Survey of training programs' means for promoting neurology and attracting trainees

To the Editor: In their recent article, Adair et al. note some of the defining factors influencing program choice in neurology training opportunities.[1] Their not-too-subtle conclusion is that many neurology training programs, and by extension the entire specialty of neurology, may be in for a rough ride: "available (residency) positions continue to exceed the number of American medical school graduates interested in neurology."

Throughout the article's ensuing "wherefores and whys," the proffered explanations seem to circumvent, and leave largely unexplored, one of the most obvious means to increase interest in neurology training programs and the entire field of neurologic science in general: neurologists' active involvement with neuroimaging.

As many neurologists now find themselves with fewer economic opportunities relative to other, perhaps more procedurally oriented, medical subspecialties (as with cardiology), it seems that neuroimaging is the veritable "elephant in the room."

After years of ambivalence with regard to neuroimaging, the neurologic community has recently begun to review practice domain imperatives and priorities. Neurologist involvement with neuroimaging has been an active topic within the American Society of Neuroimaging for many years, and it is now seriously considered by many in the American Academy of Neurology (AAN), both rank and file and academic members.

With the recent addition of Neuroimaging into the United Council of Neurologic Subspecialties (UCNS), the entire concept of neurologist neuroimaging now deserves re-examination, updating, and, hopefully, emphasis at the AAN Board of Directors level.

Some practicing neurologists remember Bill Oldendorf's cautionary advice to neurologists some 20 years ago, urging neurologists' active involvement ("on the ground floor") with the "new magnetic imaging technology that has absolutely nothing to do with Roentgen."

Somewhere along the path, neurologists dropped the neuroimaging ball (or was the ball volitionally "thrown down," as some would suggest?), so that many neurologists have unwittingly deferred the right and privilege of interpreting (usually their own referrals) neuroimaging studies to other specialties with less clinical knowledge, and often with less interpreting expertise.

Neurological advocacy, at both the resident training and practitioner levels, and promotion of clinical "practice domain" policy start at the top: until neuroimaging is officially sanctioned by the AAN as a rightful extension of the neurologist's purview, with appropriate educational ramifications at the residency training and CME levels, little will change, and a golden opportunity for the specialty will be lost.

William G. Preston, MD, FAAN, *Laguna Hills, CA*

Disclosure: The author reports no conflicts of interest.

Reply from the Authors: The Graduate Education Subcommittee (GES) of the AAN Education Committee appreciates Dr. Preston's insights about our survey. His remarks raise an important subject not addressed directly in our report.

To begin framing the issue of how to match "the best and brightest," the GES sought to determine how departments introduce our specialty to medical students. Program features that influence current residents' (i.e., former students') judgments about training site selection provided complementary information about match success. A more fundamental question about what motivates choosing a career in clinical neuroscience, broached by Dr. Preston and reviewers, awaits more ambitious investigation.

Dr. Preston suggests reasonably that "active involvement" in neuroimaging might provide a means of generating interest in neurology. The recommendation appeals to common sense on several levels. Fascination with neuroanatomy and its application to clinical problems often impels students to cultivating further interest in the organ system. Imaging technology now resolves structures in detail rivaling or superior to inspection of gross specimens in the anatomy laboratory. The intellectual satisfaction of solving clinical conundrums, through integrating clinical observation with anatomic and physiologic studies, provides a major impetus for pursuing the clinical neurosciences. However, Dr. Preston's letter identifies a more pragmatic motivation: neuroimaging offers the potential of enhancing revenue for the practicing neurologist, not to mention the academic department. Medical curricula pay little formal heed to economic aspects of return for services rendered and the fiscal realities of practice survival.

Undoubtedly, many students develop some hunch about income differential between specialties, and it would be surprising if future surveys fail to find that such considerations play at least some role in deciding about a career in neurology.

The most appealing of Dr. Preston's comments concerns whether our specialty can "do well by doing good." In contrast to many specialties, most neurologists view imaging studies they order, evaluating both technical quality and judging the relevance of findings. As mentioned, the neurologist may have more experience interpreting neuroimaging than the radiologist. And while both share the responsibility for a study's disposition, only the radiologist receives financial return.

Efforts on behalf of neurology by the American Society of Neuroimaging and UCNS have resulted in means through which trainees may acquire certification for proficiency in neuroimaging. However, substantial additional efforts will be required for regulatory approval of neurologists treading on radiologists' turf. Such advocacy would be facilitated if neurology can demonstrate comparable or superior outcomes for our "active involvement."

