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Diffusion-Weighted Imaging II: Is There a New Gold Standard in Acute Stroke Imaging?

ABSTRACTS & COMMENTARY

Sources: Albers GW, et al. Yield of diffusion-weighted MRI for detection of potentially relevant findings in stroke patients. *Neurology* 2000;54:1562-1567; Lansberg MG, et al. Comparison of diffusion-weighted MRI and CT in acute stroke. *Neurology* 2000;54:1557-1561; Hacke W, Warach S. Diffusion-weighted MRI as an evolving standard of care in acute stroke. *Neurology* 2000;54:1548-1549; Powers WJ. Testing a test: A report card for DWI in acute stroke. *Neurology* 2000;54:1549-1551.

Ct scanning is the current standard of care for evaluation of acute ischemic stroke. The manuscripts by Lansberg and associates and Albers and colleagues with accompanying pro and con editorials by Hacke and Warach and Powers debate whether magnetic resonance imaging (MRI) with diffusion-weighted imaging (DWI) should supplant computerized tomography (CT) as the study of choice for acute stroke.

Lansberg et al studied 19 consecutive acute stroke patients, evaluated with both CT and DWI. DWI was more accurate for identifying acute infarction, showing evidence of acute infarction in all cases, compared with 42% or 63% on CT, depending on the reader. DWI was more sensitive for identification of major coronary artery (MCA) involvement of more than 33% (14% or 43% on CT compared to 57% or 86% for MRI, again depending on the reader). MCA involvement of more than 33% on CT is a key contraindication to intravenous thrombolysis as shown in the ECASS II trial. Lesion volume on DWI rather than CT was found to most closely correlate with the ultimate infarct size.

In a series of 40 patients, Albers et al describe the use of DWI in extending knowledge beyond that of a clinical impression and conventional MRI alone. Using DWI, Albers et al demonstrate that the clinical localization was incorrect in 30% of patients and identified the wrong vascular territory in 18% of cases. DWI may assist in determinations of stroke pathogenesis. Acute lesions in multiple vascular territories, implying an embolic etiology, were found in 13% of patients. DWI may also help differentiate between acute and chronic lesions. Twenty percent of lesions thought to be acute on

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conventional MRI were actually old.

In the "pro" editorial, Hacke and Warach interpret these data in favor of MRI over CT. As they observe, MRI with DWI/PWI and magnetic resonance angiography (MRA) is superior to achieve detailed anatomic localization, define the extent of acute stroke, determine a vascular anatomy, and draw informed conclusions about appropriate therapy. One concern is the MRI is not sufficient to rule out acute intracerebral hemorrhage prior to anticoagulation or thrombolysitc therapy but, according to Hacke and Warach, MRI is sufficient to distinguish between hyperacute intracerebral hemorrhage and ischemia.

In the "con" editorial, Powers notes that MRI techniques have not been subject to the kind of rigorous experimental design or validated outcomes data needed to accept MRI as a new imaging standard. As Powers observes, the Lansberg et al study is limited by "incorporation bias," using late DWI as a standard (the test itself) to validate the accuracy of early DWI imaging. Neither does Lansberg prove that DWI end points correlate with patient outcomes, the true measure of clinical benefit. Similarly, the Albers et al study does not demonstrate that the additional "clinical pearls" gained from DWI ultimately achieved diagnostic accuracy that could not be gained by other means: e.g., further clinical evaluation, follow-up imaging, or testing such as echocardiography or Doppler ultrasonography.

COMMENTARY

Initial interpretations of DWI data suggested that a diffusion abnormality was an all-or-nothing ischemic event. Data now indicate that these lesions may vary significantly in time, course, and extent. As the works of Li and Kidwell indicate, early DWI changes may or may not prove to correlate with irreversible ischemic damage. Resumption of blood flow at an early time interval after a stroke may allow resolution of DWI abnormalities seen in the hyperacute stage. DWI abnormalites may also transiently vanish and then reappear at a later time interval. This may be due to ongoing excitotoxic injury or late apoptosis. In addition, an ultimately normal DWI does not ensure normal tissue. Histological data suggest that, even with a normal DWI, neuronal damage may still exist.

