TEXAS NEUROLOGICAL SOCIETY WINTER 2013

Broca's Area

The Voice of Texas Neurology

President's Message

Sara G. Austin, MD

Dear TNS Members,

Welcome to this edition of Broca's. If you are like me you are overwhelmed with reading material, so thank you for taking the time to read this. We are gearing up for the 2013 Winter Conference and it should be a good one. Dr. Stanley Fisher has worked very hard to get us really good speakers and topics. The summer conference also promises to be interesting. It is focused on MS, and Dr. Ed Fox is the program director. We've had some big changes this year that we are adapting to. Ky Camero is our new executive director and is doing a great job. Please stop by and say hello during the winter conference.

I have realized this year that there are plenty of people in the TNS that spend a lot of their time making sure things get done – many thanks to those folks including Randy Evans who puts together Broca's, Bill Gilmer who interfaces with the TMA, Brent Bluett, our new Resident Representative, who is on the board, Bob Fayle and the education committee, Rob Leroy who is treasurer, ….and I can (and should) go on and on. I am proud to be part of the TNS, and it's been work, I mean fun, to be the president. I hope to see you all in Austin in February. Thank you again for being a member. We neurologists need to stick together!

All the best,
Sara G. Austin, MD

2013 WINTER CONFERENCE
February 22-24
Hyatt Regency Austin

(More details on page 4)

Big cuts in reimbursement for NCS for Medicare patients starting 1/1/13

By Sara G. Austin, MD and Bill Gilmer, MD

By the time this goes to print, you likely will have already discovered the unhappy lump of coal Santa Claus left on neurologist's doorstep this holiday. Here is the story as we understand it. On November 1, 2012, CMS released new codes for NCS in which they cut payment for these physician services by 50-75% as of January 1, 2013. The AAN had no warning this drastic action was even being considered. In fact, the AAN actively participates in the yearly payment update process that occurs through the AMA Relative Value Scale Update Committee (RUC) and had expected a "correction" (decrease) of perhaps 20%.

We all know EMG/NCS study volumes have exploded in the past several years largely due to a huge number of studies done by companies offering screening tests as an alternative to doing an appropriate history and physical by a physician. Every major Society has established standards that state that EMG/NCS is an extension of the neurologic examination. We all realize that the time required for a neurologic consultation is terribly undervalued, and many of us have offset this loss by procedures such as EMG/NCS, not unlike other medical specialties such as cardiology or primary care.

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Neurology Day!

Remember to mark your calendar for **April 2nd**. This is the **First Tuesday at the Capital** that has been designated for neurologists.

Mark Your Calendar

2013 Winter Conference
February 22-24
Hyatt Regency
Austin

2013 Summer Conference
July 19-20
Westin La Cantera
San Antonio

Editor's Notes

Randolph W. Evans, MD

This issue

I thank our officers and other contributors for their excellent submissions to this issue.

We look forward to seeing you at the TNS 16th Annual Winter Conference in Austin, February 22-24. Stanley Fisher, program chair; Gary Clark and Marvin Fishman, pediatric co-chairs; and Bob Fayle, education committee chair; and the education committee have planned a terrific program. Be sure to make your Hyatt Regency hotel reservation before February 1 to receive the discounted room rate and to register by February 15 to receive the early bird registration fee of $250 for the 2.5 day meeting.

Harris County Neurological Society

The members of the Harris County Neurological Society thank Aziz Shaibani for his superb service as outgoing president. Aziz organized outstanding quarterly programs several of which are available in video format on the TNS website. He will be the 2014 Winter Conference program chair. Reeta Achari will be the new HCNS president. I look forward to her enthusiastic leadership.

Neurology: still surprising, fun, and fascinating after 30 years

Despite the growing aggravation we all have with the business of medicine, the practice of neurology can still be surprising, fun, and fascinating as these two examples illustrate. You certainly have many examples in your own practice which help with frustration tolerance for the non-medical aspects.

Threes

It was an ordinary Tuesday fall morning. The first patient was a 33 year old woman with a six month history of left meralgia paresthetica immediately following a gynecological procedure which was improving on gabapentin. That was a simple case to start the morning. The next patient was a 48 year old obese woman with an over 1.5 year history of right meralgia paresthetica. Hmm. The third patient was a 42 year old obese woman with a three week history of right meralgia paresthetica immediately following a gynecologic procedure. That got my attention. Could I go for the record books and have a fourth in a row? No, the string was broken. The next two patients had headaches.

So this was improbable seeing three patients in a row with an uncommon disorder (33 per 100,000 patient-years). Do medical cases really come in threes or was this a case of selective memory and attention to random chance events? Pubmed and Google searches didn't reveal any specific literature on this topic. Physicians must be applying the common adages that good and bad things come in threes. (Consider Mencius' (372-289 BC) famous quote, "The best things in life come in threes, like friends, dreams, and memories.") Groups of three are particularly memorable including the Three Musketeers, Stooges, Pigs, Blind Mice, Wise Men, Ring Circus, Cheers, Dog Night, Men in a Tub, the Trinity, and so on.

Blip syndrome

About the same time as the "threes," I saw this patient.
This is a 47 year old male with a history of "little small short circuits" which had been occurring for seven months and were usually occurring two to three times per day but up to 10 times per hour. He described a blip in his head that he did not see or feel but sensed for a half a second. The symptom occurred when sitting, standing, or walking but not lying down. He had no alteration of consciousness, spinning, paresthesias, weakness, trouble speaking, associated headache or other neurologic symptom except for a brief feeling of slight imbalance.

There was a history of migraines without aura since childhood occurring about three days per week with an intensity of 3-4/10 relieved by aspirin in about one hour. There was a past medication history of hyperlipidemia. Neurological examination was normal.

He had seen another neurologist. A MRI of the brain with and without contrast and MRA of the brain were negative. Evaluation by an ENT physician including audiogram and ENG were normal. I obtained an EEG which was also normal. I suggested he follow-up with his cardiologist to exclude arrhythmias.

So what is the diagnosis?

I had never seen a similar case in 30 years of practice but recalled an article I had read of a peculiar syndrome (Lance, JW. Transient sensations of impending loss of consciousness: the "blip" syndrome.*J Neurol Neurosurg Psychiatry* 1996; 60:437-438). The Australian neurologist, Jim Lance, had similar sensations himself for about six months which he recognized were not associated with any cardiac extrasystoles (which he also had) which he named the "blip" syndrome (Lance JW. The blip syndrome. *MedJAust* 1994;160:585).

Lance then reported 12 additional cases (8 women; 3 physicians) ranging in age from 33 to 75 years with symptoms present for periods of two months up to five years with a typical frequency of episodes of one to four per month (five had two or more per day and one had 12-15 per day on some occasions) with each lasting a split second up to two seconds. Interestingly, 4/12 were migraineurs as was this case and none had seizure disorders. Patients variably described sensations including a short circuit in the brain, mind going blank for a second with pressure in the forehead and felt that she could lose balance, impending loss of consciousness, and like a wave going through her. Testing on some of the patients including EEGs, CT scans, EKGs, and carotid ultrasounds were normal. The etiology of these episodes is not certain. Lance compared blip syndrome to other benign disorders such as déjà vu, night starts, and exploding head syndrome.

