Authors' Voluntary Retraction: H. Wilkie, A. Osei-Lah, B. Chioza, L. Nashef, D. McCormick, P. Asherson, and A.J. Makoff: Association of the μ -opioid receptor subunit gene and idiopathic generalized epilepsy. *Neurology* 2002;59:724–728

To the Editor: I am writing with reference to our paper published in *Neurology* some time ago: H. Wilkie, A. Osei-Lah, B. Chioza, L. Nashef, D. McCormick, P. Asherson, and A.J. Makoff: Association of the μ -opioid receptor subunit gene and idiopathic generalized epilepsy. *Neurology* 2002;59:724–728.

We reported that two SNPs in *OPRM1* (A118G and G-172T) were significantly associated with IGE. Unfortunately, a number of genotyping errors have recently come to light. Another research worker in our group has now repeated the genotyping in over 90% of the original DNA samples. Wherever there were discrepancies between the two sets of results, the genotyping was repeated twice more. The corrected results are presented in the table, which shows that neither SNP is significantly associated with IGE.

Clearly our results do not now replicate the significant association reported by Sander et al. (*Epilepsy Res* 2000;39:57–61). As the main message of our paper is incorrect, we therefore wish to retract it. We sincerely apologise for inadvertently allowing erroneous data to reach the public domain. We have now genotyped

SNP/genotype	Probands	Controls	Overall significance, χ^2/p value/df
A118G			
AA	141 (0.72)	159 (0.71)	3.31/0.19/2
AG	48 (0.24)	61 (0.27)	
GG	8 (0.04)	3 (0.01)	
G-172T			
GG	188 (0.92)	205 (0.93)	0.34/0.84/2
GT	16 (0.08)	14 (0.06)	
TT	1 (0.00)	1 (0.00)	

many more SNPs from *OPRM1* and will be submitting this work for publication in due course.

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Andrew Makoff, PhD, Philip Asherson, PhD, Lina Nashef, FRCP

Perinatal stroke in term infants with neonatal encephalopathy

To the Editor: We read with interest the article by Ramaswamy et al.¹ on stroke in term infants presenting with early neonatal encephalopathy. We agree this is an unusual presentation of neonatal arterial territory stroke. We found a similar proportion with stroke (8/197, 4%) in encephalopathic term infants.² However, we found that the majority of these infants do not have evidence of infection, cardiac disease, or prothrombotic disorders.³

Our experience is that cognitive outcome is generally good following unilateral arterial territory stroke. Hemiplegia will develop if basal ganglia, posterior limb internal capsule (PLIC), and hemispheric tissue are involved in the primary lesion.⁴ Cognition is poorer if there are bilateral infarcts or other imaging abnormalities. We have observed a poor cognitive outcome in two infants with small middle cerebral artery (MCA) territory infarcts but no hemiplegia—both are mildly dysmorphic without specific diagnoses despite extensive investigation. The other children with poor cognitive outcomes are those few who later develop seizures.

We think the poor cognitive outcome reported here is mainly due to bilateral tissue involvement; extensive white matter involvement is associated with cognitive deficit.⁵ If stroke is associated with basal ganglia lesions, those will dominate the motor outcome.

In Case 3 (figure)¹ in addition to the right-sided middle/posterior MCA territory infarction, we believe that the image shows severe abnormality in the left basal ganglia and PLIC. It is possible that the left-sided lesions together with the watershed lesions account for the severe outcome rather than the MCA territory lesion alone.

Several of the cited cases are also complicated. Case 1 is an infant of a diabetic mother, a high-risk group. Case 2 has a large infarction on the background of infection that may affect white matter and can be difficult to assess on early conventional images. Cases 5 and 6 had additional lesions not confined to one arterial territory.

We feel the emphasis given to poor outcome with MCA territory infarction is misleading and that the important issue in terms of cognitive outcome is the extent of the additional watershed or bilateral lesions. Nonetheless we strongly support that clinicians need to be aware that neonatal stroke, when it occurs in an unusual context, is more likely to be associated with a poorer

cognitive outcome and such infants deserve close neurodevelopmental follow-up.