DWI is a crucial extension of our diagnostic acumen, as the works of Lansberg et al and Albers et al clearly show. There is little doubt that it vastly improves sensitivity over CT. It is also probably sufficient to rule out hemorrhage prior to thrombolysis. However, as Powers argues, data from randomized trials incorporating DWI into acute stroke management are needed before a new gold standard can be declared. **—alan z. segal & ayeesha kamal** (*Dr. Kamal is Chief Resident of Neurology at New York Presbyterian Hospital.*)

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2. Kidwell CS, et al. Thrombolytic reversal of acute human cerebral ischemic injury shown by diffusion/perfusion magnetic resonance imaging. *Ann Neurol* 2000;47:462-469.

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Robot-Aided Stroke Stimulation

ABSTRACT & COMMENTARY

Source: Volpe BT, et al. A novel approach to stroke rehabilitation: Robot-aided sensorimotor stimulation. *Neurology* 2000;54:1938-1944.

Volpe and colleagues applied a scientifically developed robotic device that passively manipulated the rostral portion of the shoulder and connected elbow of patients recovering from moderate to severe stroke-

induced hemiplegia. Of 56 such patients, all receiving standard physiotherapy, 30 were randomly selected for robotic treatment and 26 served as controls. All patients started the program within two weeks following the paralytic onset. By chance, the 26 controls tended to have larger strokes and slightly greater degrees of paralysis. The robotic shoulder device was applied for one hour per day, five days a week at least five consecutive weeks in duration. Standard physical therapy was carried throughout for all patients. All patients could understand the procedure and respond to the mechanized therapy. Functional outcomes at onset and outcomes of completed robotic therapy were measured by motor assessment consisting of a Motor Status score, a Motor Power score, and Functional Independence Measurement. Clinical outcome was evaluated on all patients by a blinded, experienced physical therapist. Robotic therapy significantly increased power and coordination between shoulders and elbows, as well as total at functional activity of the whole arm-shoulder areas. Hand and wrist functions, however, failed to improve with the treatment. The relative duration of permanent improvement in the robot group vs. the controls has not yet been reported, nor has the present recovery of the improved member been described as accomplishing everyday needs.

COMMENTARY

This report reflects several years' efforts in prosthetic engineering aimed at finding ways to apply extracorporeal, interactive machines that would improve paralytic arm use following partial or severe stroke. The robotic instrument described here improved both shoulder strength and shoulder-elbow coordinated activity in poststroke patients. The lack of manual function, however, represents a considerable limitation to functional recovery. The results of this study differ greatly from the semipermanent success of "constraint induced movements (CIM)" reported by Miltner et al (*Stroke* 1999;30:586-592) and abstracted in the June *Neurology Alert* (6:82). The CIM report described remarkable recoveries of chronic hand-wrist dysfunction when applied two years after severe arm strokes. In that report, constraints were placed on the patients' normal hands and arms for an estimated 90% of wakeful time every day for two weeks. After that, the heretofore dysfunctional member returned to a relatively high level of skillful activity, which, when reviewed, continued for at least many months. Several questions appear. What is the durability of the robot-activating, applied increase in strength? How much more strength could be induced by longer daily robot exercises, and could peripheral stimulation to the hand evoke transynaptic, presently silent cerebral sensory-motor circuits to induce manual activity? Finally, was the overcoming of hemiparalytic manual function in the CIM program only a reflection of functional disuse rather than a reflection of cerebral plasticity? Answers to these questions will not come easily, but they amplify the increasing contributions of neuroscientific methods to the previously empirical discipline of neurological rehabilitation. **—fred plum**

Novel Phenomenal Experience

ABSTRACT & COMMENTARY

Source: Brugger P, et al. Beyond re-membering: Phantom sensations of congenitally absent limbs. *Proc Natl Acad Sci USA* 2000;97:6167-6172.

Every now and then your alert editor feels compelled to comment on a remarkable, novel neurological experience that brings new understanding about how the brain works, but for which no therapy is possible. Such follows.

Most neurologists have encountered the phenomenon of a phantom limb, an inner memory that remains in an amputee's mind following removal of an arm or leg due to trauma or medical disease. Only a few patients, however, have been recorded in whom severe prenatal losses of limbs have been associated with cerebral conceptualization that normal members remain. Thus this anecdote.