John C. Adair, *Albuquerque, NM*; John R. Corboy, *Denver, CO*

Disclosure: The authors report no conflicts of interest.

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Subarachnoid hemorrhage is followed by temporomesial volume loss: MRI volumetric study

To the Editor: Bendel et al. reported an interesting study on the volume loss of temporomesial structures after aneurysmal subarachnoid hemorrhage (SAH).[1] The authors emphasize that amygdaloid atrophy was more prominent in patients treated with open surgery and provided evidence indicating that this finding may relate to retraction injury. However, hippocampal but not amygdaloid volumes correlated with deficits on neuropsychological tests. Furthermore, hippocampal volumes were unusually associated with intellectual functions other than memory.

Considering these results, perhaps the authors could assess if the results of neuropsychological tests were associated with radiologically documented lesions in other brain structures, particularly the frontal white matter. This question is pertinent because damage to subcortical white matter in the frontal lobes is known to occur often after SAH[2] and the neurocognitive deficits noted by the authors are similar to those expected with frontal lesions.

Alejandro A. Rabinstein, *Rochester, MN*

Disclosure: The author reports no conflicts of interest.

Reply from the Authors: We appreciate the interest of Dr. Rabinstein in our article.[1] He emphasizes the importance of radiologically documented lesions in brain structures other than temporomesial. Recent studies show that frontal high signal intensity lesions are often detected in T2-weighted images after SAH and the repair of the ruptured aneurysm.[2,3] Furthermore,

frontal lobe lesions are associated with neuropsychological impairments after SAH.[4]

We concluded that temporomesial volume loss is frequently detected on MRI 1 year after SAH.[1] In our volumetric study population, we reported parenchymal high signal intensity lesions in 67.5% of the patients with SAH and these lesions were detected in the frontal lobes in 59.7% of the patients. The presence of a frontal high signal intensity lesion in the T2-weighted images was not significantly associated with the hippocampal volumes measured. As expected, the presence of a lesion in the frontal lobes was associated with significantly worse results on the neuropsychological tests.

We have further analyzed the correlations between the neuropsychological test results and hippocampal volumes separately in patients with and without a frontal lesion. We found significant correlations between impaired neuropsychological test results and reduced hippocampal volumes in both subgroups of the patients with SAH, suggesting that the detected hippocampal volume loss seems to be associated with impaired neuropsychological performance regardless of a possible coexisting MRI-detectable lesion in frontal lobes.

Those correlations were seen in a larger number of the tests applied in patients with a frontal lobe lesion, indicating that the effect of the hippocampal volume loss is probably more pronounced in patients with a documented frontal lobe lesion. Although our recent article was focusing on the reduced temporomesial volumes after SAH, many patients also have other radiologically documented lesions on brain MRI. We assume that both the temporomesial volume loss and other local parenchymal lesions together contribute to the development of neurocognitive deficits which are often detected in patients after SAH.

Paula Bendel, Timo Koivisto, Ritva Vanninen, *Kuopio, Finland*

Disclosure: The authors report no conflicts of interest.

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Speech and language delay are early manifestations of juvenile-onset Huntington disease

To the Editor: Yoon et al.[1] make a worthwhile effort to specify the functional pathogenicity of a well-described disease.[1] As it stands, the report creates the impression of a specific vulnerability of speech and language to the Huntington disease process; a conclusion not supported by the supplied data.

Evidence of lesser involvement of certain nonverbal cognitive processes is necessary. Of the few such tests reported (design copying, matrix reasoning) none of the scores approaches normal, nor is any history of normal nonverbal development given for any of the three cases.

Considering the format of the report, it may be a work in progress. The authors should be commended and encouraged to further determine specific cognitive pathology.

Peter B. Rosenberger, MD, *Boston, MA*

Disclosure: The author reports no conflicts of interest.

Reply from the Authors: We thank Dr. Rosenberger for his thoughtful comments. While we speculated that the speech and language delay manifested in patients with juvenile Huntington disease may be due to the disease process itself, it was not our intent to imply a specific vulnerability of language function compared to other aspects of cognition.

