Not all TIAs are equal

To the Editor: The study of Rothrock et al¹ concerning the clinical and angiographic correlation of crescendo transient ischemic attacks (TIAs) is a timely one. More detail is required about clinical and pathophysiologic subtypes of TIA, particularly in view of the modest results of the recent UK aspirin trial² and the implications for the ongoing trials of carotid endarterectomy in both the US and Europe.

We have recently drawn attention to the characteristic features of crescendo capsular TIAs,^{3,4} termed the "capsular warning syndrome": low incidence of angiographic abnormalities but high incidence of early stroke. This form of crescendo TIA is a clear pathophysiologic subgroup of some importance.

A more logical and practical approach to future trials of medical or surgical therapy in patients with TIA may be to study such discrete pathophysiologic subsets in smaller numbers.

Geoffrey A. Donnan, MD
Peter F. Bladin, MD
Melbourne, Australia

Reply from the Authors: Drs. Donnan and Bladin imply that TIA should not be considered a diagnosis in and of itself, and that "blanket" application of any therapy, old or new, to the TIA population at large is likely to yield statistically disappointing results. We agree completely. As for patients with presumed lacunar TIAs and normal arteriograms, we, too, have noted a high incidence of early stroke but a relatively good, long-term functional outcome.⁵

John F. Rothrock, MD
San Diego, CA

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Carnitine palmitoyltransferase I and II

To the Editor: Zierz and Engel^{1,2} published experiments favoring the existence of a bifunctional carnitine palmitoyltransferase (CPT), catalyzing both CPT I and II. In their opinion, the absence of latent CPT activity in intact mitochondria proves that CPT II does not exist. However, their forward and backward assays are unable to distinguish between CPT I and II. Moreover, latency of citrate synthase does not imply that CPT is also latent.

Arguments in favor of the existence of CPT I and II in muscle mitochondria and their independent measurement were formulated in an earlier letter to this journal.³ CPT I and II were also measured independently in mitochondria from liver and heart. Hepatic CPT I showed a partial latency; the other activities were overt.⁴ We detected two patients with CPT II deficiency, each of whom had an asymptomatic sister with CPT II deficiency in leukocytes. The activity of CPT I was normal. In one other male patient we found a combined CPT I and II deficiency.⁵

We postulated that in patients with CPT II deficiency long-chain acylcarnitine, formed by the action of CPT I, protects the muscles from neutral lipid storage during fasting.⁴ In most reported patients with CPT deficiency, CPT I and II were not measured independently. The absence of lipid accumulation, then, implies that they suffer from CPT II deficiency.

Additional evidence on the existence of separate CPT I and II was

Figure. Fatty acid catabolism by mitochondria. PCS = palmitoyl-CoA synthetase, CPT = carnitine palmitoyltransferase, ATP = ATP synthetase. The catalytic site of CPT I is on the inner face of the outer mitochondrial membrane, while the malonyl-CoA binding site of the regulatory subunit is on the outer face of the membrane.⁶ Redrawn from reference 7.

recently presented by Murthy and Pande,⁶ who showed by fractionation study of liver mitochondria that CPT I is located on the inner face of the outer membrane (figure).

Hans R. Scholte, PhD
Rotterdam, The Netherlands
Frans G.I. Jennekens, MD
Utrecht, The Netherlands

Reply from the Authors: CPT catalyzes the reversible conversion of palmitoyl-CoA to palmitoylcarnitine using L-carnitine.⁸ According to the laws of thermodynamics and mass action, the direction of the reaction (ie, forward, producing palmitoylcarnitine, or backward, producing palmitoyl-CoA) is solely dependent on the actual concentrations of the substrates and products. Regardless of whether two forms of CPT exist (and if two forms do exist, whether these are different proteins or the same protein differently deployed on mitochondrial membranes), any CPT assay always measures only the portion of CPT that has access to the substrate under the assay conditions. Neither the so-called "forward" assay, nor the so-called "backward" assay, nor the "isotope-exchange" assay can distinguish between CPT I and CPT II. This applies not only to our assay, but also to the studies of Scholte et al.^{3,5,7} The notion proposed by Scholte and coworkers⁷ that the forward assay measures only CPT I is incorrect.