An intelligent, well-educated woman, age 44, was born with symmetrical near-absence of all four limbs. As an adult, her upper limbs measured about 25 cm long and consisted of bilateral conically shaped humeri inserted into normal shoulder sockets. The latter arrangement permitted full proximal arm movements. Rudimentary thighs measuring 10 cm extended from dysplastic hip articulations. Despite these limitations, she correctly uses many common, day-to-day objects, typewrites with her arm stumps, and writes with her mouth. For her entire life she has mentally perceived the presence of complete forearms and lower limbs. Only a direct gaze or a mirror provides her with awareness of her normally felt members. She vividly conceived positions of her absent hands or lower extremities when presented with appropriate tasks that would normally require specific, relatively fine manipulations or lower leg-foot activities. Physiological testing indicated normal distal reaction times in the limb stumps and explicitly copied the action times that would be present with normal manipulations. Functional magnetic resonance imaging (fMRI) identified manual task-engendered bilateral activation of dorsal and mesial premotor regions (supple-

mentary motor cortex) as well as superior posterior parietal cortical areas. Hand and finger representation areas in the primary motor cortex were silent. Self-perceived, conceptual activation of phantom flexion-extension foot movement activated normally placed, appropriate regions in contralateral premotor and parietal areas. Motor area 4 expressed only minimal activity on either side. Transcranial magnetic stimulation (TMS) evoked large areas of detectable deltoid muscle activity and also induced phantom movement sensations in the contralateral member after latencies of more than one second.

COMMENTARY

This is the first detailed scientific report to confirm in the brain the origins of phantom limb perception of abnormal cerebral physiological patterns associated with congenitally absent limbs. Several points are brought forth in the discussion. One is that persons experiencing phantoms following traumatic amputations and studied by fMRI retained physiological activity in the primary sensorimotor cortex. A second is that 19th-century studies reported atrophic cerebral gyruses associated with congenitally absent limbs, but fMRI failed to identify such an abnormality in this woman's fMRI. A third is that only a few cases of congenital aplasia appear to exist, none with satisfactory explanation. (A mini-epidemic of such developmental defects was associated with the drug thalidomide, an agent that appeared during the early 1960s. It seems possible that the drug might relate to this woman's tragedy.) Brugger and colleagues made the final, obvious point that phantom perception is not exclusively expressed by older children or adults suddenly deprived of one or more limbs. **—fred plum**

AD and NPH are Not Mutually Exclusive

ABSTRACT & COMMENTARY

Source: Golomb J, et al. Alzheimer's disease comorbidity in normal pressure hydrocephalus: Prevalence and shunt response. *J Neurol Neurosurg Psychiatry* 2000;68:778-781.

From among 117 patients who underwent shunt operation for clinically diagnosed normal pressure hydrocephalus (NPH), Golomb and colleagues selected 77 who demonstrated cognitive impairment on examination. Fifty-six of the NPH subjects and caregivers consented to excision a 5 by 2 mm cortical biopsy adjacent to the shunt penetration site. Biopsies were considered to be positive for Alzheimer's disease (AD) if even one neuritic plaque was identified by Bielschowsky silver-stained section. Global deterioration scale (GDS) ratings were obtained on all subjects and included evaluation of gait, praxis, memory, language, and executive functions. Videotaped assessment was rated by two independent viewers. The battery was administered preoperatively and an average of 4.3 months postoperatively.

Neuritic plaques were found in 23 biopsies. Twelve subjects showed diffuse plaques and six showed neurofibrillary tangles. The density of neuritic plaques was rated sparse in 10 cases and moderate in 13. Using CERAD criteria, Golomb et al diagnosed definite AD in seven patients, probable AD in nine, and possible AD in seven. As a group, patients with AD neuropathology were more cognitively impaired and exhibited greater gait dysfunction. There were no differences in age, gender, or in parameters such as frequency of incontinence between those found to have AD pathology and those who did not.

Many patients improved significantly after shunting, whether or not they demonstrated AD pathology. A small improvement in cognition was recorded for the AD-negative group (Z score increase of 0.29; $P < 0.01$), whereas the corresponding measure in the AD-positive group did not reach statistical significance (Z score increase of 0.23; $P < 0.06$) but showed a trend toward improvement.