F.M. Cowan, MD, PhD, E. Mercuri, MD, M.A. Rutherford, MD, MRCP, London, UK

Reply from the Authors: We thank Drs. Cowan et al. for their comments. It appears that our data¹ and their data² differ in that underlying etiologies were not as common in their series. However, in the 8/197 newborns with evidence of acute injury and neonatal encephalopathy and stroke, we were unable to substantiate whether these affected babies had abnormalities such as prothrombotic disease, infection, or cardiac disease, since the data are not provided.

Cowan et al. reported that many babies had congenital malformations, metabolic disease and infections; these are exclusion criteria for our cohort.¹ However, in the past, this group has reported that 8/11 newborns with cerebral infarction had poor neurologic outcome in association with prothrombotic risk factors, while only 1/13 with normal outcome had these associations.⁶ These data appear to support the data in our cohort.

In addition, in the largest series published to date, 127 out of 215 neonates with stroke had a prothrombotic state.⁷ It seems that the majority of infants reported to date, including data from Cowan's group, do have evidence of some underlying risk factors for stroke.

We would like to believe that cognitive outcome is "generally good" following unilateral arterial stroke in the newborn, but data from one of the largest neonatal stroke cohorts^{8,9} suggest that >50% have abnormal cognitive and neuromotor outcome. Cowan et al. hypothesize that poor cognitive outcome in their and our series is due to 1) bilateral tissue involvement, or 2) extensive white matter disease. Cases 3, 5, and 6 did exhibit involvement of the watershed zones bilaterally, but areas of signal abnormality were minor. This bilateral involvement may portend a worse prognosis as suggested, but larger series will be needed to confirm this hypothesis. Case 2 did not have other brain regions involved (despite the history that might suggest vulnerability to white matter).

The cases that we reported are complicated in regard to their neonatal course, but these complications only confirm that newborns with neonatal encephalopathy must be considered for neuroimaging evaluation, and that stroke should be in the differential. We never saw "extensive white matter involvement" that Cowan et al. hypothesize might be associated with cognitive abnormalities later in life.⁵

Our study does not confirm the hypothesis that poor cognitive outcome is due to bilateral hemispheric involvement or extensive white matter disease. Only larger cohorts of newborns with stroke followed for long periods of time will be able to answer this query.

D.M. Ferriero, MD, V. Ramaswamy, BSc, S.P. Miller, MD,
A.J. Barkovich, MD, J.C. Partridge, MD, MPH, *San Francisco, CA*

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Postictal paresis in focal epilepsies—Incidence, duration, and causes: A video-EEG monitoring study

To the Editor: Gallmetzer et al.¹ provided us with a thorough study of postictal paresis (PP) in patients with presurgical epilepsy evaluation.

After an introductory quote by Todd² including “limbs will remain paralytic for some hours,” they undertake a systematic analysis of PP. They show that the average duration of PP is 173 seconds with a maximum of 22 minutes. They also “found no differences regarding the incidence and the duration of PP depending on the location of the seizure onset zone” and that “a morphologic lesion does not seem to predispose to longer PP.”

While the results seem to hold for the study population, both the introduction and discussion suggest the results may be generalized to the average patient with a Todd’s paresis. Thus, a deficit lasting much longer than 22 minutes can hardly be a Todd’s paresis and location of lesion and seizure onset is unrelated to the occurrence and duration of a Todd’s paresis. This assumption, however, is not justified due to the lack of generalizability of the study results.