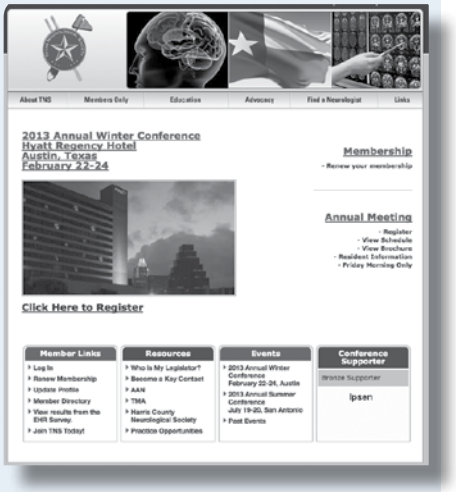
Whenever I see disorders reported as rare, I wonder whether they are truly rare or just rarely reported? Have you seen the blip syndrome?

Visit TNS online at texasneurologist.org

Remember to access the TNS website for more information on the Winter and Summer Annual Conferences.

Be sure to also check out the latest lecture on the Harris County Neurological Society page – Dr. John T. Kissel and his talk on Myalgia: Is It a Neuromuscular Disorder?

Don't forget to access and view UT Houston Grand Rounds under the education tab. If you have trouble viewing any information, you can contact the TNS office at 512-370-1532.



TNS Business Meeting

Saturday, February 23rd, TNS will conduct a brief business meeting. The society will present the Lifetime Achievement award to one of its most esteemed members and vote on bylaw changes. *See bylaw changes on page 17.*

Member News

Dr. Martin Heitzman (El Paso) - former TNS President died the last weekend in October after a long battle with cancer.

Dr. Jack Alpert wrote a book: Alpert, Jack N. *The Neurologic Diagnosis: A Practical Bedside Approach*. New York: Springer, 2012. Print.

Thank You to our Summer Meeting Supporter

IPSEN

TNS 16th Annual Winter Conference

Robert Fayle, MD
TNS Education Committee Chair

It's time for the winter meeting! Please mark your calendar for the TNS 16th Annual Winter Meeting: Friday, February 22 to Sunday, February 24, 2013. Registration is now open, and you can register online at www.texasneurologist.org. The meeting is located again at the Hyatt Regency Austin, 208 Barton Springs Road, Austin, TX 78704. For questions/ information about registration, call Amy Lawson, Meeting Registrar, at 512-370-1532.

The meeting promises to be another excellent program. The Pediatric Neurology program begins Friday morning with Pediatric Sleep Medicine, followed by discussions on Clinical uses of whole exome and whole genome sequencing, impact of genetic testing in pediatric epilepsy and a discussion of ethical issues in pediatric neurology.

The adult program begins Friday afternoon at 1:00PM. The program features diverse topics of both clinical neurology and clinical applications of cutting edge basic science, which have been selected to appeal to the interests of the practicing neurologist.

This year, we are also inviting neurology resident poster presentations from all the resident training programs in the state. Meeting attendees are urged to visit the poster section.

The faculty for the meeting is outstanding in its excellence. Once again our winter meeting will be a valuable educational meeting in an easy-to-reach location. Your officers and the Education Committee are pleased to present the 2013 winter meeting, and we hope to see you in Austin.

Hotel Information

Hyatt Regency Austin • 208 Barton Springs Rd • Austin, TX 78704
(888) 421-1442

Located on the south bank of Lady Bird Lake, between 1st Street and the Congress Avenue bridge. The Hyatt is the only hotel on Lady Bird Lake with a view of downtown Austin. The famous Lady Bird Lake 9-mile Hike & Bike trail is just outside the hotel's back door.

TNS has a special Hyatt room rate of $185 for singles and doubles, $210 for triple, $235 for quadruple. Room rates do not include tax, currently 15%. **Please call (888) 421-1442 by January 12 to make your reservations.**

Be sure to mention that you are part of the TNS meeting.
You can also book your room online at **https://resweb.passkey.com/go/TNS2**.

Parking

TNS has a special reduced rate of $5/day self-parking, for overnight and day guests. Valet parking is also available for $21/day.

2013 Coding Changes Neurologists Should Know About

Stuart B. Black, MD, FAAN
TNS Medical Economics Committee Chair
Chief of Neurology
Co-Director: Baylor Neuroscience Center
Baylor University Medical Center at Dallas

On July 6, the Centers for Medicare & Medicaid Services (CMS) released the new 2013 proposed Medicare Fee Schedule. In total, 119 CPT codes were deleted and 186 codes were added to the 2013's CPT code list. In addition 253 codes and 18 CPT modifiers were changed or altered. Many of these variations could have an influence on neurologists. To fully appreciate the effect some of these changes will have on individual practices, physicians must again direct their thinking on Relative Value Units (RVU) and the Conversion Factor (CF); both products of the Resource Based Relative Value System (RBRVS) which was put into effect January 1992. While the objective of this condensed article is to focus on a few select 2013 CPT codes which may impact neurologists, perhaps a very brief review of RVU and CF will help some readers comprehend the foundation of how these CPT values alter our reimbursements.

RBRVS attaches a relative monetary value to each CPT code. Every medical service represented by a CPT code has three components. A RVU is a numeric value that has been developed to represent the three components of each medical service:

1. **Physician Work (w)**: This is the physician's time, skill, mental effort, risk and psychological stress of an adverse outcome. The physician's work accounts for 52 % of the total RVU
2. **Practice Expense (pe):** Includes staff salaries, rent, office supplies and equipment. The Practice Expense accounts for 44% or the total RVU
3. **Medical Liability Insurance (m):** This accounts for 4% of the total RVU

CMS also factors in a geographic adjustment factor known as the Geographical Practice Cost Index (GPCI). The GPCI adjusts the RVU for regional differences in the cost of living, rent, wages and disparity in malpractice premiums. Thus one can calculate an RVU as follows: Total adjusted RVU = RVUw x GPCIw + RVUpe x GPCIpe + RVUm x GPCIm. The reimbursement for any given CPT code is based on the RVUs assigned to the code multiplied by a Conversion Factor that translates the RVUs into a dollar amount. Thus, the physician reimbursement for any CPT code is Payment = RVU x CF. The 2012 physician fee schedule conversion factor was $34.0376 (just as a comparison, the CF in 2007 was $37.89 for a code worth 1 RVU). If Congress were not to act on the statutory formula used to determine Medicare physician payments, the result would be a decrease of 27%; or a CF of $24.8441. Recognizing a 27% decrease in reimbursements would be disastrous, let us look at a few of the CPT coding changes that have been approved and are scheduled to go into effect January 2013.

1. Nerve Conduction Tests

By now, most neurologists are aware that CMS has set the 2013 RVUs for electrodiagnostic codes. The RVUs for NCS codes are significantly reduced primarily due to changes in the practice expense. The impact to neurologists performing these procedures is considerable; with estimates of up to 50% decrease in reimbursements for some standard procedures. NCS codes *95900, 95903, 95904, and H-reflex codes 95934* have been deleted. Seven new nerve conduction codes *(95907 – 95913)* have been established.

In the new CPT codes, the unit of service is the number of nerve conduction studies performed; as compared to the prior codes *95900 – 95904* where the unit of service was each nerve. The new codes *95907 – 95913* no longer differentiate between motor and sensory, F-wave and H-reflex. Per the new ruling, "For purposes of coding, a single conduction study is defined as a sensory conduction test, a motor conduction test with or without an F-wave test, or an H-reflex test. Each type of study (sensory, motor with or without F-wave, H-reflex) for each nerve includes all orthordromic and antidromic impulses associated with that nerve and constitutes a distinct study when determining the number of studies in each grouping (e.g. 1-2 or 3-4 nerve conduction studies)".