Golomb et al concluded that AD may be a contributing factor in the dementia exhibited by patients diagnosed with NPH, and that patients with and without AD pathology may exhibit comparable responses to shunt placement. They recommend that patients who are deemed good candidates for shunt placement on clinical and radiographic grounds should not be denied surgery because of the suspicion that they also suffer from AD.

COMMENTARY

Regrettably, this article has serious faults. The incidence of shunt-related complications was not vigorously addressed. It is unclear whether the subset of patients who completed the testing accurately represented the entire cohort. The only hard data presented are based on a four-month evaluation of 37 of the original group of 56 patients providing biopsies. Furthermore, Golomb et al omit naming which statistical tests they applied to draw conclusions from such a small number of persons. The criteria used for diagnosing AD by tiny neocortical biopsy are particularly troubling, as Golomb et al admit. We have longitudinally evaluated one of the NPH patients who underwent shunt placement and biopsy at their site. Their records indicate that they were given a diagnosis of AD based on the biopsy results. The patient has shown no cognitive decline for 42 months since the shunt was placed. This speaks both to

the success of the VP shunt placement in that case and the inherent dangers of making a diagnosis of AD based on a tiny cortical biopsy.

We concur that NPH patients should not be categorically denied the option of surgical treatment just because of possible, coexisting AD. Nevertheless, other factors such as the relative risks of neurosurgery and the long-term effects of shunting must be considered before shunt placement in AD patients. Most important, we need better means of identifying true NPH cases and predicting shunt responsiveness, whether or not coexisting AD is present. **—norman r. relkin & fred plum**

Spinal Dynamics in Monomelic Amyotrophy

ABSTRACT & COMMENTARY

Source: Hirayama K, Tokumaru Y. Cervical dural sac and spinal cord in juvenile muscular atrophy of distal upper extremity. *Neurology* 2000;54:1922-1926.

Seventy-three japanese patients—68 men and five women—with juvenile onset (ages 11 to 19 years) muscular atrophy of the upper extremity (monomelic amyotrophy, MA) were compared to 20 disease-free controls (16 men, 4 women). The goal was to determine what effect, if any, movement of the cervical dural sac and spinal cord during neck flexion contributed to the pathogenesis of MA. All patients had typical clinical features of MA, including an insidious onset of weakness and atrophy in one (n = 59) or both (n = 14) arms. Signs and symptoms initially progressed, but spontaneously arrested after several years. Upper motor neuron, sensory, and sphincteric symptoms and signs remained absent. Diseased control patients suffered from cervical or lumbar spondylosis, spastic paraparesis, spinocerebellar degeneration, vascular malformation, syrinx, and peripheral neuropathy. All subjects underwent radiographic study between the ages of 12 and 52 years, including myelography, postmyelographic computerized tomography, and/or MRI of the cervical spine in full flexion with measurement of the anteroposterior diameter taken in multiple positions. For statistical analysis, younger (< 30 years at myelography) and older groups were separated and compared using student's t-test.

Striking dynamic changes of the position and diameter of the cervical dural sac on myelography were seen in 64 of 73 (88%) of both younger and older MA patients on neck flexion but not extension. Of 49 patients who underwent postmyelography CT in full neck flexion, 46 (94%) showed tightening and forward movement of the dural sac, with 40 (80%) displaying asymmetrical flattening of the spinal cord, even to boomerang shape, corresponding to the affected limb. MRI findings provided further confirmation of cord dynamics and flattening in 41 of 47 so studied (87%). None expressed any abnormal intrinsic cord signal. No controls showed cord flattening during neck flexion on CT myelography or MRI. MA may result from traumatic cervical myelopathy consequent to the activities of daily living (neck flexion).