The typical patient seen by a neurologist with a Todd’s paresis will have clonic features followed by a paresis often in relation to an identified structural lesion such as an astrocytoma or ischemia in proximity to the motor cortex and with a duration sometimes greatly exceeding 22 minutes. The majority of these patients are unlikely to be referred to a tertiary referral center for presurgical epilepsy evaluation with video-EEG monitoring. In the study population: 16 of 35 patients had clonic features preceding the PP; only 18 patients of 44 had an extratemporal lesion (with no description of the spacial relation to the motor cortex); only 5 of these 18 had a tumor, while none had ischemia as the underlying lesion; and only 4 of 44 patients had an unequivocal extratemporal seizure onset zone.

While all these are typical features of patients with PP presenting to a neurologist, they are present only in a minority (or not at all) in the study population.

The study seems valid in reaching conclusions for the population presented, but a generalization to the overall population presenting to the neurologist with a Todd’s paresis is not warranted on the basis of information presented, let alone the classification of this paper as a review implying a general overview of the subject matter reflected in the title.

Patrick C.A.J. Vroomen, MD, PhD, *Groningen, The Netherlands*

Reply from the Authors: We thank Dr. Vroomen for his interest in our study.¹ We aimed to identify the typical clinical features of PP in patients with medically refractory epilepsy undergoing prolonged video-EEG monitoring. We also wanted to provide information on the clinical features of “pure” PP following epileptic seizures not confounded by other precipitating causes such as ischemia or tumor-related edema. Our patient population was therefore a selected one, which was also clearly stated.

Dr. Vroomen mentioned that the “typical” patient with a PP is a patient with an underlying structural lesion (tumor or cerebral ischemia) that has a relation to the motor cortex, that PP is often of longer duration and that patients with a PP typically do present with ictal clonic phenomena. This statement reflects Dr. Vroomen’s opinion and he does not cite published data. PP of longer duration was reported in patients with a history of stroke.³ In these patients, it may be difficult to distinguish a “pure epileptic” paresis from a “combined paresis” resulting from both ischemia and an epileptic cause.

PP was not confined to patients with a seizure onset in the motor cortex, which actually represents one of our main findings, but our study population was too small to investigate a relationship between a specific seizure-onset zone and the incidence of postictal paresis. Clonic phenomena were the most common seen motor phenomena in our patients, occurring in 55.6% of all seizures (n = 40) and in 65.9% of all patients (n = 29) at least during some seizures. The incidence of clonic phenomena in our patients confirms a recent study where tonic or clonic phenomena were also found in about three-quarters of the patients showing PP.⁴

P. Gallmetzer, MD, C. Baumgartner, MD, *Vienna, Austria*

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Mutation of the slow myosin heavy chain rod domain underlies hyaline body myopathy

To the Editor: Bohlega et al.¹ report the results of linkage and mutation analyses for a family with “hyaline body” myopathy. A heterozygous missense mutation, H1901L in slow/beta cardiac myosin (myosin heavy chain [MyHC] I, *MYH7*), was reported.

We previously described² another heterozygous missense mutation, R1845W, in the distal rod region of MyHC I, (*MYH7*) in a sporadic case as well as in a family with autosomal dominant inheritance. Since we found that the disease was caused by mutation in *MYH7* and its main morphological characteristic is large subsarcolemmal accumulation of myosin in type 1 muscle fibers, we proposed the name “myosin storage myopathy” (OMIM

608358) for this entity, which previously has been ascribed various terms in the literature including “hyaline body” myopathy. Interestingly, two additional sporadic cases with identical mutations as in ours are referred to in the recent third edition of *Myology*.³

Both mutations described are located at an “f” position of the heptad repeat of the coiled-coil structure of the MyHC rod that forms the backbone of the thick filaments of the sarcomeres. This confirms our hypothesis that “myosin storage myopathy” is caused by defective assembly of the thick filaments, since residues at “f” position of the coiled-coils may interact with other myosin dimers or with other proteins that are essential for the assembly of the thick filaments. This is also of interest in relation to familial hypertrophic cardiomyopathy, which in many cases is caused by mutations in *MYH7* and thus may be allelic to “Myosin Storage Myopathy.” Since patients with Myosin Storage Myopathy do not show cardiomyopathy there may be MyHC-interacting proteins, which are specific for skeletal muscle, and interact with the distal rod region of the MyHC.