We agree that more detailed cognitive testing in a larger study population would be required to demonstrate such a specific vulnerability. The purpose of the article was to raise awareness of speech and language delay as signs/symptoms that predate motor involvement, which has traditionally been used to define onset of disease in these patients.

This information may be used, in addition to family history and with appropriate genetic counseling, to consider the possibility of a diagnosis of juvenile Huntington disease in a child at risk.

Adam L. Boxer, Grace Yoon, *San Francisco, CA*

Disclosure: The authors report no conflicts of interest.

Reference

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Atypical antipsychotics in the elderly with Parkinson disease and the "black box" warning

To the Editor: The black box warning concerns increased mortality after use of atypical antipsychotic (AA) drugs in elderly demented patients with psychosis.[1] Such warning will likely result in changing use of the medications. Dr. Friedman emphasizes that it might be a disservice to our patients to withhold AAs.

The explanation of the increased mortality remains unclear and the clinician is left with no guidelines for the future use of the AAs as well as the older antipsychotic drugs. Inherent pharmacologic properties of the drugs may not be the sole explanation. It is possible that the drugs sometimes are used inappropriately due to misdiagnosis of worsened delirious encephalopathy. Thus, the clinician may have failed to recognize presence of hypoxia, cardiac ischemia, or infections.

In the absence of more obvious signs of acute illness in demented patients, the AA drug is prescribed for exacerbated agitation or hallucinations. Some patients may be particularly susceptible for misdiagnosis and ultimately death, including the very old, the severely demented, those admitted for acute or chronic care, and those with multiple coexisting medical problems.

Until further studies are available and more authoritative guidelines provided, these medications probably can be used without the warning of death as a side effect but with an understanding among the caretakers of the need for reevaluation of the confused patient.

Underlying acute medical problems should be explored in the demented patient with altered mental status. Use of AAs should be reserved for those instances when acute medical illness appears unlikely after careful clinical re-assessment.

Carl E. Rosenkilde, MD, PhD, *Mount Kisco, NY*

Disclosure: C.E.R. has served as a certified PANSS trainer for the PANSS Institute, LLC.

Reply from the Author: I share Dr. Rosenkilde's concerns about the potential underuse of atypical antipsychotics in the elderly demented. I do not think we can explain away the increased mortality by invoking misdiagnosis or increased medical problems in this population because the "black box" warning was based on placebo-controlled trials.

Unless we find fault with the individual trials we must assess the causes of increased death rates. It is possible that the diagnoses used for entering the studies were incorrect but I do not think we can ignore a study because of unfounded suspicions,

especially when the results are supported by several studies involving different drugs.

On the other hand, I do believe that Dr. Rosenkilde is generally correct in his analysis. My own hypothesis, which is probably the most common one, is that the side effects of sedation, orthostatic hypotension, and worsened (often unrecognized) parkinsonism are the culprits. The questions are not so much whether these drugs increase mortality, but whether alternatives such as benzodiazepines, first-generation antipsychotics, or cholinesterase inhibitors are any safer, and how they compare on measures of efficacy and quality of life.

Joseph H. Friedman, *Warwick, RI*

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Occurrence of CNS demyelinating disease in patients with myasthenia gravis

To the Editor: We read with interest the article by Gotkine et al., who suggest an association between myasthenia gravis (MG), CNS-specific demyelinating disease (CNSDD), and a forme fruste of systemic lupus erythematosus (SLE) in a series of five patients.[1] We suggest a more specific neurologic diagnosis for these patients.

Patients 1, 3, and 5 had MG, antinuclear antibodies (ANA), and subsequently presented with myelitis or recurrent myelitis. Patient 1, a 28-year-old woman, had two episodes of longitudinally extensive transverse myelitis (LETM, initially from C4 to C6; subsequently from C2 to T10) with edema and enhancement. The brain MRI was normal. This description is more consistent with the newly recognized neuromyelitis optica (NMO) spectrum of disorders.[2]