COMMENTARY

Blessedly few MA patients come to autopsy, but one such patient demonstrated ischemic changes in C5 to T1 anterior horn cells supporting a vascular etiology (*J Neurol Neurosurg Psychiatry* 1987;50:285-290). MA has also developed in limbs following trauma and plaster cast immobilization (left elbow sprain and tibial tuberosity fracture in 1 patient each), possibly implying an association with the injury (*Muscle Nerve* 1997;20:425-430). Focal self-limiting motor neuron disease would be most consistent with the clinical syndrome but, in two brothers, one with amyotrophic lateral sclerosis and one with MA, the former expressed the superoxide dismutase 1 (SOD1) mutation whereas the latter did not (*Arch Neurol* 1997;54:46-50). Finally, a study of growth curves in seven MA adolescents correlated the age of onset with periods of most rapid height growth and radiographic evidence of disappearance in the slackness of dorsal roots. The phenomenon supports the notion that anatomical factors play a role in MA etiopathogenesis. **—michael rubin**

Early Onset Parkinson's Disease and Mutations in the Parkin Gene

ABSTRACT & COMMENTARY

Source: Lucking CB, et al. Association between early-onset Parkinson's disease and mutations in the parkin gene. *N Engl J Med* 2000;342:1560-1567.

The last five years have witnessed major advances in understanding the genetics of Parkinson's disease (PD). Four genetic loci have been linked to autosomal dominant PD and two genes have been discovered—a-synuclein and ubiquitin carboxy-terminal

hydrolase L1. Nevertheless, the vast majority of patients who present with PD do not have a family history of autosomal dominant inheritance.

Family members of patients with PD often ask the treating neurologist to comment on their risk of developing the disorder. Facts indicate such. An increased risk of PD among first-degree relatives of affected patients exists, and the risk increases with the number of diseased family members. Lack of an autosomal dominant inheritance, however, does not ensure that a patient does not have a genetic form of PD. Recently, mutations in a gene called parkin have been reported in several families with autosomal recessive PD. Affected persons have been reported in a variety of ethnic groups. Their clinical and pathologic profiles are similar to those of idiopathic PD, with dopa-responsive parkinsonism accompanied with selective loss of dopaminergic neurons in the substantia nigra and locus ceruleus. Unlike idiopathic PD, however, Lewy bodies are absent.

Lucking and associates report a colossal effort by a consortium of European investigators to determine the relative role of parkin mutations in patients with early PD. Seventy-three families with autosomal recessive levodopa-responsive PD with onset before the age of 45 were studied. Families originated from Italy, France, England, the Netherlands, Germany, Portugal, Spain, North Africa, and Vietnam. One hundred patients with sporadic PD beginning before age 45 were studied as controls. Patients were examined and had their blood drawn to screen for point mutations, deletions, and rearrangements in the parkin gene.

Mutations in the parkin gene were found in 36 of the 73 families with autosomal recessive PD. Among the 100 patients with sporadic early-onset PD, 18 had mutations in parkin. Compared to patients with sporadic PD, parkin patients had an earlier age of onset (32 vs 42 years) along with an equal male to female ratio, and more commonly presented with lower extremity dystonia, hyperreflexia, and symmetrical rigidity. They responded excellently to levodopa, but frequently developed dyskinesias within five years of starting the drug. While most patients with parkin mutations developed the disease in their early 30s, their age ranged widely from 7 to 58 years. Genetic analysis of the parkin gene revealed a high proportion of rearrangements in exons 2, 3, and 4, implying that these regions of the protein may be particularly important.

COMMENTARY

This study answers several important questions facing neurologists and their patients. The lack of a known family history of PD does not exclude the possibility that an individual may have genetic Parkinson's, especially when symptoms begin before the fourth decade. Among rare families with autosomal recessive parkinsonism, parkin mutations are surprisingly frequent. In the long run, they may be responsible for the majority of these cases. Patients with parkin mutations are too similar to idiopathic PD patients to be distinguished on clinical grounds alone, although the presence of hyperreflexia or lower extremity dystonia may help. Finally, patients who are diagnosed with parkin mutations can be reassured that their illness, while progressive, usually proceeds extremely slowly and is responsive to treatment with levodopa. **—steven frucht**

Venous Insufficiency and Polyneuropathy

ABSTRACT & COMMENTARY

Source: Reinhardt F, et al. Peripheral neuropathy in chronic venous insufficiency. *Muscle Nerve* 2000;23:883-887.

