchymal cysts. The authors themselves mentioned that viable cysticercal cysts are not likely to produce focal deficits until the cyst is inflamed and associated with perilesional edema, so I presume that Patients 19, 22, and 23 may have a ring or disk enhancing CT/MRI lesions (no photograph is available in the article). The authors also speculated that these lesions were inflamed cysticercal cysts. In fact, ring or disk enhancing CT lesions are not specific for neurocysticercosis only—several other infective diseases such as CNS tuberculoma, fungal infections, cerebral abscesses, and other parasitic infections like toxoplasmosis can produce similar images, and frequently it is difficult to differentiate between them.² Because all patients were immigrants from Mexico and Central America, other infective possibilities are also equally likely. Patient 20 had an infarct of the right occipital lobe and the authors think that infarction is because of occlusion of the right internal carotid artery due to a cysticercal cyst in the right sylvian fissure. The infarction of the occipital region is inexplicable based on occlusion of the internal carotid artery. Patient 21 had symmetrical infarctions of both occipital regions. The authors think that bilateral posterior cerebral artery occlusions were a result of transtentorial herniations due to obstructive hydrocephalus. This is not apparent from the CT picture (see the center figure in the original article¹) that the patient had definite hydro-

cephalus or raised intracranial pressure; instead, it seems that there is some amount of cerebral atrophy with ex vacuo dilatation of ventricles. The CT picture is consistent with occlusion of top of basilar artery (top of basilar syndrome) presenting with cortical blindness. This study included four patients with suprasellar pathologies. In only two patients (Patients 14 and 15) was histopathologic confirmation available. There can be several diseases producing similar cystic lesions in suprasellar regions, such as epidermoid cysts, subarachnoid cysts, and craniopharyngioma. It is not possible to differentiate these diseases solely based on imaging and clinical features. Anticysticercal therapy was used in none of the patients. It is very well documented that albendazole is effective for all forms of neurocysticercosis, including subarachnoid³ and intraventricular forms. Even partial resolution of these cystic lesions after albendazole therapy would have been a major point in favor of neurocysticercosis.⁴

Ravindra Kumar Garg, MD, DM, Lucknow, India

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Transdermal nicotine in PD: A randomized, double-blind, placebo-controlled study

To the Editor: The relation between PD (currently the second most frequent neurodegenerative disease in the elderly) and smoking is controversial.

Vieregge et al.¹ studied the effectiveness of transdermal nicotine patches as an add-on treatment for symptoms of PD. The authors found no significant drug effects after their double-blind, placebo-controlled trial. Similar findings were previously reported.²

It was also reported in a narrative review of observational studies that there was an inverse association between PD and smoking.³

I think that this protective effect should be excluded, or at least not generalized, on seeing the results of experimental and recent observational studies.

First, many recent case-control studies did not confirm the inverse association. The results of the pooling of five European population-based, case-control studies found no overall association between cigarette smoking and PD.⁴ The authors re-analyzed their results according to age of patients, and ever smoking was protective only in the lowest-quartile age groups.⁴

Second, the standardized mortality rate (SMR) of the last follow-up results on male British doctors argues against the possible inverse association. The 40-year observations reported annual mortality in ever smokers of 16 per 100,000; meanwhile, in life-long nonsmokers annual mortality was reported as 20 per 100,000.⁵ The calculated SMR is of 0.80 (95% CI = 0.41 to 1.54).

Finally, all prospective studies reported were restricted to men. The authors considered risk estimates of Hammond and Hirayama studies for both sexes.³ Both studies followed up cohorts of both sexes but reported risk estimates for men only. Hirayama attributed his stratified analysis to the low frequency of smoking in women of his Japanese cohort. Another report (Wolf et al., 1991) based on Framingham study results, one of the most respectable, well-designed cohort studies, could not identify such an inverse association, except after stratifying their results by sex.

Again, only men showed the protective effect of smoking against PD ($p < 0.05$). It is suggested that the apparent protective effect of smoking might be misleading, as smoking is less frequent in women, yet the incidence of PD is the same.

These inconsistent findings may be due to an etiologic heterogeneity and raise concerns about the conclusion previously settled by many observational studies.