In the article by Bohlega et al, the numbering of the mutated residue conflicts with the base substitution that was presented. According to Swiss-Prot (<http://ca.expasy.org/>), the mutated amino acid sequence number is H1901 and not H1904. The residue 1904 in human MyHC I is not at an “f” position in the heptad repeat sequence, whereas the amino acid at position 1901 is.

A. Oldfors, MD, PhD, H. Tajsharghi, MD, PhD,
L.E. Thornell, MD, PhD, *Göteborg, Sweden*

Reply from the Authors: We thank Dr. Oldfors et al. for their comments. We previously described a heterozygous mutation (an A to T transversion at nucleotide position 25596; M57965) resulting in a histidine to leucine amino acid change at residue 1904

(H1904L) in all affected members of a family with autosomal dominant hyaline body myopathy.¹ Numbering was based upon definition of an Assembly Competent Domain (residues 1874 to 1902).⁴

The H1904L mutation was adjacent to and not encompassed within this region. This same mutation would be cited as H1901L based upon Swiss-Prot (<http://ca.expasy.org/>). Aside from what numbering was chosen, the mutation does occupy an “f” position in the heptad repeat of the coiled-coil or tail domain of *MYH7*.⁵ This is interesting in light of another mutation of this domain occurring in the “f” position and producing essentially the same phenotype.^{2,3}

In contrast, mutations of the tail domain in other positions of the heptad have been associated with familial hypertrophic cardiomyopathy⁵ indicating allelic heterogeneity at this locus.

Brian F. Meyer, PhD, *Riyadh, Saudi Arabia*

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Seizure-alerting and -response behaviors in dogs living with epileptic children

To the Editor: There may be considerable reporting bias in the study on possible seizure-alerting behavior of dogs.¹ This is vindicated by the statement that there were “no false-positive behaviors reported,” which statistically would be highly unusual.

It is possible that some dogs may have directly induced the seizures. There is a long list of physiologic stimuli that can induce fits; and some of the behaviors described in the dogs could be responsible. Certainly a toddler being “forcibly” sat on by a dog might be subject to a variety of somatosensory and proprioceptive stimuli, including developing hyperventilation or becoming anoxic.

Peter J. Flegg, *Blackpool, UK*

Reply from the Authors: The reporting bias is unavoidable in this type of pilot project,¹ which is acknowledged in the discussion. Our family’s estimates of “sensitivity” are also clearly placed in their appropriate context, and do not represent a true lack of false-positives.

It seems highly unlikely and physically implausible to us that dogs would perform specific behaviors to produce seizures in their family members.

Adam Kirton, MD, *Calgary, Alberta, Canada*

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Stroke patterns, etiology, and prognosis in patients with diabetes mellitus

To the Editor: We read the article by Karapanayiotides et al.¹ with interest. Although the relationship between diabetes and stroke has been reported, the effect of diabetes as the cause of stroke is unclear. Hypertension is common in patients with diabetes and the combination of hyperglycemia and hypertension can increase the diabetic complications including stroke.²

In our stroke center, we evaluated 414 patients (204 male, 210 female, age range; 62.3 ± 11.5) admitted consecutively from January 2002 to January 2004. The ischemic stroke etiology deduced from our data is presented in the table. Unlike the article by Karapanayiotides et al., we found no etiological differences between diabetic and nondiabetic patients. Kiers et al.³ analyzed stroke topography in diabetics, and the patients were classified according to their glucose levels and type and site of stroke. They also found no differences between the groups. The stroke production mechanism secondary to diabetes may be due to cerebrovascular atherosclerosis, cardioembolism, or rheologic abnormalities.⁴

Similar etiologies can be seen in diabetic and nondiabetic pa-

tients. Schulz et al.⁵ proposed that there was a discrimination concerning the association of the risk factors and stroke subtypes in the hospitalized and nonhospitalized patients. Contrary to the hospital-based studies, there was only a weak and inconsistent association between small-vessel disease (SVD) and hypertension and no association with diabetes. Our study is hospital based and shows no etiological difference between the diabetic and nondiabetic individuals. The effect of diabetes on the etiology of ischemic stroke is still unclear.