For example, a unilateral Carpal Tunnel evaluation may have generated 14.92 RVUs in 2012 with a reimbursement of $507.84. The RVUs for 2013 would be 7.9 with a reimbursement of $268.90. The percent RVU change from 2012 to 2013 would be -47%. Thus, each type of NSC is counted only once when multiple sites on the same nerve are stimulated or recorded. When using the new 2013 codes, the numbers of these separate tests are to be added to determine which codes to use. A list and definition of the new NCS codes can be found on the AAN website.

TNS 10th Annual Summer Conference — First Notice

Edward Fox, MD, PhD, FAAN
Summer Program Director

The **TNS Summer Conference** will be held on July 19-20, 2013 at the Westin La Cantera in San Antonio, Texas.

This year, the National Multiple Sclerosis Society is co-sponsoring the meeting, and one half-day of the conference will focus on new advances in MS.

Additional lectures regarding other hot topics in neurology, as well as an ethics hour, will be part of the program.

Stay tuned for further details and plan to attend!

2. CPT Revises Definition of New and Established Patient.

The AMA Editorial Panel and CPT have clarified the definition of a new patient visit. A "new patient" means the patient has not received professional care from the primary physician or another physician of the **exact** same specialty and **subspecialty** who belongs to the same group practice, within the past three years. Conversely, an established patient is one who has received professional services from the physician or another physician of the **exact** same specialty and **subspecialty** who belongs to the same group practice within the past three years. These revisions were actually published in the 2012 Current Procedural Terminology (CPT) E/M guidelines.

To assist neurologists in determining whether a new patient encounter may be reported in accordance with the new CPT guidelines, the AAN has published a position statement which defines the neurology subspecialties. According to the AAN, "Neurologists of different subspecialties who see a patient within three years of another neurologist in the same group should report a new patient E/M code for that visit. It is appropriate for neurologists to determine whether they are of a "different subspecialty" according to one of the designated subspecialties or other specialties recognized within the medical community." The AAN has compiled a list of neurology subspecialties to assist neurologists in making the determination whether a new patient encounter may be reported using the decision tree. The categories of subspecialties are listed as follows: ACGME (The Accreditation Council for Graduate Medical Education, UNCS, and subspecialties recognized by ABPN. The Health Care Provider Taxonomy Code Set-Psychiatry & Neurology is also listed as a code set which is an external, nonmedical data code set designated for use in an electronic environment including transactions mandated under HIPPA.

However, as many of you would already be aware, just because CPT makes a change, it does not mean that CMS will follow suit. It appears that the "subspecialty" designation used by CMS will continue to be their Provider Specialty Code list which requires board certification for most specialties and subspecialties. Basically, CMS claims that processing systems do not recognize physician subspecialties. Therefore, CMS's position for billing new and established E/M codes is still interpreted to mean a patient who has not received professional services from the physician or another physician in the same group practice and same specialty within the previous three years. For Medicare services, the same specialty is defined according to the physician's primary specialty enrollment in Medicare. Since private payers may or may not follow the CMS determination, this may be a good time for sub specialized neurologists to revisit payer contracts to define whether subspecialty services may be an additional recognition. Another option is for subspecialists within a group practice to consider having a separate provider number from other members of the group.

3. Chemodenervation for Chronic Migraine

Effective January 1, 2013, physicians will be able to report the new CPT code 64615 when using Botox for chronic migraine. The October 15, 2010 FDA approval of Botox "…to prevent headaches in adult patients with chronic migraine" followed the pooled results from the double-blind, randomized, placebo-controlled Phase 3 Research Evaluating Migraine Prophylaxis Therapy (PREEMT) 1 and 2 trials. Those patients eligible for Botox must meet the IHS criteria for chronic migraine; specifically headaches ≥ 15 days per month for ≥ 3 months with headaches occurring on ≥ 8 days being classified as migraine headaches without aura or headaches that respond to migraine specific medications.

There will be various barriers from insurance companies regarding the use of Botox for chronic migraine. For example some carriers will require extensive documentation that treatment with other migraine preventive medications have failed. Some carriers have already indicated that precertification requirements must include "…adults who have tried and failed trials of at least 3 classes of migraine headache prophylaxis medication of at least 2 months (60 days) duration for each medication". These criteria are mandated even though none of the other listed medications are proven effective against chronic migraine and none are FDA approved for the indication.

The results of the PREEMT study, FDA approval, and the new CPT code (64615) for chemodenervation to treat chronic migraine is a major step forward in the treatment for a group of patients who had previously been excluded from migraine prophylaxis trials because they were considered too highly disabled and treatment resistant.

4. E&M codes *99487, 99488 and 99489* are three new CPT codes created for Complex Chronic Care Coordination.

These codes were developed to specifically address the CMS request to ensure that care coordination services were valued within the E&M management services. All of these codes represent an innovative step in reporting the health claims for non face-to-face work and time and/or one face-to-face office or other outpatient, home or domiciliary evaluation. Specifically, the primary focus of these codes is to allow physicians and other qualified health care providers (NPs, PAs, etc.) to report the work and time they spend on a patient's care; including the non face-to-face-care for a patient who may be confined to home or resides in a rest home or assisted living facility. These codes can only be reported once per calendar month.

It is not yet known whether CMS will add these transition CPT codes to the Medicare fee schedule. It is also not clear whether these codes will be specific for a Medical Home or ACO as opposed to other specialties as neurology.

5. There are other new CPT codes for Autonomic Function Tests *(95924 and 95943)*, Intraoperative Neurophysiology *(95940 and 95941)* and Pediatric Polysomnography *(95782 and 95783)*.

Two new Transition Care Management Service codes *(99495 and 99496)* have been created to report services for the follow up care of an established patient once they have been discharged from a facility setting to their community setting. The facility setting may be an acute hospital, rehabilitation hospital or comparable environment. The CPT Transition Care codes can be reported by a physician or qualified health care provider under the physician's direction. The codes encompass one face-to-face visit with the patient after discharge and the non face-to-face care provided during the service period. As with the Complex Chronic Care codes, it is not entirely clear what group of physicians will be reimbursed for the Transition Care codes.

The American Academy of Neurology website has excellent reviews and comments on the 2013 CPT updates that will affect neurologists. There is also a detailed evaluation under the Coding and Reimbursement section on the change in RVU values for Nerve Conduction Studies. The decreases in reimbursements are also demonstrated for various NCS procedures. As stated in the opening paragraph of this discussion, the objective of this article was to focus on a few of the new 2013 CPT codes which may be of interest to various TNS members. There are many more changes, some tied to the Accountable Care Act which will significantly influence the medical economic environment for neurologists. The TNS will try to keep our membership as updated as possible as these transitions occur.

What disables? Tips for charting in chronic diseases.

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Neurologist
Chief Medical Consultant
Texas Department of Assisted and Rehabilitation Services
Disability Determination Services

Neurologists care for patients with chronic, debilitating conditions, some of which lead to disability. The definitions for disability vary. The Social Security Administration defines disability as: (1) the patient cannot do work they did before; (2) SSA determines they cannot adjust to other work because of their condition; and (3) the disability has lasted or is expected to last at least one year (or result in death). The Americans with Disability Act (ADA) defines disability with a three-part definition of disability. Under ADA, an individual with a disability is a person who: (1) has a physical or mental impairment that substantially limits one or more major life activities; or (2) has a record of such an impairment; or (3) is regarded as having such an impairment.

Critical to the administrative assessment of disability is the evidence and findings gleaned from physicians' office notes. However, physicians chart for their own treatment purposes and to satisfy Medicare requirements for documenting service provided, and are not charting with legal and administrative reviews in mind. A frequent result is that the longitudinal treatment record may not be an accurate reflection of the true health and functional condition of the patient. An additional complication is the advent of the electronic medical record (EMR), which often contains unedited macros. Following are some tips to help charting in general, and to help strengthen office records for your patients with chronic conditions.