Mohamad Farouk Allam, Cordoba, Spain

Reply from the Authors: There are no new data in Dr. Allam's comments on epidemiologic findings of smoking in PD. All we know from many studies is that patients with PD, when asked about prior smoking habits, do answer that they were less likely to smoke cigarettes than the respective controls. Whether prospective studies not designed for this particular question are at all suitable to clarify the association is debatable. Our intention was simply to test a possible therapeutic approach that took into account the methodologic drawbacks of some earlier studies.

A. Vieregge, MD, M. Sieberer, MD, H. Jacobs, MD, J.M. Hagenah, MD, P. Vieregge, MD, Lubeck, Germany

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Prospective study of local cutaneous reactions in patients receiving IV phenytoin

To the Editor: We read with interest the work by O'Brien et al.,¹ as we recently published a prospective study of the purple glove syndrome (PGS).² The differences in rate of localized cutaneous reactions (LCR) in their series and ours may be due to several factors. The high incidence of LCR in their series (25.2%) may be related to the rate of infusion of phenytoin on different hospital services. At Henry Ford Hospital, the rate of administration is dictated by the Department of Pharmacy and is set at no more than 20 mg/min throughout the hospital, including the Department of Emergency Medicine.²

The reported rates of LCR in this report do not directly compare to our series, where we reported only the rate of PGS. The authors of this study have combined the rates of PGS with other cutaneous reactions. They did not differentiate between PGS and local inflammation as a result of direct trauma of the vein due to a catheter insertion, IV fluid extravasation, and local cutaneous infection.

In our series, only 2 exposures of 179 developed PGS. This small incidence makes us reluctant to say that the rate of PGS is elevated in the elderly. We suspect that the increased incidence of cutaneous findings in the elderly in this report is a nonspecific phenomenon of aging due to the known changes in the skin that occur with increasing age, rather than a specific cutaneous reaction to phenytoin. We would be interested to see the PGS rates separated from other cutaneous skin findings. In addition, we wonder if O'Brien et al.¹ have any data on the rates of these other nonspecific skin reactions in an age-matched control group in their hospital.

Jorge G. Burneo, MD, *Birmingham, AL*; Gregory L. Barkley, MD, *Cleveland, OH*

Reply from the Authors: We thank Drs. Burneo and Barkley for their interest in our article.¹ We note that they found an incidence of PGS in three (not two) of 179 exposures in their recently published prospective study.² We agree that local practices are likely to influence the rate of reactions following IV phenytoin administration, particularly as they relate to the risk of extravasation of the highly irritant solution. The slow rate of infusion (20 mg/min) dictated at their hospital may well have contributed to the low rate of the PGS that they found. In contrast, at our hospital, the

infusions could be administered up to 50 mg/min, and the actual rate of administration was rarely recorded. Differences in the patient populations may have also been a contributing factor, with our patients being older (median age 65 vs 57 years) and therefore potentially more vulnerable to developing IV extravasations.³

We also agree that differences in terminology and definition may also be important. The site of the edema, discoloration, and pain complicating IV phenytoin infusions is dependent on the site of its administration, and in many cases will be prominent proximally in the forearm or arm rather than distally in the hand, as in classic PGS. For this reason we preferred the site-independent term "LCR," rather than attempting rather arbitrarily to classify reactions as "PGS" according to their site. Burneo et al.² classified reactions as PGS only when they occurred distal to the IV site. Of interest, their incidence per exposure (1.7%) was similar to that of our previous retrospective study from the Mayo Clinic (9/6/77; 1.3%), where a more strict definition of PGS was used.³ However, we dispute the suggestion that the LCR reported in our current study represented nonspecific reactions related to direct trauma, uncomplicated fluid extravasation, infection, or age-related changes. To be classified as LCR, the reactions needed to have discoloration and edema, to be present within 24 hours of the infusion, and to have progressed after the commencement of the infusion—all features that would not be expected with any of the above alternate explanations. Further, as a comparison group, we prospectively examined the limbs of 16 general ward patients who were given IV benzodiazepines during the last 4 months of our study (mean age 53 vs 65 years for the phenytoin patients, $p = 0.27$; Student's t -test), with no cases fulfilling the criteria for a phenytoin LCR detected.

