TEXAS NEUROLOGICAL SOCIETY SUMMER 2023

Broca's Area

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Reeta Achari, MD
TNS President

President's Message

I have the great privilege to be President of this great society as it celebrates its 50th year in February 2024. In reviewing the documents of incorporation, I am reminded that the ideas and courage of a few brilliant people effect so many over time. It's a very Texan thing to start with a great idea and then put it into action. From mending hearts to walking on the moon, Texas has played a pivotal role in many landmarks of innovation. Texas is also rich in cultural diversity, with settlers past and present coming from all over the globe, bringing with them fresh perspectives and skills. It is with these things in mind that I write this message to you.

The only constant is change. This may be frustrating and challenging but is nonetheless true. We have just come to the other side of a global pandemic that took many lives but could have taken many more were it not for the rapid innovation and exchange of data facilitated by information technology. Physicians adapted treatments to the needs of patients as information became available in real time and saved countless lives. We learned to deliver care remotely and telemedicine has become an integral part of medical care. No doubt limitations exit, but innovation will improve its use and provide ever better information for diagnosis and treatment.

As with all innovation, some elements have worked well while others have not fulfilled their promise. The electronic health record was designed primarily for billing efficiency but has now become ubiquitous and has taken physicians away from patients and time away from patient care. In an effort to eliminate supposedly illegible notes, the EHR has created note bloat, with meaningless and oft times inaccurate information cluttering patient charts. The promise of universally available patient information remains unfulfilled.

The new frontier of artificial intelligence looms over us. With proper algorithms, AI will play a helpful role in many aspects of medicine. But it has a very long way to go before it can understand the nuances of history-taking and the keen observational skills required by neurologists. Nor is AI ready to decipher and decode the multiple cultures which physicians deal with daily, whether with patients or colleagues in other specialties.

Into this mix, we must also consider the survival of clinical practice. Over the last 30 years, the practice of medicine has undergone a drastic change from independent physicians, to employed models, both hospital and academic. Most recently, venture capital firms are inserting themselves in the practice of medicine, threatening to further erode the patient-physician relationship as the foundation of care. Innovative and alternative models which keep physicians independent and our patients the focus will be needed. Many physicians re already going back to the past traditions of direct payment to provide excellent care for patients without the limitations imposed by insurance companies.

Tradition is important. Old does not mean outdated. We cannot abandon the strengths of the neurological examination and localization. Rather, we need to adapt and innovate. Past and future must be bridged. Senior neurologists must interact with each generation so that what was most valuable for patient care is carried forward into a new and exciting future. I hope that this will be the start of conversations about where we have been and where we are heading.

It's an exciting time to be a neurologist, as I'm sure it was 50 years ago.

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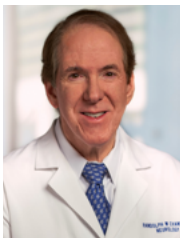

Editor's Notes

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 Annual Summer Conference at La Cantera, San Antonio. Rebecca Romero, program director, Erin Furr-Stimming, committee chair, and the education committee have planned an excellent program.

THE HOFFMAN-TINEL SIGN OR TROTTER-DAVIES-HOFFMAN-TINEL SIGN AND CARPAL TUNNEL SYNDROME

Rather than "Tinel's sign," should we more accurately use the eponym "Hoffman-Tinel sign"[1] or perhaps Trotter-Davies-Hoffman-Tinel sign? In March, 1915, the German, Paul Hoffman (1884-1962), reported "light percussion of a finger during extension as a diagnostic maneuver produces a pins and needles feeling." In October, 1915, the Frenchman, Jules Tinel (1879-1952), described it as "application of pressure to an injured nerve trunk induces a sensation of tingling." Both were using percussion to examine soldiers with injured peripheral nerves at the front lines during World War I looking for evidence of regeneration.

In 1909, Trotter and Davies reported that sensations elicited distal to the point of nerve resection are referred to the area or point of nerve resection but did not discuss the clinical relevance.[2]

Tinel has gotten credit perhaps because Hoffman's publication was not as well known or complete as Tinel's or perhaps because Germany lost the Great War.

During World War II, Tinel was active in the French Resistance. He was imprisoned for hiding allied airmen with his son who died in a concentration camp.

Jules Tinel (1879-1952) from Wikipedia

CARPAL TUNNEL SYNDROME AND GEORGE PHALEN

Carpal tunnel syndrome was first described with 2 cases, one post-traumatic and one idiopathic, by James Paget in 1854.[3] He recommended a splint for treatment.

Orthopedist and hand surgeon, George Phalen, helped to define and popularize idiopathic carpal tunnel syndrome [4] and is remembered for his sign. In a 1951 article of 11 cases of carpal tunnel syndrome, he reported the use of Tinel's sign to diagnose this compression neuropathy and also reported, "… the numbness and paresthesias in the fingers of these patients could be increased by sharply flexing the wrist for a period of 60 seconds."[5] Phalen practiced in Dallas from 1970 until he retired in 1980.