Tip #1: Avoid ambiguous terms.

This applies to notations of history, impressions and also the physical examination. For example, the phrase "doing well" needs context and clarification. The patient is "doing well" as compared to what? Are they functioning normally compared to a healthy person, or doing well compared to a chronically ill patient with a similar condition? In the administrative world, it is not uncommon for a patient with Multiple Sclerosis, for example, to allege on disability application that they have persistent fatigue and cognitive symptoms, but the physician's longitudinal record over time records a "normal" system review at each visit, and the conclusion is the patient is "doing well."

Tip #2: Take a complete system-pertinent review.

Ask and document responses at each visit for the affected body system, and record positive and negative responses. Chart notes often say "other systems negative" or "system review negative," without specifying what questions were specifically asked and answered. For patients with chronic diseases in the office for a scheduled appointment, this can leave a reviewer with the impression of greater health and function than is truly present.

Tip #3: Ask specifically about "soft" symptoms.

In addition to asking patients about traditional neurological symptoms such as paresthesias, ask about fatigue, energy, sleep patterns, malaise, and generalized weakness. Such symptoms can be persistent, are often over-looked, and can be a significant contributing factor to the claimant feeling disabled. Sensory symptoms, such as vision and hearing changes, are often overlooked in the routine office visit for ongoing care of a chronic condition.

Tip #4: Be precise in the physical examination.

Motor weakness should be documented per affected muscle group, and not regionally. Statements such as "RLE 4/5" provides little useful information, whereas a detailed exam per affected muscle group provides more precise and useful information. Similarly, describing a gait as "limping" or "unsteady" can be further delineated and clarified. Describing the motor examination as "non-focal" can be useful shorthand during phone or hallway conversations, but lacks precision in office notes.

Tip #5: Chart the static conditions.

In routine office notes (a patient coming for medication refills or routine visit for a chronic pain condition), a patient can be seen for years before a new physical examination is charted. Many notes may simply say, "unchanged," but the reference baseline examination was several years in the past. Similarly, when a patient is seen for a chronic pain condition (e.g., failed back syndrome), there can be long periods in a physician's longitudinal record before a physical examination is found.

Tip #6: Avoid administrative jargon.

The physician is in the unique position to provide information about the patient's conditions, and the intensity and severity of their symptoms and functioning. Leave global, administrative or legal terms out of requested physician statements, unless they are amply clarified. For example, a statement such as, "My patient is disabled," carries little accepted meaning and no content. There are many definitions of disability and a stand-alone statement like this provides no perspective or context. By whose definition is the patient disabled? Is the physician trying to advocate on behalf of their patient and merely echoing what the patient is saying in the office visit? However, a statement about the nature of the condition, the intensity and severity of symptoms is quite valuable. Contrast the above statement with something like this: "I have been treating Mr. Smith for eight years for Chronic Progressive MS. He has lower extremity spasticity with weakness, uses a cane at all times for balance. His cane use is in all condition, both for home and community ambulation. He uses a motorized scooter when shopping. His energy level is poor, he has significant fatigue, and takes two to three naps daily to help with the fatigue."

Avoid statements such as, "Mr. Jones can only do Light work." Vocational terms such as medium or light work are industry-specific. Rather, give the medically-based physical limitations as precisely as possible.

Tip #7: Beware the Electronic Medical Record

The EMR may improve health care in terms of gather data, making records accessible, and documenting visits for the purposes of Medicare payments, but using an EMR requires vigilance. Default, "normal" macros will populate an office visit. Left unedited, these electronic notes can leave a misleading and potentially false view of a patient's health.

Physicians are grounded in clinical medicine, but their records exist in a larger, legal and administrative context. Awareness of these overlapping worlds (clinical, legal and administrative) will help improve the quality of our office notes and ultimately improve patient care. Better charting will serve the patient well if they should enter the administrative world of disability assessment.

"Big cuts in reimbursement " continued from page 1

CMS identified NCS as "overvalued" about a year ago based on the fact that the number of studies performed had risen faster than expected based on demographics. They referred it to the AMA's RUC committee which did an assessment and ended up recommending about a 15% decrease in reimbursement. CMS (Centers for Medicare Services) got the recommendations but ignored them and made the unilateral decision to drastically reduce reimbursements (much as a proposed Independent Payment Advisory Board (IPAB) might do) to reduce expenses. What they did not do was to simultaneously increase the value of cognitive care – time spent with patients – that is drastically underpaid. Our calculations predict about a 65% reduction in reimbursement for routine studies. As you know EMG reimbursements were also reduced slightly last year.

Here is what we have been doing about it. The AAN has been working together with the American Association of Neuromuscular and Electrodiagnostic Medicine (AANEM) and the American Academy of Physical Medicine and Rehabilitation (AAPM&R) to explain the ramifications of this action to Congress and CMS. They met with CMS in November to begin discussing this in person. They have written official comments to CMS, and have sent an email around to all physician members encouraging us to comment directly to CMS. They will continue to need ammunition and real life stories of how this action will harm patients. The AAN legislative staff has visited offices on the Hill to line up support for signatures on a Congressional letter to CMS. The AAN is also talking to the AMA about problems that result when CMS makes decisions like these without physician input (essentially ignoring the AMA, the RUC, and physician input).

The TNS has also been working on it. We submitted our own comments to CMS in November. We will follow any action emails that the AAN puts out with our own emails to remind members to act. Please voice your comments to your representative in Congress and the CMS. When you receive special requests from AAN or Vocus (an easy way to express an opinion to congress) please do so that day! As they say – "Vote early, Vote often!" We live in a representative democracy. That means the only way things change is by convincing our representatives that they must take action.

Stuart Black, MD, our medical economic committee chair, has been very helpful talking to private insurers about how they might deal with this change. They uniformly were surprised with this and have not decided yet how they will respond. At least from what they have told Stuart, none of them completely tie their fee schedule to Medicare and so they will all be thinking through this change very carefully. We will continue to talk with them.

Visit www.texasneurologist.org and www.aan.com for more up to the minute information.

Expert Opinion: Headache, Temporomandibular Disorders, and Bruxism

Steven D. Bender, DDS
Fellow, American Headache Society
Fellow, American Academy of Orofacial Pain
Director; The North Texas Center for Head, Face & TMJ Pain
Plano, Texas

Case History:

A 29 year old female presented with a 61/2 year history of daily, migraine like headaches. She wanted to find out if her temporomandibular joint "problems" contributed in any way to her daily head pain. She indicated being aware of sleep bruxism since high school and reported utilizing a dental splint since 2000. Prior to her consultation in our office, she had previously consulted with an acupuncturist, general dentist, oral surgeon, chiropractor, endocrinologist, internist, neurologist, physical therapist, pain medicine physician, psychologist and psychiatrist. Previous therapies included medication; both abortive and prophylactic, inpatient treatment at a nationally known headache center, nerve blocks, rhizotomies, cryotherapy and a dental splint. She also reported PFO closure in 2008. She indicated that the most robust relief resulted from the cryotherapy but it would only last for approximately two months before the pain returned to the previous baseline level. Of significance in her medical history was hypothyroidism and treated depression. Her current medications included; thyroid, Celexa, Xanax, fenofibrate, fish oil, a multivitamin, B-complex, Atarax, Phenergan and Zofran. Her pain presentation was continuous and bilateral involving the temples, occiput, periocular and neck areas. She indicated her pain would vary in presentation and she used the descriptors; throbbing, dull, stabbing, pressure, burning and shooting. Along with her pain she indicated nausea, vomiting, photophobia, phonophobia and osmophobia. She gave no history of aura. A family history of headaches included her mother, sisters and maternal grandmother.