D. Uluduz, MD, B. Ince, MD, M. Bozluolcay, MD, *Istanbul, Turkey*

To the Editor: We read the article by Karapanayiotides et al.¹ with interest. The authors reviewed clinical features, etiologic patterns, topography, and outcome of stroke in diabetic patients. In this study, clinical subtypes of ischemic stroke were rated according to the their own topographic criteria but perhaps Trial of ORG 10172 in Acute Stroke Treatment (TOAST) classification⁶ would have offered a more precise selection of patients with lacunar strokes.

Karapanayiotides et al. evaluated outcome indicators including

Table Etiologic distributions of patients

Etiology	Total, n = 414	Patients with diabetes, n = 108	Patients without diabetes, n = 306
Large-artery disease, n (%)	137 (33.1)	34 (31.5)	103 (33.7)
Small-vessel disease, n (%)	83 (20.0)	21 (19.4)	62 (20.2)
Cardioembolism, n (%)	108 (26.0)	28 (25.9)	80 (26.2)
Other determined, n (%)	25 (6.0)	7 (6.5)	18 (5.9)
Undetermined, n (%)	61 (14.9)	18 (16.7)	43 (14.0)

five-grade activities of the daily-living scale and six clinical features encoded in the Lausanne Stroke Registry (LSR) that offered a crude approximation of the NIH Stroke Scale. We believe that applying the Rankin score to evaluate disability grade and to assess acute neurological deficit by the Scandinavian Stroke Scale (SSS) could determine a better profile of patients with ischemic stroke by analyzing differences in disability and acute neurological deficit grades between diabetic and nondiabetic subjects.

Due to the high prevalence of hypertension in diabetic subjects (almost 60%), and to the confirmed role of high blood pressure in lacunar strokes pathogenesis,⁷ the absence of an interaction between diabetes and hypertension in patients with lacunar strokes is not easily conceived.

Our case-control study⁸ compared diabetic (102 patients) and nondiabetic controls (204 patients) matched by age and sex with acute ischemic stroke and evaluated the relationship between diabetes and stroke subtype. In our study, diabetes results associated with lacunar ischemic stroke subtype, with a history of hypertension and with a higher SSS score at admission. The association of diabetes with lacunar subtype remains significant also after adjustment for hypertension.

This suggests peculiar anatomopathologic findings of the diabetic cerebral infarction as showed by Peress and Kane⁹ that reported from autopsy reports a higher detected incidence of lacunar infarcts in diabetic subjects who died after an ischemic stroke. The higher SSS score of our diabetic patients converges with the higher frequency of lacunar stroke subtype among diabetics, because lacunar stroke could lead to a lower neurological deficit grade at admission due to brain infarct size.

A. Tuttolomondo, MD, A. Pinto, MD, D. Di Raimondo, MD, P. Fernandez, MD, G. Licata, MD, *Palermo, Italy*

Reply from the Authors: We thank Drs. Uluduz and Tuttolomondo et al. for their interest in our study.¹ We would like to clarify some points. First, the TOAST classification pertains to etiology and not to topography of stroke. The classification used in LSR is identical to the TOAST classification. In this regard, all patients with SVD represented “lacunes.” In our cohort, SVD and subcortical infarctions were more frequent in diabetics. Had we limited our analyses only to “classical” lacunes we should have missed the important association with the broader spectrum of subcortical infarction.