NMO (also known as optic-spinal multiple sclerosis or Devic's disease) is an idiopathic inflammatory demyelinating disorder of the CNS characterized by LETM and optic neuritis (ON), and usually has a relapsing course. Mixed CSF pleocytosis, as in this case, occasionally with neutrophil predominance, absence of oligoclonal bands, and lack of brain lesions on MRI, is typical. Relapsing ON or relapsing myelitis often represent limited forms of the NMO phenotype (NMO spectrum disorders) and a high proportion of these cases are seropositive for the NMO-specific autoantibody, NMO-IgG, which is directed at the astrocytic water channel protein aquaporin-4.[3] The sensitivity of NMO-IgG for the diagnosis of NMO is 73% and specificity 90%. Its sensitivity for relapsing LETM is 52%; for first event LETM, 40%.[2,3] In a prospective study of patients who experienced a first event LETM, NMO-IgG seropositivity predicted with greater than 50% certainty recurrence of LETM or development of ON within 1 year.[2] NMO is recognized to be associated with other autoimmune disorders in up to 40% of cases. Its association with MG has been documented in several reports.[4]

None of the patients in this report seem to satisfy the ARA criteria for SLE. ANA is non-organ-specific autoantibody associated with a variety of immune reactions. Our group recently reported that patients with NMO spectrum disorders who are seropositive for NMO-IgG have a higher rate of seropositivity for ANA and SSA/SSB antibodies than those who are seronegative for NMO-IgG.[5] We suggest that the diagnosis suggested by Gotkine et al. of a CNSDD occurring with MG and a forme fruste of SLE is most likely an NMO spectrum disorder. Testing for NMO-IgG would be helpful for diagnosis and prognosis.

Brian G. Weinshenker, Anu Jacob, *Rochester, MN*

Disclosure: Dr. Weinshenker will receive royalties related to a patent for NMO-IgG held by Mayo Medical Ventures. Dr. Jacob reports no conflicts of interest.

To the Editor: We read with interest the article by Gotkine et al.[1] concerning occurrence of CNSDD and MG. We also encountered one patient with recurrent myelitis among 325 patients with MG.

A 53-year-old woman was diagnosed with MG at age 29 years. Thymectomy was performed at age 30 years and the pathologic finding revealed hyperplasia. After she was treated with pyridostigmine and prednisolone until age 42 years, MG was remitted completely without medication. Three years later, she developed weakness and dysesthesia in the upper limbs. Cervical MRI showed T2-hyperintensity signal areas in the C2–6 segments with enhancement. Methylprednisolone treatment improved motor and sensory deficits without sequences. She experienced paresthesia ascending from the lower limbs to body and upper limbs and painful tonic seizures in the upper limbs at age 51. Neurologic examination showed paresthesia and hyperreflexia in the four extremities and Lhermitte sign was presented.

Immunologic analyses showed negative acetylcholine receptor antibodies and increased ANA. Brain MRI was normal. Spinal MRI disclosed T2-hyperintensity signal areas in the C3-T1 segments with ring-shaped enhancement. On visual evoked potentials (VEP), P100 latency was delayed (115 msec in the right and 118 msec in the left). Two years later, optic neuritis occurred. The final diagnosis of NMO was made.

According to recent diagnostic criteria for NMO,[6] the features of spinal MRI reveal longitudinally extensive cord lesions more than three segments in our and four patients of Gotkine et al.[1] Those pathognomic distributions of spinal lesions suggest a possibility of NMO. Gotkine et al.[1] describe prolonged VEP in Patient 4. We wonder whether VEP were performed in Patients 1, 3, and 5 and whether VEP were performed in the other four patients. We would like to know serum NMO-IgG antibodies in Patients 1 and 5 who have no lesions on brain MRI. How strong is the possibility of NMO in those patients? NMO is associated in patients with MG after thymectomy.[4,7] Besides NMO, recurrent myelitis without ON could contribute to common immunologic mechanism as a subgroup of CNSDD in thymectomized patients with MG.

Ken Ikeda, Yo Araki, Yasuo Iwasaki, *Tokyo, Japan*

Disclosure: The authors report no conflicts of interest.

To the Editor: Gotkine et al.[1] describe three patients with MG who underwent thymectomy and subsequently developed either recurrent or monophasic acute myelitis. The authors suggest that these patients may have a form fruste of SLE, given their elevated ANA titers, though they have not exhibited any non-neurologic manifestations of SLE and had no anti-DS DNA Ab in serum.

Alternatively, we would like to propose that the three patients may have form fruste of NMO. ANA positivity is not an infrequent finding in NMO[8] and would not preclude this diagnosis. All three patients had extensile cord lesions three or more vertebral segments in length, which are highly characteristic of NMO; absence of MS-like plaques in brain and oligoclonal bands in CSF is also suggestive of this disease. It would be interesting to examine the patients for stigmata of past attacks of ON on neuro-ophthalmologic examination and on VEP, as well as to test their NMO IgG status.