My physical examination revealed a normal neurologic screening, BMI of 33.1, normal pulses and afebrile. She was alert and oriented in all spheres. Her ENT screening was also normal. Opthalmoscopic exam revealed grossly normal fundi. There was mild pain to palpation of the masseter and temporalis muscles bilaterally as well as the left cervical spine. Mild pain was also reported to palpation of the right and left occipital nerve areas. Her intraoral examination was normal as was her mandibular range of motion. There was evidence of tooth wear as well as scalloping of the tongue lateral borders and ridging of the buccal mucosa noted. No pain was reported at the right or left TMJ nor was there pain reported with mandibular movement.

Based on her history of sleep bruxism as well as signs and symptoms of such, the decision was made to fabricate a custom intraoral splint for sleep. After approximately two months of nightly wear, she reported a decrease in her headache severity although she still had daily pain. At 10 months, her headaches had completely resolved and she remains headache free at 18 months.

Question:

When is appropriate to consider bruxism and the TMJs in the refractory headache patient?

Expert Opinion:

Headache and temporomandibular disorders (TMDs) are very prevalent conditions in the general population[1-8]. TMDs are defined as a collection of symptoms and signs involving masticatory muscles, the temporomandibular joints (TMJs) or both[9]. The pain reported by TMD patients is typically located in the muscles of mastication, in the preauricular area, or in the temporomandibular joint (TMJ)[10].
Clinical and epidemiological studies have demonstrated an association between headache and TMDs, suggesting that individuals with headache and those with TMDs, often share similar signs and symptomatic presentations[8, 11, 12].
Inflammatory mechanisms have been shown to be involved in temporomandibular joint pain and dysfunction[13]. Milam proposed a possible etiology for inflammatory mechanisms of the temporomandibular joint structures by what was described as a hypoxic-reperfusion injury[14]. This process occurs when the capsular pressure of the temporomandibular joint exceeds the end-capillary perfusion blood pressure of the feeding vasculature. The area then undergoes reperfusion via mouth opening or relaxation of the elevator muscles. It was speculated the reperfusion resulted in the release of free

radicles and initiated inflammatory processes. Capsular nociceptive fibers triggered by pathologic loading of the highly innervated synovial tissues may also stimulate the release of calcitonin gene-related peptide and substance P, leading to further inflammatory processes. Pathologic loading is often attributed to sleep parafunctional behaviors such as sleep bruxism (SB)[15]. Sleep bruxism may also be responsible for stomatagnathic muscle nociceptive signaling. Christensen reported that muscle pain was noted in subjects who voluntarily clenched for 20-30 seconds[16]. Kydd and Daly reported that nocturnal clenching events can last as long as 20-40 seconds[17]. These sustained isometric contractions observed in sleep bruxism could lead to tissue injury and subsequent nociceptive signaling from both the myogenous and arthrogenous components of the temporomandibular joint complex. Previous investigations have also described mechanisms for nociceptive referral from the temporomandibular joints (TMJs) to the cranial structures[18, 19].

What is Bruxism?

The American Academy of Orofacial Pain defines bruxism as diurnal or nocturnal parafunctional activity including clenching, bracing, gnashing, and grinding of the teeth[20]. Bruxism may be classified as awake bruxism (usually but not always diurnal) or sleep bruxism (SB) (usually but not always nocturnal). Both types of bruxism are either primary (idiopathic), in which case there is no associated medical condition, or secondary (iatrogenic), in which case there is an associated medical condition. Awake bruxism mostly involves teeth clenching or tapping and jaw bracing, with or without tooth contact[21]. Grinding is rarely noted during waking hours. The overall prevalence of awake clenching is about 20 percent in the adult population, with more women reporting clenching awareness than men[21]. As it appears that SB differs in etiopathology from awake oromandibular parafunctional activities, care should be taken to differentiate the two as different entities[22].

In 2005, the International Classification of Sleep Disorders classified sleep bruxism as a "sleep-related movement disorders"[23]. Previously it had been termed a "parasomnia", or a disorder of arousal. Sleep related movement disorders are considered simple, stereotypic, repetitive, and localized movements during sleep that also includes periodic limb movement disorder and rhythmic movement disorder[24]. Although SB is comprised of rhythmic, repetitive mandibular movements, it can also involve forceful, as well as prolonged clenching of the dentition. Nishigawa and colleagues demonstrated that bite force during SB can exceed the amplitude of maximum voluntary bite force during the daytime by as much as 111.6%[25]. Up to 65% of SB patients of all ages report frequent headaches[26, 27] Risk factors that have been shown to exacerbate SB are; (1) smoking, caffeine and heavy alcohol drinking[28, 29]; (2) type A personality and/or anxiety[28, 30-32]; (3) sleep-related breathing disorders[28, 33] and; (4) periodic limb movements[28, 34, 35]

Due to the continued disagreement about the definition and diagnosis of this SB[36], the literature on the subject is often difficult to interpret. Currently, there is no single clear pathophysiologic mechanism identified as responsible for SB. Studies have demonstrated that approximately 60 percent of "normal" sleepers exhibit what is known as rhythmic masticatory muscle activity (RMMA) during sleep[37]. RMMA is defined as three masseter muscle bursts or contractions within an episode, in the absence of teeth grinding[37]. This type of oromotor activity corresponds to the chewing automatism previously described by Halasz[38] and is not necessarily correlated with tooth grinding. The frequency of RMMA during sleep is three times greater in SB subjects[37]. RMMA can be induced seven times more frequently in SB patients, suggesting that SB is probably related to a heightened responsiveness to transient arousal during sleep[39]. Also, many of these experimentally induced arousals were accompanied by tooth-grinding. In SB subjects, episodes occur at a frequency of 5.8 times per hour of sleep as compared to 1.8 episodes for non SB individuals[37]. Grinding noises are reported to occur in approximately 44 % of SB/RMMA events[40, 41]. SB episodes tend to occur primarily in non-rapid eye movement sleep (NREM) stages 1 and 2 (light sleep) with only 10% occurring during rapid eye movement sleep (REM) [37, 42, 43]. SB episodes appear more in the second and third NREM to REM sleep cycles as compared to the first and fourth cycles[44]. Also, SB episodes will occur more frequently in the ascending period of sleep within a cycle[44]. Ascending into lighter sleep has been shown to be associated with an increase in sympathetic tone and in arousal activity[45, 46]. SB has been demonstrated to occur subsequent to a sequence of physiological events that consists of an increase in sympathetic nervous system activity, cortex activation, heart rate increase, and ultimately an increase in jaw depressor muscle activity[47]. 88% of the time, SB episodes tend to occur along with cyclic alternating patterns (CAPs)[42]. CAPs consist of activation of electroencephalogram (EEG) and electrocardiogram (EKG or ECG) patterns and occur approximately every 20–60 seconds during non-REM sleep[48]. These events are thought to be physiologic events that support sleep quality. Some of the suggested causes for sleep bruxism include; stress and anxiety, occlusal factors, genetics, sleep related breathing disorders and neurochemical factors.