Scholte and Jennekens misinterpret our data and conclusions^{1,2}: We did not postulate the existence of a bifunctional enzyme or an absence of CPT II in skeletal muscle. Rather, we showed that the earlier experimental approaches that imply the existence of CPT I and II in liver mitochondria do not distinguish between CPT I and II in skeletal muscle mitochondria.¹ From this, it follows that selective CPT II deficiency of muscle mitochondria cannot be a serious consideration until the existence and distribution of two forms of CPT in normal skeletal muscle mitochondria has been clearly established.¹ Further, it needs to be emphasized that the latency of citrate synthase is a generally accepted measure of the intactness of mitochondria.

We question the conventional model of CPT I and II in muscle mitochondria.¹ Other recent studies question the conventional model in mitochondria isolated from other tissues. At least four different models of CPT within mitochondria have been proposed to date.⁹ The cartoon presented by Scholte and Jennekens depicts only one of these four possible models.

The recent report by Murthy and Pande⁶ supports the existence of two topographically separate forms of CPT in liver mitochondria. These workers have not demonstrated two separate forms of CPT in skeletal muscle mitochondria. Our data¹ are consistent with two forms of CPT in liver mitochondria, but fail to show two forms of CPT in muscle mitochondria.

The problems of the data and conclusions provided by Scholte et al.^{3,5,7} had been previously discussed by Layzer⁹ in his reply to another letter from Scholte and Jennekens to *Neurology* in 1981.³ Layzer pointed out that the notion of selective CPT II deficiency was tenuous because it was based on studies of homogenates of previously frozen and thawed muscle. Layzer's reply was also noteworthy in that he correctly predicted the existence of an abnormally regulated CPT with altered kinetic properties as the cause of CPT "deficiency."

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Andrew G. Engel, MD

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Pathologic diagnosis of Alzheimer's disease

To the Editor: This letter concerns the article by M.C. Tierney et al.¹

In their report, Tierney et al appropriately concluded that there is a need for universally accepted pathologic criteria for the diagnosis of Alzheimer's disease (AD). This conclusion is based on their comparison of nine different diagnostic criteria for AD that were, in part, based on the quantitation of neurofibrillary tangles (NFT) and plaques (PL). Not surprisingly, these criteria differed with respect to their accuracy, sensitivity, and specificity.

Nevertheless, we would like to draw attention to a possible source of interobserver error in the implementation of the criteria for the diagnosis of AD used by Tierney et al that required the quantitation of NFT and/or PL per $\times 25$ microscopic field. Objectives and oculars attached to different brands of microscopes may circumscribe different viewing areas (ie, microscopic fields) despite being assigned the same magnification factor (see table). Thus, the quantitation of NFT and PL should be reported as the number of each per mm^2 rather than per magnification factor (ie, $\times 25$). As shown in the table, the number of items counted with a $20\times$ objective will differ considerably depending upon whether or not such counts are performed with a Nikon Optiphot (0.4 mm^2 viewing area) versus an Olympus (1.29 mm^2 viewing area) microscope. This potential source of error was considered in the NIA criteria for AD² and should be considered by others who propose to contrast the pathologic criteria for AD with respect to accuracy, sensitivity, and specificity.