Terence J. O'Brien, MD, FRACP, Frank J. Vajda, MD, FRCP, FRACP, *Melbourne, Australia*

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Fatigue is not associated with raised inflammatory markers in multiple sclerosis

To the Editor: Fatigue is an important complaint in patients with MS. It is probably one of the most frequent and sometimes one of the most disabling symptoms. In the course of the past year, many groups have increased the research in this important aspect of MS. So far, there has been no clear explanation for its pathogenesis. Several treatments have been tried, but they are mostly empirical and have not been completely successful.

The recent study by Giovannoni et al.¹ is very interesting, and some considerations may be made. Their results indicate a lack of correlation between fatigue and some inflammatory markers and depression, showing clear differences to other studies.^{2–4} We have studied the pathogenesis of fatigue in MS.⁵ Our experience agrees in some aspects with their study, especially in stressing the lack of relationship with depression and in the relevance of fatigue in patients with mild MS. However, we obtained different results in the correlation with the immune activation.

One of the reasons for all these discrepancies may be the problem with the concept of "fatigue." Patients describe fatigue in many different ways. This was the reason we addressed the problem of the definition of fatigue when applied to patients with MS. In an initial study, we distinguished between "signs of fatigue" (objective signs of lower functioning [speed, strength, potency]) and the "symptom fatigue" (subjective feeling of tiredness).⁶ In a subsequent article,⁷ we concluded that the symptom fatigue is not a single uniform complaint. We found that the symptom fatigue has at least three components: asthenia (fatigue at rest), fatigability (fatigue after exercise), and worsening of symptoms with the

activity. Our data also indicated that the pathogenesis of these modalities may be different: asthenia would be related to the immune activation, whereas fatigability would be related to the pyramidal deficits.⁵

The consideration of fatigue as one single symptom may be the reason for the difference in opinion among the authors. We think that if people working with MS group all types of fatigue as one symptom, it loses specificity and the research on the pathogenesis or treatment will be inaccurate. If we use the term *fatigue* for different symptoms or signs we will probably get different results. If we do not have a single definition for fatigue and its subtypes, it will eventually be impossible to obtain homogeneous conclusions in this field.

Jorge Iriarte, MD, *Pamplona, Spain*

Reply from the Authors: Dr. Iriarte raises several valid points on the difficulties facing researchers working on MS-related fatigue. His previous work² supports the hypothesis that fatigue is heterogeneous and should be viewed as a symptom complex or as a syndrome with different underlying pathogenic mechanisms.¹ Whether there is a difference in the percept arising as a result of fatigue at rest (asthenia), with exercise, or as associated with worsening symptoms requires further study. Cognitive neuroscientists may be able to address this question, using, for example, functional imaging, in normal subjects. A clue that fatigue may be due to focal brain disease was presented in a recent article by Colombo et al.,⁸ in which patients with MS with fatigue had a significantly higher lesion load in the parietal lobe, internal capsule, and periventricular trigone compared with patients with MS

without fatigue. Although this study needs to be reproduced, it may, however, be flawed by the problem highlighted in our article¹ concerning the validity of current fatigue rating scales. Therefore, we recommend that before researchers embark on new experimental paradigms, this problem should be addressed by the development of specific and well-validated fatigue outcome measures.

G. Giovannoni, PhD, *London, United Kingdom*

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Cholesterol and Alzheimer's disease: Is there a link?

To the Editor: We read with great interest the article by Simons et al.¹ In 1994, we reported an association of soluble form of amyloid β protein with high-density lipoproteins and functional role for amyloid β in cholesterol esterification and lipid synthesis.²

However, it is premature to make definite conclusions on the beneficial role of cholesterol lowering in patients with Alzheimer's disease (AD). This is because understudied neural cholesterol functional biochemistry is not that clear. The effect of statins on cognitive function is also unclear.^{3,4}

It is possible that cholesterol homeostasis failure itself plays a primary role for neurotransmission and synaptic plasticity failure, and Alzheimer-like neuronal degeneration.^{5,6} Thus, cholesterol pathology may represent the primary cause for several AD hallmarks not limited to brain amyloid.⁷

In addition, the authors state that apolipoproteins and lipoproteins do not play a role in lipid transfer from the brain to the circulation. Several recent studies point to the existence of such a mechanism.^{8,9}

Alexei R. Koudinov, MD, PhD, *Rehovot, Israel*;
Temirbolat T. Berezov, MD, PhD, *Dr Sci*,
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Prolonged cortical silent period after transcranial magnetic stimulation in generalized epilepsy