Evaluation

The most widely used and accepted method for evaluating the condition of the stomatagnathic musculature is by digital palpation[49-51]. Application of about 4-5 pounds of pressure (the pressure necessary to blanch the finger nail

bed) applied with the palmer surface of the index, middle and fore fingers across the muscle fibers can be diagnostic of muscular abnormalities[52]. The examination should identify tender areas as well as potential trigger points, which are thought to be resultant from abnormal motor end-plate activity releasing excessive amounts of acetylcholine[53]. A cursory stomatagnathic muscle examination would include the following muscle groups; temporalis, masseter, sternocleidomastoid, splenius capitis, semispinalis capitis and the anterior portion of the trapezius muscle. The lateral pterygoid muscle, involved in opening and protruding the mandible, must be functionally assessed as it is not possible to manually palpate this muscle[54, 55]. The parafunctioning patient may not necessarily present with painful masticatory symptoms. Examination of the oral structures may reveal worn dentition as well as scalloping of the oral tongue lateral borders and ridging of the buccal mucosa[56-59].

The temporomandibular joint can also be assessed by digital palpation. The location of the mandibular condyle can be identified in the area anterior to the tragus of the ear by having the patient open and close several times and feeling for the movement of the lateral aspect. It is important to have the patient then clench their teeth in order to ensure proper positioning of the finger tips. If muscle contraction is felt, it is probable that the fingers are resting on the area of the deep portion of the masseter muscle and not the lateral aspect of the condyle. Joint popping or crepitation can also be assessed by light digital palpation or by using the bell end of a standard stethoscope.

Conclusion

Temporomandibular disorders include a variety of musculoskeletal disorders that may affect mandibular function. Pain and dysfunction of the temporomandibular joints and associated structures can be a source of headache and orofacial pain. However, the SB patient may not always demonstrate significant pain in these structures. SB may produce subclinical nociceptive signaling from the stomatagnathic structures to the trigeminal nucleus increasing central sensitization. Recognition, evaluation and effective management of the SB patient has the potential to increase headache treatment efficacy and potentially reduce the need for pharmacotherapy, especially in those who present as refractory to the traditional treatment protocols.

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Adult onset cerebellar ataxia-a review

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What is the main objective of this review?

- To provide a brief overview of the diagnosis and treatment of progressive adult onset ataxia.

What are the ataxias?

- They are clinically heterogenous disorders caused by pathological processes affecting the cerebellum and cerebellar pathways resulting in impaired coordination.
- The cerebellum's main function is to integrate information relayed to it and facilitate the execution of precise movements.
- Lesions of the cerebellum and its connections can result in breakdown and incoordination of movement.

What processes can cause ataxia?

- The pathophysiology of cerebellar ataxias is as diverse as the various neurological and systemic diseases affecting the cerebellum.
- Broadly classifying ataxias into **genetic** and **non-genetic** conditions is a first step in discovering their underlying mechanism.
- **Non-genetic** ataxias are caused by **acquired conditions, sporadic neurodegenerative disorders**, or from unknown processes in which case the descriptive term **idiopathic late-onset cerebellar ataxia**[1] (**ILOCA**) is used to describe the disorder.

What are common cerebellar symptoms?

- **Difficulties with gait and balance** commonly described as: "losing balance," "staggering," "walking like a drunk," "cannot walk a straight line," etc.
- Other complaints: dizziness, blurred vision, slurred speech, difficulty with swallowing, clumsiness, sloppy handwriting, poor fine motor skills, and tremor.

What is involved in the evaluation of an ataxic patient?

- As with all neurological disorders, a detailed history and thorough examination are prerequisites for an accurate diagnosis and set the stage for the diagnostic investigation.

How are history and examination findings interpreted?

- Motor and non-motor symptoms, family history, acquired risk factors (exposure to toxins and certain general medical conditions), and tempo of progression are key elements of the history.[2]
- **Cerebellar symptoms:** (see above) point to an ataxic disorder
- **Non-cerebellar symptoms:** the following are more tightly correlated with disease than others:
    - Postural dizziness, erectile dysfunction, urinary symptoms, and dream-enactment behavior (suspicious for Rapid eye movement behavior disorder or REMBD): Multiple System Atrophy-C (MSA-C)
    - Profound cognitive and behavioral changes: sporadic Creutzfeldt-Jakob disease (CJD); paraneoplastic, infectious, and immune-mediated limbic encephalitides
    - (Other neurological symptoms, when corroborated by examination findings, may help with the diagnosis)
- **Family history:** very helpful when present, but doesn't exclude a genetic cause when absent or unknown. Usual patterns of inheritance are autosomal dominant (AD) or recessive (AR) and X- linked. Consanguinity between parents should alert to an autosomal recessive disorder.
- **Common risk factors for cerebellar damage:**
    - frequent and excessive alcohol consumption; exposure to toxins such as mercury; use of medications like phenytoin, lithium, and chemotherapeutic agents
    - HIV, hepatic cirrhosis, multiple sclerosis (MS), and autoimmune diseases
    - gastric-bypass procedures and malabsorption states causing deficiency of vitamins E and B1

- **Rate of progression** of ataxic symptoms:[3]
  - Acute and abrupt: strokes and structural brain lesions
  - Hours and days: infectious or parainfectious cerebellitis; immune-mediated disorders such as Miller-Fisher syndrome (MF); acute toxin exposure; rapid metabolic derangement; MS
  - Weeks to months: paraneoplastic disorders; anti-glutamic acid decarboxylase (GAD)-antibody syndrome; steroid-responsive encephalopathy and ataxia (SREAT or Hashimoto's encephalopathy); gluten ataxia in Celiac disease (GA); vitamin deficiency states [e.g. ataxia with vitamin E deficiency or AVED, B1 (thiamine) deficiency]; general medical conditions such as hepatic encephalopathy; infections (HIV, CJD); MS; sensory polyneuropathy and ganglionopathy (SPN and SG)
  - Chronic and indolent (months to years): genetic ataxias; toxins (primarily alcohol); MS; storage –disorders (lipid, lysosomal, peroxisomal); sporadic neurodegenerative disorders (MSA-C); ILOCA; SPN and SG; atypical parkinsonian conditions such as Progressive supranucleary palsy (PSP); Neurosyphilis (NS)
  - Rates of progression vary in individuals. All possible etiologies should be considered when the clinical course is not firmly established.
- **Cerebellar signs**: nystagmus, saccadic dysmetria, impaired cancellation of vestibulo-ocular reflex, dysarthria, limb ataxia, titubation, dyssenergia, impaired check on rebound testing, end-intention tremor, wide stance, and difficulty with tandem stance and gait.
- **Extracerebellar signs** and related diseases:[3,4]
  - Orthostatic hypotension, dysphonia, dystonia, pyramidal signs, and parkinsonism: MSA-C (the most common non-genetic degenerative ataxia)
  - Dystonia, parkinsonism: several SCAs; DRPLA; Wilson's disease and Neuroacanthocytosis (NAC) in a younger cohort
  - Action tremor, dysexecutive syndrome, neuropathy, parkinsonism: Fragile-X tremor ataxia syndrome (FXTAS)
  - Chorea: Huntington disease, HD; dentatorubropallidoluysian atrophy, DRPLA; SCA 17; Ataxia telangiectasia, AT; SCAs 1,2,3
  - Myoclonus and cognitive impairment: hepatic encephalopathy; CJD; anti-GAD syndrome; POLG (polymerase γ) mutation
  - Pyramidal signs, sensory loss: strokes; acquired and genetic myelopathies; hereditary spastic parapareses; spinocerebellar ataxias (SCAs); Friedrich's ataxia (FA); MS; NS
  - Sensory loss, hyporeflexia: AR ataxias; SPN and SG ("sensory ataxia"); GA; MF; AVED; NS
  - Cognitive and psychiatric symptoms: CJD; Wernicke-Korsakoff syndrome; SCA 17; late-stage AD ataxias; AR adult-onset inborn errors of metabolism; leukodystrophies; NS; Whipple's disease
  - Eye-movement abnormalities: MS; ataxias with oculomotor apraxia 1 and 2 (AOA1, AOA2); SCA 2; Whipple's disease; Ataxia telangiectasia, AT; MF; PSP (impaired vertical saccades)
  - Visual loss: MS; ataxia with vitamin E deficiency (AVED); SCA7; mitochondrial disorders
  - Neuromuscular deficits: mitochondrial disorders
  - Telangiectasias: AT
  - Achilles xanthomas and early cataracts: Cerebrotendinous xanthomatosis, CTX

