

Correspondence

Consent issues in the management of cerebrovascular diseases

To the Editor: In the special article on consent issues in the management of cerebrovascular diseases,¹ the authors put the cart before the horse by emphasizing the tradeoff between immediate risk and long-term benefit. They note that for carotid endarterectomy, accurate local data on morbidity and mortality for the surgical and medical options are essential components of the adequate-information component of informed consent. They neglect evidence that this information is usually not available²⁻⁴ and is difficult to empirically ascertain,³ and do not mention that there is evidence suggesting little or no benefit in many locales.^{3,5} The following discussion concentrates on endarterectomy for high-grade symptomatic stenosis.

The benefit demonstrated in NASCET⁶ is based (among other caveats) on a 2.1% major complication (major stroke or death) rate with benefits diminishing until they disappear at a rate of 10%.⁶ In clinical practice, many surgeons are performing endarterectomies with unacceptable complication rates. From 1992 to 1993, the perioperative mortality rate among Medicare patients alone (1.4% to 2.5%)³ is close to or exceeds the major complication rate in NASCET. These rates combine asymptomatic and symptomatic cases. If, as found in clinical trials, the perioperative mortality rate in the symptomatic case is greater than in the asymptomatic case,³ the previously cited rates mask an even greater mortality rate in the symptomatic case.

The difficulty in determining individual complication rates is illustrated in the following example. Consider three surgeons with complication rates of 0.1 (no benefit), 0.06 (diminished benefits), and 0.021 (full benefit). Using a Bernoulli model,⁷ the average number of operations before the first major complication would be 9, 15, and 46, respectively.⁷ In New York state during 1995, 48% of surgeons performed 5 or fewer operations and 73% performed 15 or fewer.⁴ These figures combine symptomatic and asymptomatic cases. Failures occurring more than a year after nine successful operations will tend to be ignored.

The conclusion that there is no benefit with a complication rate of 10% is likely based on the assumption that locally performed uncomplicated operations result in the same risk reduction in stroke as that obtained by the carefully chosen NASCET surgeons. However, the complication rate may be a surrogate marker for surgical proficiency⁸; i.e., a higher complication rate may correlate with less (or no) risk reduction for successful operations. In this case, even assuming the local risk of stroke in a medically treated group is the same as that found in NASCET, the lack of benefit would occur at a complication rate of less than 10%.

However, a local population cannot be assumed to have the same underlying prognoses as that of the trial population.⁸ The two major trials of endarterectomy for symptomatic stenosis^{6,9} had similar eligibility criteria but the risk of ipsilateral stroke in the medically treated groups averaged 5.6%/year in ECST⁹ and 13%/year in NASCET,⁶ a 7.2% difference ($p < 0.003$). The difference has been attributed to different methods of measuring angiographic stenosis.¹⁰ Differences will be exaggerated in practice, where surgeons may operate on the basis of different imaging methods such as ultrasound or MRA.¹⁰ Local indications for endarterectomy may differ from the eligibility criteria for the trials in other ways. In New York state, between 1990 and 1995, 12.2% of endarterectomies were performed on patients with potential sources of cardioembolism.⁵ Thus, determining the local benefit of endarterectomy would require a repeat trial under local conditions, with a sufficient number of patients. This is a practical impossibility in view of the prevalence of low-volume hospitals.^{3,4}

Few people would choose to fly knowing that their plane had been tested only under ideal weather conditions.

Martin M. Pincus, MD, PhD, *Brooklyn, NY*

Reply from the Authors: I thank Dr. Pincus for his informative letter. He convincingly shows that it may be difficult or impossible in practice for neurologists to know confidently the "accurate local data on morbidity and mortality" that we stipulated was a condi-

tion for a patient's true informed consent before carotid endarterectomy. This circumstance produces a clinical quandary: whether a neurologist should recommend carotid endarterectomy or medical treatment in the setting of symptomatic high-grade carotid stenosis, in the absence of any knowledge of the operating surgeon's record of morbidity and mortality.

Given the NASCET data, it would be wrong for a neurologist to recommend endarterectomy without at least some knowledge of the endarterectomy morbidity and mortality data of the operating surgeon. Only with this knowledge can a neurologist make a reasonable assessment of whether the expected benefits of endarterectomy exceed that of medical treatment. This is why we stipulated that this information should be required for a patient's true informed consent. Clearly, it is also necessary for a rational treatment recommendation by the neurologist. The nagging unanswered question is how accurate this knowledge must be for a neurologist's rational treatment recommendation and a patient's informed consent.