Second, the SSS and the Rankin scale were developed much later than the LSR. Third, the fact that hypertension and diabetes are strongly and independently associated with SVD does not obligatorily imply an interaction between them. The lack of interaction between hypertension and diabetes has also been documented in patients entering the North American Symptomatic Carotid Endarterectomy Trial.¹⁰

Fourth, a higher relative frequency of “lacunes” may be a reasonable explanation for a less severe neurologic deficit on admission in diabetic stroke sufferers.⁸ However, it would be interesting to see if this difference in stroke severity was translated into a better functional outcome.

The differences in risk factor profile and stroke subtypes between hospitalized and nonhospitalized patients probably do not apply to our population because LSR is essentially a community-based registry. Data from the State of Vaud (Lausanne urban and rural area) indicate that >90% of all stroke patients are admitted to our institution, which is the only public hospital in the area of Lausanne and has the only department of neurology in the entire county. Furthermore, in contrast with the UK where only about half of stroke patients are hospitalized,⁵ in Lausanne, general practitioners always refer patients with stroke. The hospital-based study mentioned³ by Uluduz et al. included only 176 stroke patients and was not powered enough to detect differences between diabetics and nondiabetics. In the study by Schulz et al.,⁵ SVD was associated only with females and not with “traditional” risk factors such as age and hypertension. This was a CT-based study that may have underestimated the burden of “lacunar” infarcts relative to MRI. An MRI population-based study¹¹ showed that except from the “classical” risk factors for SVD, (i.e. increased age and blood pressure), history of diabetes at entrance into the study was associated with SVD-related stroke. These results are in accordance with our study.

Theodoros Karapanayiotides, MD, *Athens, Greece*;
Gerald Devuyst, MD, Julien Bogousslavsky, MD, *Lausanne, Switzerland*

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Self-treatment of benign paroxysmal positional vertigo: Semont maneuver vs Epley procedure

To the Editor: I read with interest the editorial by Furman and Hain¹ concerning the home treatment of benign paroxysmal positional vertigo (BPPV). This is also reported by Radtke et al.^{2,3} The first two treatments were designed when the understanding of the mechanism of the disease was erroneous. The condition was known as cupulolithiasis, believed to result from adventitious deposits in the cupula of the posterior semicircular canal (PSC) changing the organ into a gravity sensor.

Following the observations of Parnes,⁴ it was accepted that the pathology of BPPV consisted of the presence in the fluid of the PSC of aggregated otoconia detached from the vestibular maculae, changing the hydrodynamics of the endolymphatic fluid inside the canal. The result is an abnormal function of the crista sensory organ, albeit with different characteristics than the predicted by cupulolithiasis, and more consistent with clinical observations whereby vertigo is produced by quick head rotations only in the plane of the affected PSC, a key in the diagnosis of the condition.

Epley's article,⁵ concerning the canalith repositioning procedure as a treatment for BPPV, provided strong evidence that particles could be extracted from the canal by rotating the patient's head in the geometrical plane of the canal, facilitating the particle's excursion towards the vestibule as propelled by the force of the gravitational vector upon the slug of heavier particles if executed within an appropriate time course. The success of the standard Epley maneuver for treatment of the PSC variety of BPPV has been reported as having an 88% success rate,⁶ and the new understanding of the pathophysiology has been shown to be theoretically correct.^{7,8}

Using the modified Epley maneuver, as proposed in Radtke's paper³ does not result in the removal of the particles from the canal. After one day of treatment, more than 80% of patients were still vertiginous (see figure 2),^{2,3} raising questions about the effectiveness of the treatment as a repositioning maneuver. What is it about the modified Epley maneuver that could account for the disparity? In the standard Epley maneuver (during steps 1 and 2), the head is hanging 30 to 40 degrees from the horizontal plane below the plane of a hard table surface where the patient rests during the test. This is to facilitate the relocation of particles toward the distal limb of the canal (see figures 9-2 and 9-3).⁹

During step 3, the particles pass the zenith of the canal curvature to enter the common crux and progress away toward the vestibule where they enter during the fourth step. As illustrated in the two papers by Radtke, the angle of head extension appears to be smaller than necessary based on the well-known anatomy of the canals. More importantly, in the unsupervised home exercises, the maneuver is subject to uncontrolled errors due to either the patient's incorrect execution (being vertiginous at the time) or to differences in accessory equipment (e.g., table, pillows.). The modified Epley maneuver obviously does not accomplish the removal of the particles from the canal as well as the standard Epley. Instead, the modified Epley represents no more than a variation of the Semont Liberatory Maneuver, a type of "shake and loose" treatment.