What diagnostic tests are recommended for ataxia evaluation and what is their relevance?[1,2]

- **Brain MRI** is indispensable. It may reveal:
  - Structural lesions and strokes
  - Atrophy of the cerebellum and brainstem: chronic processes such as genetic ataxias
  - Abnormal signal and atrophy of the basal ganglia: Wilson's disease; HD; mitochondrial disorders; NAC
  - Putaminal atrophy and cruciform hyperintensity in the pons ("hot-cross bun" sign): MSA-C.
  - Middle cerebellar peduncle lesions: FXTAS
  - White matter abnormalities: MS; adult-onset leukodystrophies (Alexander disease, AD; Adrenoleukodystrophy, ALD)
  - Diffusion-weighted abnormalities ("cortical ribboning") and symmetric thalamic changes ("pulvinar" sign): CJD
- **Spinal cord MRI** is suggested for myelopathic signs
  - Severe cord atrophy: FA; Alexander disease
- **Serum testing** is guided by the clinical evaluation and imaging:
  - 1st tier: blood chemistries; renal and liver function tests; ammonia; complete blood counts with differential (CBC diff); erythrocyte sedimentation rates (ESR); Antinuclear antibodies (ANA); thyroid and vitamin levels (B12, B1, E, B6, A); folate; glucose tolerance test; methylmalonic acid; infectious serologies (HIV antibody, Lyme antibody, RPR); Serum protein electrophoresis with immunofixatoin (SPEP with IFE)

  - 2nd tier (tests for rare ataxias and potentially treatable conditions, to be ordered if 1st tier testing is inconclusive): creatine kinase; lactate; pyruvate; □-fetoprotein *(elevated in AT and AOA 2)*; fasting lipid profile; paraneoplastic antibodies (Hu, Yo, Ri, Ma, TA, CARP8, CV2, Tr, LEMS, MGLUR1, CRMP5, GQ1b, amphiphysin, PCA-2, NMDA, VGKC, ganglionic acetylcholine receptor antibodies); anti GAD65 antibodies; SSA, SSB antibodies (Sjögren's antibodies); antigliadin antibodies (IgA and IgG); serum iron studies; alkaline phosphatase; thyroperoxidase (TPO) antibodies; 24 hour urine copper and zinc; serum copper and ceruloplasmin; urine heavy metals; Human T-Cell lymphotropic virus I, II; T. Whippelei PCR; cholestanol levels (if CTX is suspected)
  - 3rd tier (rarer genetic conditions typically seen in a younger cohort with ataxia and other symptoms such as dystonia, peripheral neuropathy, visceral involvement and cognitive impairment): peripheral blood smear for acanthocytes (for NAC); lysosomal screen; plasma amino acids; urine organic acids; serum ketones; fasting very long chain fatty acids (for ALD)
  - **Cerebrospinal fluid studies** are obtained for paraneoplastic, immune-mediated, infectious and inflammatory disorders: protein; glucose; CBC diff; cultures; IgG synthesis, index, rate; oligoclonal bands; cytology; lactate; 14-3-3 protein; paraneoplastic antibodies; viral encephalitis panel; VDRL
- **CT or PET scan** of the body to look for occult malignancy.
- Additional tests include
  - EEG (helpful in CJD)
  - Electromyogram and nerve conduction studies
  - Autonomic studies
  - Sleep study (to look for REMBD)
  - Rarely, nerve and muscle biopsies for mitochondrial ataxias
  - Rarely, brain biopsy for leukodystrophies
  - Magnetic resonance spectroscopy of the brain
  - Dopamine transporter SPECT (DaT) scan (abnormal in MSA-C)
- **Genetic tests:** (The reader is referred to the literature for a detailed discussion of genetic ataxias6,7). The patient should be appropriately counseled about the implications and costs of genetic testing before it is ordered. Testing may reveal:
  - AD mutations: SCAs (most common worldwide is SCA 3 or Machado-Joseph disease), DRPLA, and the rare episodic ataxias (EA 1, EA 2)
  - AR mutations: have usual age of onset <20 years but later onset FA, AT, AOA 2 have been reported; POLG mutations
  - X-lined mutation (premutation in the FMR1 gene): FXTAS
  - Mitochondrial DNA mutations
  - (Specialized gene tests for inborn errors of metabolism, leukodystrophies, and storage disorders should be ordered if the rest of the evaluation raises suspicion for these rare conditions)

What if no cause is identified after extensive testing?

- A large number of sporadic ataxias don't seem to have an identifiable etiology.
- When followed over time about a 1/3rd of ILOCAs may evolve to MSA-C[1]
- Unidentified genetic mutations may account for the rest of these ataxias

How are ataxias treated?

- **Specific interventions** for acquired ataxias include:
  - Steroids and other immunomodulating therapies for SREAT, paraneoplastic disorders, and other immunological disorders
  - Treatment of underlying malignancy when detected
  - Gluten-free diet for GA
  - Acetazolamide for EA2, SCA 6
  - Varenicline (Chantix®) for SCA 3[7]
  - Bile acid replacement for CTX
  - Commonsensical measures: elimination of toxins; correction of deficiency states; and, treatment of medical disorders causing ataxia
- N**on-specific pharmacological** agents of potential benefit:
  - Amantadine, alpha-lipoic acid, buspirone, branched-chain amino acids, creatine, coenzyme Q10, vitamin E, physostigmine, riluzole, and selective serotonin reuptake inhibitors

- Cerebellar tremor may improve with primidone and antiepileptics; oscillopsia with memantine and GABA agonists; spasticity with central anti-spasticity drugs.
- **Multidisciplinary approach** is necessary in MSA-C due a multitude of progressive motor and non-motor symptoms.
- **Rehabilitative therapies:**
  - should be offered to all patients with ataxia
  - Continuous exercise programs have shown positive results[8]

Can you summarize the above discussion?

- The diagnostic approach to adult onset ataxias should be systematic and guided by the history and examination.
- Non-genetic ataxias may involve an extensive and expensive evaluation which may be done in a tiered fashion.
- MSA-C is the most common sporadic ataxia. ILOCA is a diagnosis of exclusion.
- A positive familial history signals a genetic disorder. Patients undergoing genetic tests should be appropriately counseled.
- Effective management of ataxic disorders requires a multidisciplinary approach involving disease-specific and symptomatic drug treatment as well as rehabilitative measures.

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Bylaw changes

To be voted on during the TNS Business Meeting (see page 4).

Constitution-Article VI- Board of Directors (Trustees)

-This Board shall consist of the President, the President-Elect, the Vice President, the Secretary/Treasurer, the Immediate Past-President, ~~three~~ four Members-At-Large of the Society, the Education Committee Chair, the Delegate to the Texas Medical Association (TMA), the Editor of the newsletter (*Broca's Area*), and the Program Directors of the two annual meetings. The Board may extend non-voting, ex-officio board membership status to certain members as it deems advisable. Qualifications for office and procedures for nomination, election and removal from office shall be stipulated in the By-Laws.