J.Q. Trojanowski, MD, PhD

M.L. Schmidt, PhD

Philadelphia, PA

Table. The area circumscribed by four different objectives in three different brands of microscope is shown here*

Microscope	Objective	Diameter	Radius	Area
American Optical	10 $\times$	1.8	0.9	2.54
	45 $\times$	0.4	0.2	0.13
Nikon Optiphot	10 $\times$	1.44	0.72	1.63
	20 $\times$	0.72	0.36	0.40
	40 $\times$	0.36	0.18	0.11
Olympus	20 $\times$	1.28	0.64	1.29
	40 $\times$	0.62	0.31	0.30

* The column at the left specifies the three different microscopes tested. The objectives used to determine the areas viewed in each of these microscopes are shown in the second column from the left. The subsequent columns give the diameter, radius, and area of the microscopic fields viewed with each objective in the different microscopes. All microscopes were equipped with $10\times$ oculars. The units for diameter and radius were mm, and those for area were mm^2 . Note that objectives with the same magnification factors on different microscopes circumscribe different areas.

Reply from the Authors: Drs. Trojanowski and Schmidt are of course quite correct in asserting that a $\times 25$ microscopic field will vary according to the instrument used, and that therefore reports of plaque and tangle frequency would be better based on some absolute unit such as 1 mm^2 .

In our study a Leitz Orthoplan microscope with a $\times 25$ planapochromat objective was used, with a field diameter of 0.76 mm and area of 0.45 mm^2 . To convert to a 1 mm^2 standard, our criteria of "one or more neurofibrillary tangles and one or more neuritic plaques per field" ought therefore to read "two or more neurofibrillary tangles and two or more neuritic plaques per mm^2 ." In other respects, our study appears to fulfill the procedural conditions laid down in the NIA criteria for AD.²

The letter from Drs. Trojanowski and Schmidt further underlines the need for universally accepted criteria for the pathologic diagnosis of Alzheimer's disease which we noted in our article.

Anthony J. Lewis, MD

Mary C. Tierney, PhD

Rory H. Fisher, MD

Maria L. Zoritto, MD

W. Gary Snow, PhD

David W. Reid, PhD

Paula Nieuwstraten, MD

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Optic neuritis and MS

To the Editor: The recent article by Rizzo and Lessell¹ is informative but contains some unwarranted conclusions. The authors depend upon clinical criteria as well as the life-table analysis to arrive at their conclusions. Previous clinical investigations have yielded few clues to indicate which patients with optic neuritis (ON) would develop clinical MS. Their cohort of white patients living in New

England raises issues of uncertainty as to the comparability of subjects and overrepresentation of some geographic "hot spots," and it neglects aspects of genetic factors. I also believe this study is seriously limited in its conclusions regarding augmented risk without the benefit of MRI data. The following personal study of 16 cases of ON, studied with MRI, suggests that this diagnostic tool offers potential predictive accuracy as to who may develop MS.

Sixteen consecutive patients were diagnosed with ON. Four patients (2 women, 2 men) displayed multiple high-signal intensity lesions in the periventricular matter on spin-echo sequences, consistent with disseminated plaque formation. All these lesions were clinically silent. No lesions were discovered in the optic nerves. Within 6 months of diagnosis of ON, the four patients developed clinical MS. Of interest is the finding that 2/4 had positive family histories of several members with MS. CSF abnormalities were present in less than 50% of the entire series. One of the men with a positive family history was Hispanic.

MRI imaging appears to be the most sensitive indicator of demyelination in clinical medicine. Because MS is such a common problem, with variability of expression, it is not surprising that Jacobs et al² did not see any overt symptoms of MS for a 2-year period after onset of ON, whereas Rizzo and Lessell found over 50% conversions during the first 2 years. If a large series of cases with ON were studied prospectively with MRI, a spectrum of clinical expression would become apparent. Perhaps the number of demyelinating plaques seen on MRI is the key factor related to prognosis. Additionally, future protocols could address the issue of responsiveness to corticosteroids and prognosis.

Familial clusters of MS have been reported as occurring 5% of the time. McAlpine et al³ have calculated that first-degree relatives are at least 5 to 15 times at greater risk for developing MS than the general population. In view of the observation that 2/4 individuals had positive family histories and became symptomatic within 6 months, it would seem reasonable to consider MRI as a useful noninvasive screening procedure for prognosis among family members. The issue of genetic aspects was not mentioned in the authors' series.