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Fatigue in MS: Cross-sectional correlation with brain MRI

To the Editor: Bakshi et al.¹ noted that fatigue severity did not correlate with MRI plaque load. A sodium channelopathy would appear to be the leading hypothesis to explain the fatigue. Brinkmeier et al.² demonstrated that CSF from MS patients decreases sodium channel currents in human skeletal muscle within 5 seconds. The speed of alteration makes an antibody or inflammatory response unlikely. A channelopathy would also explain the prolongation of the relative refractory period and changes in the supernormal period in peripheral nerves from MS patients.^{3,4} Lidocaine, a sodium channel blocker, elicits reversible visual evoked potential amplitude changes in MS patients.⁵ Although it may not involve the sodium channel, an axonal channelopathy is also hypothesized to explain the fatigue of Gulf War syndrome.

Pamela Kaires, MD, *San Diego, CA*

Reply from the Authors: I thank Dr. Kaires for her interest in our article, in which we showed the lack of association between fatigue in MS (MSF) and brain MRI atrophy or lesion load on T1- or T2-weighted images.¹ The inability of brain MRI findings to explain MSF has been confirmed recently by two other groups.^{6,7}

This lack of correlation between brain MRI disease assessments and MSF has now been shown using a variety of fatigue measures, including the 9-item¹ and 29-item⁷ Fatigue Severity Scales, the Checklist Individual Strength Questionnaire,⁶ the Daily Fatigue Score,⁶ and Fatigue Frequency measurements.⁶ A variety of brain MRI measures have been used in these studies, including noncontrast lesion load assessment by number of lesions¹ and ordinal¹ and semiquantitative⁶ arbitrary scoring systems. Our study¹ and recent data of Mainero et al.⁷ also show a lack of association between number^{1,7} and volume⁷ of gadolinium-enhancing lesions measured in cross-sectional¹ and longitudinal⁷ studies. Finally, brain atrophy measured by qualitative¹ and quantitative⁶ measures does not explain MSF. Taken together, these similar conclusions reached by three different groups using different methodologies strengthen the hypothesis that neither focal white matter lesions nor atrophy in the brain are sufficient to explain MSF.

The question is what other aspects of the MS disease process contribute to the pervasive fatigue experienced by so many patients. Other avenues of investigation toward the study of MSF include the effects of MS on cerebral gray matter and the consideration of peripheral mechanisms, both of which would be missed by conventional MRI. A growing body of evidence indicates that MS is a global brain disease that has important effects on both cortical and subcortical gray matter and that this gray matter involvement is common. Recent neuroimaging and pathologic studies have emphasized this effect of MS on gray matter. PET studies show that MS patients have widespread brain hypometabolism including cortical and subcortical gray matter.^{8,9} This hypometabolism worsens over time.⁹ Hypointense lesions on T2-weighted images are commonly seen in the basal ganglia and thalamus, and occasionally in the cerebral cortex, suggesting T2 shortening due to pathologic gray matter iron deposition in MS.¹⁰ Direct plaque-like involvement of gray matter by the MS disease process has been shown in autopsy studies.¹¹ Roelcke et al.¹² showed that hypometabolism in the frontal cortex and basal ganglia is associated with MSF and hypothesized that this may represent an effect of diaschisis due to white matter lesions. However, the inability to associate MSF with MRI white matter lesions suggests that direct involvement of cortical and subcortical gray matter rather than diaschisis should be further explored as a cause of MSF. Direct mechanisms of gray matter involvement contributing to fatigue could include demyelination, inflammation, neuronal injury, or iron deposition. In addition, as suggested by Dr. Kaires, dysfunction of cell membranes in the central or peripheral nervous system is another factor that might influence MSF.

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Ibuprofen treatment versus gradual introduction of interferon β-1b in patients with MS

To the Editor: We read with interest the article by Rice et al. on ibuprofen treatment and gradual introduction of interferon (IFN) β-1b in patients with MS to minimize flu-like symptoms.¹ They conclude that gradual dose introduction and use of nonsteroidal anti-inflammatory drugs could improve long-term tolerability. We agree. In fact, at least in Europe, this is the usual way to start therapy: begin with a half dose and then use paracetamol or ibuprofen for the management of flu-like symptoms appearing early on. Such recommendations notwithstanding, flu-like symptoms limit dosage in a number of patients. Recently, *Neurology* published our article describing how low-dose oral steroid use is helpful in reducing flu-like symptoms at onset of IFNβ-1b therapy in patients with MS.² In it, we studied 71 patients who started IFNβ-1b. Patients were randomized to receive prednisone plus paracetamol or paracetamol only and were monitored for side effects. Systemic side effects were minimal in the steroid group compared with the nonsteroid group during the first 15 days of treatment ($p = 0.005$). There were no side effects attributable to steroids. Rice et al. describe that ibuprofen alone or in combination with dose escalation increased the frequency of injection site reactions. Steroid use when starting therapy has been our policy for the last 3 years. Thus far, there are 148 patients with MS on IFNβ-1b with no drop-outs due to systemic effects.