Thirty chronic venous insufficiency (cvi) patients (mean age, 55.1 years) were compared to 20 age-matched normal controls to determine whether CVI was associated with peripheral neuropathy. Diagnosis of CVI was (apparently) based on clinical findings of venous dilation, edema, pigmentary skin changes, or ulceration, plus abnormal Doppler or duplex sonography of the peripheral venous system. Exclusionary criteria included other causes for neuropathy such as diabetes, vitamin deficiency or excess, vasculitis, Lyme disease, Sjogren's syndrome, dysproteinemia, and exposure to neurotoxic agents. All patients underwent neurological examination and compression therapy to relieve edema prior to electrodiagnostic studies. Large-diameter myelinated A-alpha fibers were studied using standard peroneal motor nerve conduction study. A-beta fibers were evaluated by sural sensory nerve conduction study and vibration threshold testing using an electromagnetic vibratester (Vibratester 100, Gottingen, Germany). Warm and cold detection thresholds using the Somedic Thermotest Type I (Stockholm, Sweden) examined thinly myelinated A-delta and unmyelinated C fibers, respectively. Laser Doppler flowmetry determined peripheral vasomotor C fiber function. Autonomic testing using the quantitative sudomotor axon-reflex test (QSART) studied postganglionic unmyelinated C fiber function. Heart rate variability testing excluded systemic autonomic dysfunction. Statistical analysis was per-

formed using the Mann-Whitney U-test with the Bonferroni correction.

Approximately one-third of CVI patients demonstrated signs of neuropathy on examination, with vibration threshold and warm and cold perception significantly reduced compared to controls. Sural nerve recordings, and sudomotor (QSART) and vasomotor (Doppler flowmetry) function were normal in all with only peroneal distal motor latency prolongation on nerve conduction study. A-alpha, A-beta, A-delta, and thermoafferent C fibers are abnormal in CVI, possibly due to ischemia, and may contribute to the development of stasis ulcers.

COMMENTARY

Although this paper does not clearly indicate either how CVI or peripheral neuropathy were diagnosed, nor exactly how many patients had peripheral neuropathy, nevertheless, the association with CVI appears convincing and correlates with the clinical experience of this reviewer. Should a therapeutic clinical trial be initiated using nerve growth factor for stasis ulcers? **—michael rubin**

Epilepsy—Stereotypes, Misinformation, and Quality of Life

ABSTRACTS & COMMENTARY

Sources: Krauss GL, et al. "The scarlet E"—The presentation of epilepsy in the English language print media. *Neurology* 2000;54:1894-1898; Long L, et al. An assessment of epilepsy patients' knowledge of their disorder. *Epilepsia* 2000; 41:727-731; Markand ON, et al. Health-related quality of life outcome in medically refractory epilepsy treated with anterior temporal lobectomy. *Epilepsia* 2000;41:749-759.

Despite decades of remarkable progress in better understanding and treating epilepsy, the disease remains mysterious and frightening to much of the public and many patients. Krauss and colleagues searched 2000 English-language newspapers and popular magazines and found 210 articles that emphasized aspects of epilepsy. Epileptologists reviewed the article for accuracy and categorized inaccuracies. The reviewers also evaluated the articles for thematic content and major themes. Thirty-one percent of the articles contained a major inaccuracy, the most frequent being scientific inaccuracies, exaggeration of treatment benefits, and exaggeration of risks of seizures. Errors were more likely in reports of new drug therapies (52%). More than one-third of stories that reported nonpharmaceutical treatments or described patients' daily living also contained major inaccuracies. Six percent of stories described demonic or supernatural imagery. Both medical and nonmedical journalists stated inaccuracies. Medical reporters were more likely to overstate the benefits of novel treatments or research, whereas nonmedical specialists tended to overstate the risks of seizures or stigmatism in patients.

Long and associates identified inaccuracies in knowledge about epilepsy equal to the above journalists' reports. Long et al developed a survey in order to develop educational resources to patients with epilepsy. The test used multiple-choice and true-false questions conducted among patients referred to a comprehensive epilepsy program at a tertiary care center. Patients subsequently found to have nonepileptic seizures or diseases were excluded. Of the analyzed group, 29 incorrectly answered the question, "What is epilepsy," and attributed the illness to either "a mental disorder" or a contagious disease. Forty-one percent of patients believed an object should be placed in the mouth to prevent tongue-swallowing during a seizure. Twenty-five percent thought pregnant women should discontinue antiepileptic medications to avoid teratogenic effects on the fetus. Twenty-five percent of patients answered that operating a motor vehicle was appropriate if they either "doubled-up on medication," did not drive alone, or "pulled over" at seizure onset. Fifty-two percent were unaware that employers are prohibited from inquiring whether potential employees have epilepsy. Age and education only weakly correlated with likelihood of correct answers. No linkage was found between the number of years with epilepsy and the likelihood of correct answers.