In conclusion, a long-term prospective study is needed with MRI and family screening data in order to accurately understand the close relationship between ON and MS.

Michael I. Weintraub, MD
Croton-on-Hudson, NY

Reply from the Authors: Dr. Weintraub accurately states that it would be unwarranted to indicate that our conclusions necessarily applied to all populations. That is why we included a statement in both the summary (first line) and in the text (first line, last paragraph) that our patients were white and living in New England.

The importance of genetic factors in the etiology of MS remains controversial. None of our patients had a first-order relative with MS at the time of their episode of ON, and thus the family history could not have helped predict which patient would develop MS.

Dr. Weintraub is troubled that we depended upon clinical criteria to diagnose MS and considers our study flawed by the lack of MRI data. Of course, MRI was not available when the patients entered the study, so the issue of using MRI to predict which of our subjects would develop MS is moot. However, we reject the notion that MRI has been established as a means of unequivocally diagnosing MS. The articles by Paty et al⁴ and by Kurtzke⁵ in the same issue of *Neurology* address this topic directly. Paty prospectively compares clinical evaluation, evoked potentials, oligoclonal banding, MRI, and CT in the diagnosis of MS and concludes that the "clinical diagnosis of MS . . . even though only 95% accurate, must remain the gold standard." Kurtzke concludes "Until we have a pathognomonic laboratory test, if we wish not to erase 120 years of clinical experience and start *de novo* to characterize this illness as a clinical entity, then I believe we must adhere to the clinical phenomena." We agree.

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Simmons Lessell, MD
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Ptosis and Hering's law

To the Editor: I read with interest the article entitled, "Unilateral ptosis and Hering's law"¹ and wish to offer one comment. Dr. Lepore studied 21 patients with unilateral ptosis and found only one patient with contralateral superior lid retraction. Hering's law states that there is bilateral equal and simultaneous innervation of synergistic extraocular muscles. By applying this law to the levator muscles, we would expect more patients in this study to have contralateral lid retraction.

With respect to the analysis of lid and ocular motility, it is important to identify the fixating eye. For example, a paretic left sixth nerve will cause a greater (secondary) deviation when the left eye fixates as opposed to the right eye. Similarly, it matters whether the ptotic or fellow eye is fixating when comparing lid fissures. While there was "no change in ptosis behind the occluder"¹ (eye with ptosis is covered, fellow eye is fixating), no mention is made of ptosis behind the occluder with the fellow eye covered and the ptotic eye fixating. Was it the failure to specify the fixating eye that led to the finding of only one in 21 patients with unilateral ptosis who demonstrated lid retraction of the contralateral eye?

In addition, other exceptions to Hering's law include dissociated vertical deviations, unilateral nystagmus, convergence, and Bell's phenomenon.

Mark S. Dresner, MD
Bronx, NY

Reply from the Author: Dr. Dresner's speculation that the presence of lid retraction of the fellow eye in patients with unilateral ptosis may be underestimated if the ptotic eye is not allowed to fixate was not confirmed by my series of patients. As part of my "routine assessment of ocular and lid motility,"¹ alternate cover testing elicited no change of lid position of the fellow eye, ie, lid elevation with occlusion of the nonptotic eye or lid descent with occlusion of the ptotic eye was not observed. Of interest, the one myasthenic patient who had unilateral lid retraction in my series did not demonstrate changing lid position on alternate cover testing. In contrast, Gay et al² found that occlusion of the ptotic eye would lessen the lid retraction of the fellow eye in three patients and, although his finding doesn't directly address Dr. Dresner's contention that lid retraction will increase in the fellow eye when the ptotic eye fixates, it supports the premise that Hering's law governs levator function.