Rice et al. suggest that fever during IFN therapy is directly mediated by the binding of IFN to thermosensitive neurons in the hypothalamus and secondarily by the release of prostaglandins, which cause fever. However, we recently showed a strong relation between peripheral blood mononuclear cells' (PBMC) release of interleukin (IL)-6 and fever at the onset of IFN therapy.³ Likewise, we observed that treatment with low-dose steroids decreases the production of IL-6 in patients without fever. We believe that the mechanism of action of IFN in the development of fever is mediated by the release of IL-6 by PBMC.

We agree that minimization of IFN side effects is essential to the long-term use of the drug. Therefore, besides the recommendations as to gradual dose introduction and use of nonsteroidal anti-inflammatory drugs, the use of steroids could be helpful in reducing flu-like symptoms and improve long-term tolerability of IFNβ-1b.

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Parkinson's disease in twins

To the Editor: Some years ago I was asked by an asymptomatic cotwin of a patient with PD whether an [¹⁸F]dopa PET scan could predict whether he would develop the illness. Vieregge et al.'s article¹ is the second recent study examining the progress of asymptomatic twins identified as having abnormal, and putatively presymptomatic, scans. The editor and authors should be congratulated for publishing what is essentially a negative study; not one of seven asymptomatic cotwins with an abnormal scan has developed PD in 6 years. An important explanation is omitted from the discussion. [¹⁸F]dopa PET is a technique that has limited reproducibility, numerous potential technical errors, and in the identi-

fication of preclinical disease, is likely to produce false positives and false negatives.² Without reproducibility data, the authors and readership cannot be confident of the reliability of identification of abnormal subjects. This suggests that the technique may have erroneously identified preclinical subjects. The recent similar follow-up study from this country³ does not give any greater confidence in the technique. In that study, 10 of 18 asymptomatic monozygotic (MZ) cotwins and 3 of 16 asymptomatic dizygotic (DZ) cotwins had abnormal PET scans; thus far, only 4 of the MZ cotwins have developed clinical PD. Three of these four had tremor and one had minimal limb bradykinesia at the time of the first scan.⁴ The implication is that the technique used in both these studies cannot identify asymptomatic cotwins of patients (and, by extrapolation, preclinical individuals in the general population) who are likely to develop typical PD with any useful level of sensitivity or specificity.

P.K. Morrish, MD, *Brighton, UK*

Reply from the Authors: We thank Dr. Morrish for the interest in our paper. However, we do not agree with the opinion that it represents a “negative” study. Rather, our results together with other recent data should lead to a re-evaluation of some of the current hypotheses about the genetic etiology of PD. Most importantly, our longitudinal results concur with those of the cross-sectional US veterans twin study⁵ that was published while our paper was in press. Both studies show that a genetic component cannot be demonstrated in the etiology of the motor syndrome of Lewy body parkinsonism according to clinical criteria when discordant MZ and DZ twin pairs are compared when gathered from an overall “sporadic” PD population. Our study also shows that five out of seven (not seven, as stated by Dr. Morrish) motor-asymptomatic cotwins (3 MZ; 2 DZ) had abnormal striatal influx constants of 18-fluorodopa during an initial study of PET⁶ but did not develop PD motor signs after 6 years of clinical observation.

Dr. Morrish questions whether the 18-fluorodopa PET technology is sufficiently reproducible to identify a “preclinical” subject with PD. As long as we regard the disorder of the abovementioned twin samples as similar to that of the overall sporadic PD population, we will not be surprised to observe considerable variations in factors such as age at onset, disease course, clinical typology, associated features (e.g., depression, dementia, vegetative system), treatment response, and disease duration between and within MZ or DZ twin pairs. This also applies to the length of the preclinical period. It is not surprising that different mathematical extrapolations of the 18-fluorodopa metabolism provided a duration of 3,⁷ 10 to 15,⁸ or of 40 to 50 years⁹ for the preclinical period in sporadic PD. This preclinical period is, at present, poorly understood in its pathobiochemical features. Moreover, such features may differ according to other factors, such as the underlying etiology, age, sex, and concomitant medication. Again, it is not surprising that PET results vary in subjects presumed to be “pre-clinical” PD cases. In support of this statement, even victims of accidental MPTP exposure show fairly diverse interindividual evolutions of clinical features and longitudinal 18-fluorodopa PET data, ranging from a normal to a definite parkinsonian state.¹⁰

Furthermore, the assumption that the entire period between the age at onset in the index twin and the time point at which the primarily asymptomatic cotwin becomes clinically affected represents his or her “preclinical” stage is not proved for the PD twins. Our earlier PET data⁶ and those of Piccini et al.³ show heterogeneity in the metabolism of 18-fluorodopa in motor-asymptomatic cotwins, with even normal values in some asymptomatic MZ cotwins. Given the current knowledge and despite methodologic explanations about PET, these findings could reflect varying metabolic states that are heterogeneous between the various discordant twin pairs. For the latter reason, we agree with Dr. Morrish’s statement that the results of the study of Piccini et al.³ are not at variance with our data.