The increasing emphasis to measure quality of life in assessing outcome of epilepsy reflects an important new emphasis on treatment. Markand and colleagues prospectively examined the change in quality of life resulting from anterior temporal lobectomy for medically refractory epilepsy and found a divergence between improved seizure control and improvements in quality of life. Markand et al used a presurgical interviewer-administered survey to assess quality of life in 90 patients referred for refractory partial seizures. Fifty-three patients received temporal lobectomy; the remainder were either unsuitable for surgery or declined it. The study compared presurgical survey results with results obtained one and two years after surgery or nonsurgery. The patients who received surgery enjoyed a significant improvement in quality of life. Further analysis, however, showed that, of all groups, only the patients who became completely seizure-free following surgery

enjoyed an improved future quality of life.

COMMENTARY

Long et al's study is limited by using a nonvalidated survey, and it is possible that some of the terms used (e.g., "mental," to describe nonorganic disease) may have been misinterpreted by patients. Nevertheless, the results highlight the gap between a patient's understanding of the medical and social implications of epilepsy, and a physician's concern for achieving relief from seizures. Krauss et al depended on print media, and it seems likely that television reporting would reveal similar or greater inaccuracies. The findings reveal an ongoing need for patient education and increased dissemination of proper information to the public. Reliable information can be obtained on the Web sites of the Epilepsy Foundation or the American Epilepsy Society, but all too often other avenues distribute a background of misinformation. The greatest opportunity to educate patients comes from the office visit. Markand et al address an important question: "Which patients benefit from temporal lobectomy?" The result that patients who become seizure-free, but still experienced auras, had a quality of life similar to those who did not receive surgery bears further attention in presurgical candidates. **—fred a. lado**

CME Questions

4. Etiologic possibilities for monomelic amyotrophy include:
a. an inflammatory etiology.
b. head trauma.
c. superoxide dismutase mutation.
d. cervical cord trauma against the vertebral column from repeated neck flexion.
e. All of the above

5. Patients with NPH and AD neuropathology on cortical biopsy:
a. do not respond to shunt placement.
b. have definite AD.
c. should not be considered surgical candidates.
d. would be expected to progressively worsen over time.

6. Chronic venous insufficiency is associated with abnormalities in:
a. A-alpha fibers.
b. A-beta fibers.
c. A-delta fibers.
d. All of the above

7. Inaccuracies about epilepsy were most often present in which of the media stories?
a. Reports from medical journalists
b. Reports from nonmedical journalists
c. Reports describing new drug treatments for epilepsy
d. Reports describing new nondrug treatments for epilepsy

8. A woman born with stumps for arms and legs:
a. senses with her brain that she has normal arms and legs.
b. manages to typewrite and carry out most daily necessities.
c. had an fMRI that showed absent activity in cerebral area 4 when she voluntarily moved her arm.
d. All of the above
e. None of the above

9. All of the following statements regarding diffusion-weighted imaging (DWI) are true *except*:
a. DWI is superior to standard MRI in distinguishing acute from chronic lesions.
b. DWI lesions in multiple vascular territories may suggest an embolic etiology for stroke.
c. DWI lesions are potentially reversible with thrombolysis.
d. disappearance of a DWI lesion guarantees an entirely normal histologic outcome.
e. DWI is probably sufficient to rule out hyperacute intracerebral hemorrhage.

10. The robotic instrument described by Volpe et al improved:
a. shoulder strength.
b. shoulder-elbow coordinated activity.
c. hand and wrist functions.
d. a and b.
e. None of the above

11. Comparing robot-aided stroke stimulation to CIM:
a. CIM measurably improved even totally paralyzed contralateral hands two years after a stroke.
b. CIM required one hour per day to improve some existing contralateral hand function.
c. robot-aided rehabilitation improved stroke-generated shoulder and arm activity but not hand function.
d. robot-aided rehabilitation extended to three months eventually evoked contralateral hand function.

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