Gay's observations notwithstanding, Hering's law is clinically manifest in very few cases of unilateral ptosis. Kansu and Subutay³ recently observed upper lid retraction in only four of 150 patients with myasthenia gravis, and even questioned the assumption that Hering's law accounts for the lid retraction in these few cases.

Dr. Dresner's thoughtful question as well as his additional listing of ocular motility "culprits" that violate Hering's law is greatly appreciated.

Frederick E. Lepore, MD
New Brunswick, NJ

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Thalamic aphasia

To the Editor: Tuszynski and Petito¹ reported an interesting patient with aphasic speech disturbances due to a discrete left thalamic infarct proved at autopsy. However, it is a pity that an incomplete review of the literature led them to believe that this was the first case of aphasia due to thalamic infarct verified at autopsy. To our knowledge, at least three cases with clinical-pathologic correlation have been published previously.²⁻⁴ All corresponded to a unilateral left thalamic infarct in the paramedian territory; the dorsomedial nucleus and the intralaminar formation were severely involved; the ventral lateral nucleus was only partially involved in two of them,^{3,4} and the pulvinar was spared. It can be argued that in the first two cases,^{2,3} other infarcts were also present; however, none was relevant to the speech disorder, except in Molnár's patient, in whom alexia was present, and was probably related to infarction of the lingual and fusiform gyrus on the left side. In the third case, which we reported in 1986,⁴ the left thalamic infarct was isolated. Speech disturbances included reduced fluency, frequent pauses, decreased volume of the voice, verbal and phonemic paraphasias, mildly impaired comprehension and spared repetition. Other neuropsychological disturbances included right-sided motor neglect and anterograde amnesia. After reviewing the cases of Molnár² and Davous et al,³ we suggested that involvement of the dorsomedial nucleus might be critical because of its connections with Broca's area, Wernicke's area, and the gyrus supramarginalis, whereas involvement of other nuclei such as the ventral lateral nucleus and pulvinar might be of less importance. Since we published our case, we have been able to further study the cerebral cortex of this patient using techniques for degenerated fibers.⁴ This study showed marked axonal degeneration in the premotor cortex, including Broca's area, somatosensory cortex, Wernicke's area, and gyrus supramarginalis on the left side. These data in mind, the case of Tuszynski and Petito may be considered a further confirmation that in thalamic infarction the dorsomedial nucleus is a critical structure for speech dysfunction, which might be related to a thalamic-cortical disconnection phenomenon, as PET studies have suggested.⁵

J. Bogousslavsky, MD

J. Miklossy, MD

J.P. Deruaz, MD

F. Regli, MD

Lausanne, Switzerland

Reply from the Authors: We thank Dr. Bogousslavsky et al for bringing to our attention their own case of thalamic aphasia as well as

two additional cases from the non-English literature. They review their previous hypothesis that thalamic aphasia is related to destruction of the dorsomedial nucleus, and correctly point out that the anatomic location of the thalamic infarct in our patient further confirms this hypothesis.

Clinical pathologic studies of thalamic aphasia, uncomplicated by other clinical symptoms and signs and by extrathalamic infarcts, are infrequent. Our particular case was useful in documenting the existence of aphasia due to a discrete left thalamic infarct since, at the time of the patient's presentation, symptomatology other than speech disturbances were absent. In contrast, other cases are complicated by multiple clinical signs and symptoms and infarcts at the time the patients present with aphasia.

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Correction

The authors have indicated two corrections in the article "Central pontine myelinolysis in severely burned patients: Relationship to serum hyperosmolality" (McKee et al, 1988;38:1211-1217), which appeared in the August issue. The formula on page 1212 for calculating serum osmolality should have been:

Serum osmolality = $2([Na] + [K]) + \text{glucose (mg/dl)}/18 + \text{BUN (mg/dl)}/2.8$.

Also, on page 1214 in the second paragraph, the sixth sentence should read:

"The gray matter of the pons was *abnormal* in seven patients" [emphasis added].

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Correction

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