To the Editor: I read with interest the article by Macdonell et al.¹ describing a prolonged cortical silent period (CSP) in untreated patients with idiopathic generalized epilepsy (IGE). This study confirms the value of transcranial magnetic stimulation (TMS) as an investigational tool for the pathophysiology of epileptic syndromes. Conversely, it raises a number of new questions. For instance, what is the net excitatory state of the motor cortex in IGE if both excitatory and inhibitory networks are overfunctioning, as the authors conclude? Is it normal? A possible answer to this would have been to measure the size (area) of the motor evoked potential (MEP) preceding the MEP, or measuring the stimulus/response (MEP recruitment) curve. Previous studies showed that the MEP size is a crucial factor for the length of the subsequent silent period,² and MEP recruitment is a good index of the net corticospinal excitability.³ Studying the CSP as the sole TMS variable may leave some uncertainty about the overall excitatory state of the motor cortex. More thorough TMS investigations have already appeared in the epilepsy field, which also dealt with the CSP, but patients were mainly of the partial type.^{4,7} It may be of some pathophysiologic importance that a lengthening of the CSP, similar to that found by Macdonell et al.¹ in IGE, emerged in patients with an epileptic focus in the primary motor cortex.^{5,6} In contrast, CSP studies in temporal lobe epilepsy were negative.⁷

Roberto Cantello, *Italy*

Reply from the Authors: In previous work using TMS in IGE, we showed that mean motor threshold measurements are slightly

lower in patients with IGE.⁸ This indicates a slight tendency towards hyperexcitability of the motor cortex in patients with IGE compared with controls. This is best explained, given the results of our studies using the CSP and long interstimulus interval recovery curves in patients with IGE, as a result of a balance between hyperexcitability of both excitatory and inhibitory networks.^{1,9}

Use of other TMS techniques to further explore these issues would add more to our understanding of the complex pathophysiology underlying IGE.

It is interesting that studies of CSP in patients with partial epilepsy with a focus in the primary motor cortex demonstrate CSP changes similar to those seen in our patients with IGE. It may be that the molecular mechanism of a number of epilepsies raises the excitability of both excitatory and inhibitory networks, either locally or more generally within the brain, depending on the nature of the epilepsy.

R.A.L. Macdonell, MD, M.A. King, MD, M.R. Newton, MD, J.M. Curatolo, MD, D.C. Reutens, MD, S.F. Berkovic, MD,
Heidelberg, Australia

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Very late onset Friedreich's presenting as spastic tetraparesis without ataxia or neuropathy

To the Editor: We read with interest the recent report by Lhatoo et al.¹ We observed similar findings.² Clinicians should search for a mutation in the frataxin gene in patients with spastic paraparesis and possible recessive inheritance. In contrast, Wilkinson et al.³ did not find any mutation in the frataxin gene in eight patients with isolated spastic paraparesis. Although it rarely occurs, Friedreich's ataxia should be considered in patients with isolated spastic paraparesis.

Pierre Labauge, MD, PhD, *Nîmes, France*

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Corrections

Epidemiologic study of 203 sibling pairs with Parkinson's disease: The *GenePD* study

In the article, "Epidemiologic study of 203 sibling pairs with Parkinson's disease: The *GenePD* study" by Maher et al. (*Neurology* 2002;58:79–84), the name of one of the authors was inadvertently omitted. Cheryl H. Waters, MD should be the 14th author listed. The authors apologize for this error.

Areas of consensus in withdrawing life-sustaining treatment in the neurointensive care unit

In the Letter to the Editor "Areas of consensus in withdrawing life-sustaining treatment in the neurointensive care unit" by Pullicino et al. (*Neurology* 2001;57:1144), the last two sentences mistakenly suggest that "a neurologist who is opposed to withdraw of nutrition and hydration should actively seek out a physician who is prepared to do this." The authors would like to emphasize that the physician who has ethical objections to the withdrawal of food and water should not be involved in the patient transfer in any way. The conclusion of the letter should read, "If the patient decides to refuse ordinary treatment, there may, in some instances, be little that the physician can do to prevent this, but there remains at least the duty to attempt to persuade the patient otherwise or, failing that, for the physician to remove himself from the case so as not to be guilty of complicity in suicide."¹

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Areas of consensus in withdrawing life-sustaining treatment in the neurointensive care unit

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