PET investigators have repeatedly pointed out that both reproducibility and sensitivity of 18-fluorodopa PET measurements are important in the estimation of both the progression of PD^{11,12} and of the process of normal aging.^{7,9,13} Piccini et al.³ have demonstrated that their technology of 18-fluorodopa PET can identify single asymptomatic cotwins who develop PD later on (although this report lacks detailed clinical criteria and descriptions). We concur with Dr. Morrish that PET data of PD twins, particularly

those of asymptomatic twins, deserve careful evaluation before interpretation in a clinical context. However, current knowledge of clinical genetics and of PET technology teaches that in PD the inconsistencies of such an interpretation cannot be exclusively attributed to the limitations of the PET method. It is useful to remember that PET technology per se does not and cannot prove that the metabolic differences between MZ and DZ pairs or within familial PD are genetic in origin.

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Catalepsy after stroke

To the Editor: We read with interest the report by Saposnik et al.¹ on catalepsy after stroke and agree that the phenomenon is commonly overlooked. We have avoided using the term catalepsy because of its psychiatric connotation, and also because the motor phenomenon seen after stroke is not typically associated with the waxy hypertonia reported in the catatonian type of catalepsy. We prefer to call it *motor persistence*,² because it is, in a sense, the exact opposite of motor impersistence, when patients cannot maintain a specific posture on command more than a few seconds. Motor persistence may correspond to the tonic counterpart of hyperkinetic motor behaviors contralateral to hemiplegia in acute stroke,³ which is a phasic motor disturbance. Interestingly, we found that motor persistence is more frequent with right-side lesion, with coexisting hemineglect,³ whereas hyperkinetic motor behaviors were more commonly present with left-side lesion.³ Because Saposnik et al. found a left predominance of lesions associated with motor persistence,¹ further studies may clarify the lateralization issue.

Julien Bogousslavsky, MD, Joseph Ghika, MD, *Lausanne, Switzerland*

Reply from the Authors: We appreciate the interest and comments of Drs. Bogousslavsky and Ghika on our work. We think it is difficult to distinguish between catalepsy and motor persistence. The latter implies the inadequate maintenance of a posture or position upon the examiner's command,² whereas catalepsy has been defined as the tendency to maintain postures induced by the examiner.^{4,5} Our patients maintained postures passively applied to them, without any verbal command. Furthermore, three of them were aphasic (unable to understand commands). One interesting feature of most of our patients was that they had left-sided hemispheric lesions, whereas motor persistence was described mostly with lesions of the right prefrontal and premotor regions.² An update of our series of catalepsy in stroke showed that six out of seven patients had left-sided lesions (unpublished data).

Nevertheless, even taking into account these differences, we may be looking at the same motor phenomenon, regardless of lateralization or way the posture is induced. We believe that both the phenomenon named motor persistence by Drs. Bogousslavsky and Ghika and the cataleptic postures we found are commonly overlooked in clinical practice. We agree that further studies are

needed to better understand the mechanisms underlying these phenomena.

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Corrections

In the article "Rate of functional decline in Huntington's disease" (*Neurology* 2000;54:452–458) by Marder et al., one of the participating sites was omitted from the Appendix. William Mallonee, MD, David Palmer, MD, Gregory Suter, Hereditary Neurological Disease Centre, Wichita, KS, should have been included. The authors apologize for the error.

In the article "Frequency and duration of hospitalization of patients with AD based on Medicare data: CERAD XX" (*Neurology* 2000;54:740–743) by Fillenbaum et al., the percentages for race in the first numeric column of table 1 were reversed. They should read: white 87.0, black 13.0. The authors apologize for the error.

In the article "Long-term treatment with vagus nerve stimulation in patients with refractory epilepsy" (*Neurology* 1999;53:1731–1735) by Morris et al., several individuals were omitted from the Vagus Nerve Stimulator Study Group Appendix. Brad Vaughn, Chapel Hill, NC; Adrian Upton, Hamilton, Ontario, Canada; Jack Wilberger, Pittsburgh, PA; H. Stefan, Erlangen, Germany; and Edward Faught, Birmingham, AL, should have been included. The publisher apologizes for the error.

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Long-term treatment with vagus nerve stimulation in patients with refractory epilepsy

Neurology 2000;54;1712
DOI 10.1212/WNL.54.8.1712

This information is current as of April 25, 2000

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