We recently reported four patients with MG who underwent thymectomy and developed clinically definite NMO; two of our four patients had an elevation in ANA titers.[4] Concurrently, Furukawa et al. published two cases of MG followed by NMO: one patient had thymectomy prior to NMO onset, while the other has been previously diagnosed with SLE.[7]

There are presently no fewer than 13 reports in the literature of thymectomized myasthenic patients developing NMO or recurrent acute myelitis.[4,7,9] In all these cases, MG was diagnosed prior to the first CNS event. Moreover, in all but one patient with definite MG and NMO, thymectomy preceded NMO onset.

We agree with the conjecture of Gotkine et al. that thymectomy may lead to breakdown of self-tolerance thereby potentiating development of autoimmune disease. A systematic analysis of long-term effects of thymectomy is needed, using non-

thymectomized myasthenic patients as controls, to assess whether thymectomy indeed confers an increased risk of future autoimmune disease and the magnitude of the excess risk. It may also be worthwhile to investigate whether thymectomy predisposes to development of NMO IgG autoantibodies even in the absence of clinical manifestations of NMO.

I. Kister, J. Herbert, M.L. Swerdlow, R. Bergamaschi, G. Piccolo, J. Oger, *New York, NY*

Disclosure: The authors report no conflicts of interest.

Reply from the Authors: We thank the authors for raising some important questions which reiterate the importance of the discovery by Dr. Weinshenker's group of NMO-IgG and its value in defining patients with a distinct spectrum of diseases.

We agree with Drs. Weinshenker and Jacob, Ikeda et al., and Kister et al. that the extensive longitudinal involvement of the spinal cord with cord swelling is characteristic of the type of myelitis occurring in patients with NMO-IgG. We also would point out that this type of spinal cord lesion also occurs in patients with SLE.[10]

As SLE is a syndrome with clinical criteria we once again emphasize that none of our patients had SLE (hence we defined the syndrome as a forme fruste) although we stand by our assertion that the neurologic involvement observed in some of our patients may be due to pathogenetic mechanisms which also occur in SLE.[1]

Ikeda et al. make a valid point regarding VEP. Although not fully detailed in our original article, these were normal in Patient 1 and abnormal in Patients 3 and 4. Patient 5 had not had a VEP test and was unavailable for further testing.

We are impressed by the close resemblance of clinical and laboratory features between the patient of Dr. Ikeda et al. and our Patient 1. The most noteworthy difference is the clinical and electrophysiologic evidence of optic nerve dysfunction in the patient of Dr. Ikeda et al. whereas our Patient 1 has never had visual symptoms and had normal VEP bilaterally. NMO-IgG has been reported in patients with MG with classic NMO[4] but has not yet been documented in patients with MG and isolated myelitis. Nevertheless, prior to the publication of our article we sent serum from Patient 1 (relapsing longitudinal myelitis) and Patient 3 (relapsing localized myelitis) for NMO-IgG testing. Not surprisingly, Patient 1 (the patient discussed by Weinshenker and Jacob and Ikeda et al.) but not Patient 3 tested positive for NMO-IgG.

Given that NMO occurs in patients with SLE[11] it is certainly conceivable that the myelitis of SLE is closely related, if not part of the NMO spectrum. As a corollary we hypothesize that systematic investigation of SLE patients with longitudinal myelitis will reveal a high proportion of patients with NMO-IgG.

Marc Gotkine, Oded Abramsky, *Jerusalem, Israel*

Disclosure: The authors report no conflicts of interest.

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Correction

How affected is oxygen metabolism in DWI lesions? A combined acute stroke PET-MR study

In the article "How affected is oxygen metabolism in DWI lesions? A combined acute stroke PET-MR study" by J.V. Guadagno et al. (*Neurology* 2006;67:824–829), there is an error in figure 1. The images are shown in the "neurologic convention", i.e., the left side of the brain is shown on the left-hand side of the images. However, for Patient 5 the images are show in the "radiologic convention," i.e., the left side of brain is on the right-hand side, which is a mistake and is not consistent with the other patients shown. As is clearly indicated in table 1, this patient's stroke was in the right hemisphere. The authors apologize for the error.

Correction

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