Radtke et al. should have attempted to cure the BPPV with the standard Epley when they gave the patient instructions on how to conduct the modified Epley procedure at home. It would have not taken any additional "costly and time consuming medical care." Patients treated with maneuvers other than the standard Epley are unknowingly subjected to unnecessary daily vertiginous distress that can be avoided by correct treatment.

The current understanding of the condition is that patients with BPPV of the posterior canal variety should be properly diagnosed, to avoid other coincidental vertigo provoking pathologies. They should be treated immediately with the standard Epley repositioning maneuver because it is noninvasive, quick and effective, and has minimal contraindications. Furthermore, if treatment is not successful, or there is a recurrence, the relapses do not necessarily take place in the same canal or with the same pathology and require other therapeutic procedures.⁶ A follow-up visit 1 week after the procedure reassures the patient or allows for additional treatment if needed.

There are many important questions yet to be answered about the cause of the majority of cases of BPPV. Is it due to aging,

degenerative, metabolic, or infectious causes? Almost half of the patients have been found to have unsuspected vestibular paresis of the horizontal semicircular canal or other inner ear maladies.⁹ Is it possible to diagnose the condition in the at-risk population before the attacks occur? What therapeutic steps can be taken to avoid their recurrence? There is very limited information about the etiology and treatment of other varieties of BPPV involving the other semicircular canals. Research has to move forward on the basis of clear understanding of the advances made so far. Holding onto old concepts is not the enlightened course to follow.

Endorsement of the Brandt-Daroff, Semont, or modified Epley maneuvers for unsupervised home treatment of BPPV is a disservice to patients and places them unnecessarily at risk for accidents.

Vicente Honrubia, MD, DMSc, *Los Angeles, CA*

Reply from the Authors: We thank Dr. Honrubia for his critical remarks on our paper³ on self-treatment of BPPV. We agree with most of his arguments but do not draw the same conclusions.

Cupulolithiasis is an outdated concept and current treatments should aim to clear the affected canal from mobile otoconia (canalolithiasis). Both the Epley and Semont maneuvers and their modifications for self-treatment work in accordance with the canalolithiasis concept even if the underlying principle may have been unknown to Semont when he developed his procedure.

The Semont maneuver has been found to be equally effective as the standard Epley procedure.^{10,11} The modified Epley maneuver is clearly superior to Brandt-Daroff exercises.¹² Therefore, it seems more appropriate to regard the approaches based on the Semont and the Epley maneuvers as canal-clearing procedures and not as a type of "shake and loose" treatment.

The efficacy of the self-applied Epley procedure can be compromised by imprecise execution including lesser angles of head reclination, which is critical for success. In addition, self-treatment with the Epley procedure appears to be less effective than the original physician-guided procedure, although no direct comparison has been undertaken at short follow-up intervals.

For our study, we tested the efficacy of self-treatment alone, but as we stated, we regard the standard Epley maneuver as the first-line procedure and propose self-treatment as a complementary approach. We agree that every BPPV patient entering a doctor's office should receive hands-on treatment and not only a sheet of paper. However, proper instruction for self-treatment may enable those patients who are not cured by a single course of a physician-guided Epley procedure, up to 30% according to several controlled studies,¹³⁻¹⁵ to free themselves of their BPPV before their next visit.

Patients with a recurrence of BPPV should schedule an appointment for positional testing, but no harm is done when they start self-treatment in the meantime, which offers them a good chance to be relieved from positional dizziness within a few days.

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