Article 1-Membership-Section 1-Classification

-**ASSOCIATE** - Associate membership shall be open to 1) physicians partially trained in and/or practicing clinical Neurology who are not eligible for Active membership, 2) physicians practicing in clinical fields related to Neurology, ~~3) persons including physicians and holders of an advanced degree practicing or engaged in non-clinical fields relating to Neurology, 4~~ 3) Active members who move out of the state of Texas. It shall be the duty of the Board of Directors to consider these applications, seeking additional information if necessary. [note – raise dues from $15 to $75]

~~-**AFFILIATE** – Affiliate membership shall be open to non-physicians or advanced degree holders who practice or are engaged in clinical and non-clinical fields relating to neurology or individuals with undergraduate degrees and a minimum of three years work experience in clinical neurology and/or neuroscience research. Neurology professionals include and are limited to doctors of philosophy, nurse practitioners, physical therapists, physician assistants, registered nurses, and others with special qualifications as determined by and at the discretion of the Board of Directors and upon recommendation from the Membership Committee.~~

-**RESIDENT** - Resident Membership shall be open to all residents and fellows in an approved Neurology Residency training program of the State of Texas. Application shall be made in the usual manner and shall be accompanied by proof that the applicant is in fact currently in such a training program as described above. Resident members may apply for elevation to Active status with the Society upon the completion of their training program and their meeting the above requirements for Active membership. Resident status is discontinued after completion of the Residency, dismissal from the Residency training program, or changing to a Residency Program not located in the State of Texas. Resident members have no vote in Society matters and cannot hold Society office. Resident Members are the invited guests of the Society to all Society functions including luncheon and dinner meetings at no cost to themselves.

-**PROVISIONAL** - (Active, ~~Associate~~ Allied Health Professional or Resident)- An applicant for Active, ~~Associate~~ Allied Health Professional or Resident membership shall be considered a Provisional Member in the appropriate category upon receipt of a completed membership application, payment of appropriate fee, and approval by the Board of Directors. A Provisional Member shall remain in this category until approved by a majority vote of the general membership at which time the status shall change to that of an active member of the particular category and with the attending privileges and restrictions.

-**Allied Health Professional** – this category is open to physician assistants who are licensed to practice in Texas by the Texas Medical Board and to Nurse Practitioners and Registered Nurses who are licensed to practice in Texas by the Texas Board of Nursing. Applicants must be actively engaged in neurologic patient care and/or research. Applicants must show proof of licensure. It shall be the duty of the Board of Directors to consider these applications, seeking additional information if necessary. [note – dues to be $75]

Article II- Officers- Section 3-Nomination- Subsection D

-**Qualifications:** Any member nominated for consideration shall be in good standing in the Society. While no absolute requirements exist for any post, the nominating committee shall consider factors such as duration of service (3 years for President-elect and at-large board members), balance on the board of directors (such as geographic, age, gender, sub-specialty qualifications) and the opportunity to increase the number and commitment of the members serving the Society. In general, it may be desirable to consider qualifications such as prior service to the Society and demonstrated leadership in the Confederation of Medicine as a requisite for nomination to the post of President-elect. ~~Similarly, given the historic oversight by the vice-President of the granting of awards for research, a nominee for this post should have a suitable background in neurological research.~~

Article III-Duties and Terms of Office- Section 1-Duties-Subsection J

~~**-HISTORIAN** - A Historian shall be appointed annually by the President with the approval of the Board of Directors. He/she shall maintain a proper historical record of the Society, and will sit on the Board of Directors as an ex officio member.~~

Article IV-Committees-Section1-Standing Committees- Subsection B

~~**-COMMITTEE ON ACCESS TO HEALTH CARE**~~

~~1) This Committee shall be composed of those members appointed by the President in consultation with the Board of Directors. It will include a chairperson, and at least two other physicians who are members in good standing. These two members shall serve for alternating two-year terms.~~

~~2) The Committee shall keep the Board apprised of such developments relating to the practice of Neurology as the Board requires and shall convey such Board actions to other entities as are assigned by the Board. The Committee shall work with the Texas Medical Association and with the member physicians of both organizations to improve the accessibility of care. The Committee shall provide peer review of cases that involve neurological issues, filed or threatened against member physicians of the Texas Medical Association and/or the Texas Neurological Society.~~

~~3) Peer review may be initiated by request of the President of the Texas Medical Association or the Texas State Board of Medical Examiners. It can also be initiated solely by written request of the defendant physician. The Committee may also review testimony of expert witnesses.~~

~~4) The peer review shall be completed by the Committee within thirty days of receipt of records submitted for review. The Committee shall report its findings to the President of the Texas Neurological Society.~~

~~5) The requesting physician shall indicate a preference whether the decision of the Committee shall be provided orally or by written communication to the physician.~~

~~6) The Committee shall conduct this peer review process for the determination of outcome, with no compensation provided to the members of the Committee.~~

~~7) The chairperson of the committee will be expected to, as an ex-officio board member, report to the Board or assure that another member of the committee do so.~~

~~**C. DRIVING SAFETY COMMITTEE**~~

~~1) This committee shall be composed of those members appointed by the President in consultation with the Board of Directors.~~

~~2) The committee shall keep the Board apprised of concerns relating to Driving Safety and shall undertake such actions as the Board directs.~~

~~**D. BYLAWS COMMITTEE**~~

~~1) This committee shall be composed of those members appointed by the President in consultation with the Board of Directors.~~

Article IV-Committee-Section 1-Standing Committees-Subsection E (3)

-The Committee shall facilitate the Society's educational opportunities ~~for primary care physicians, other physician groups, and for lay audiences, as well as the annual scientific session and other meetings with educational content~~ as deemed necessary by the Board.

American Academy of Neurology Update

Looking for tailored recommendations on the best tools and resources? Learning Across Your Lifetime on www.aan.com offers recommendations based upon your interests and career stage.

Here is a sampling to explore:

- **NeuroTracker:** Track your MOC and professional activities in this member only tool. Update your member profile and receive personalized resource recommendations. http://www.aan.com/go/education/neurotracker
- **NeuroPI:** Take the guesswork out of the MOC PIP requirement with this convenient online program incorporating both clinical and feedback module resources. Coming soon: Acute Stroke. www.aan.com/view/neuropi
- **NeuroSAE:** Earn Self-Assessment CME credit while evaluating topical strengths and weaknesses. Allow NeuroSAE to assist you in building a robust learning plan. Fifth Edition now available. www.aan.com/neurosae
- **NeuroLearn:** Learn when you want wherever you are with an internet connection with the Academy's new online CME program. Coming soon: The Brachial Plexus: Anatomy and Clinical Applications. www.aan.com/view/neurolearn
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Contact memberservices@aan.com for more information.

Advocate for Neurology in Washington, DC
The 2013 Neurology on the Hill will take place April 22–23, at the Ritz-Carlton Pentagon City. Participants are flown to Washington, DC, to put a face on the challenges of people with neurologic disorders and the physicians who treat them. For many AAN members, Neurology on the Hill is their first hands-on experience with the political process. Apply today on the AAN website.

AAN Attending TNS Winter Conference
AAN staff will be in attendance at the TNS Winter Conference to answer questions and obtain member feedback. Stop by our booth to discuss your membership and find tools and resources on important neurological issues. Contact dshowers@aan.com for more information.

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