The first EMG was reported in 1956.[6]

For carpal tunnel syndrome, Tinel's sign has a sensitivity of 0.47 and a specificity of 0.56.[7]

COUGH HEADACHE

In 1932, Tinel described 4 patients with headache brought on by coughing, nose-blowing, breath-holding, and bending the head forward.[8] They are not called Tinel's headaches. I present 4 of my own cases.

Case 1: An 18 year old male with headaches with bench press or squats

This is an 18 year old male with a 3-4 week history of headaches occurring with working out 3 out of 5 times going to the gym per week with bench press or squats (once with pushups) immediately when he gets a bilateral occipital and sometimes all over throbbing with an intensity of 7-8/10 at onset. When he stops, the pain decreased to an intensity of 3/10 after about 20-30" but takes up to 14 hours to resolve. Ibuprofen decreases the intensity. There is no associated nausea, light or noise sensitivity. If he jogs or walks first, he may not get a headache or the headache may not be as intense. He has no headaches with coughing, sneezing, bending over, or straining for a bowel movement.

- Past medical history was negative.
- Neurological exam was normal
- MRI of the brain without and with contrast was negative.

Question—Is this a weightlifter's headache, a cough headache, or an exertional headache?

Discussion--Weightlifting headache is a headache triggered by a Valsalva maneuver and is classified by ICHD-3 as a primary cough if secondary causes are excluded. Exertional headache is brought on by and occurring during or after more sustained physical exertion especially in hot weather or at high altitude.

The ICHD criteria are as follows:

"4.1 Primary cough headache
Previously used terms: Benign cough headache; Valsalva manoeuvre headache.

Description:
Headache precipitated by coughing or other Valsalva (straining) manoeuvre, but not by prolonged physical exercise, in the absence of any intracranial disorder.

Diagnostic criteria:
A. At least two headache episodes fulfilling criteria B–D
B. Brought on by and occurring only in association with coughing, straining and/or other Valsalva manoeuvre[1]
C. Sudden onset[2]
D. Lasting between one second and two hours
E. Not better accounted for by another ICHD-3 diagnosis.[3]

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Editor's Notes *(continued)*

Notes:

1. Headache arises moments after the cough or other stimulus.
2. Headache reaches its peak almost immediately, and then subsides over several seconds to a few minutes (although some patients experience mild to moderate headache for two hours).
3. The syndrome of cough headache is symptomatic in about 40% of cases, and the majority of patients in whom this is so have Arnold–Chiari malformation type I. Other reported causes include spontaneous intracranial hypotension, carotid or vertebrobasilar diseases, middle cranial fossa or posterior fossa tumours, midbrain cyst, basilar impression, platybasia, subdural haematoma, cerebral aneurysms and reversible cerebral vasoconstriction syndrome. Diagnostic neuroimaging plays an important role in the search for possible intracranial lesions or abnormalities. Since subtentorial tumours accounted for more than 50% of intracranial space-occupying lesions in children, cough headache in paediatric patients should be considered symptomatic until proved otherwise.

Comments:

4.1 Primary cough headache is a rare condition, accounting for 1% or fewer of all headache patients consulting neurological clinics. However, one report found one-fifth of patients with cough seen in a chest medicine clinic had cough headache.

4.1 Primary cough headache is usually bilateral and posterior, and predominantly affects patients older than 40 years of age. There is a significant correlation between the frequency of the cough and the severity of the headache. Associated symptoms such as vertigo, nausea and sleep abnormality have been reported by up to two-thirds of patients with 4.1 Primary cough headache.

While indomethacin (50–200 mg/day) is usually effective in treating 4.1 Primary cough headache, a few symptomatic cases have been reported to respond to this treatment."[9]

The MRI of the brain without and with contrast was normal.

The headache best fits primary cough headache except for duration which is more than 2 hours.

In the largest series of 74 consecutive patients with primary cough headache, 73% were male. The headache intensity was usually severe with a duration of less than one minute in 62% and more than 30 minutes in 10.8% with the longest 2 hours.[10] Heavy lifting was a trigger for 14.9%. In the 4 other smaller series, the duration was seconds to less than 30 minutes. [11]

The location is often bilateral and occipital but may be frontal, temporal, parietal, vertex, the entire head, and unilateral in 33% of cases.[10] The pain is often explosive or dull but may be pulsatile, sharp, or stabbing. Associated symptoms were uncommonly associated as follows: nausea, 9.5%; vomiting 1.4%; photophobia, 5.4%; and phonophobia, 10.8%. Most patients have more than one trigger.

In a series of 9 patients with secondary cough headache, the headaches were usually severe with a usually bilateral occipital location (other locations possible) with a duration of 10 second to 30 minutes. Nausea and vomiting were present in 11.1%.[10]

Probable primary cough headache is the best diagnosis for this case. He did not keep his follow-up appointment.

Case 2: A 17 year old male with headaches triggered by weightlifting, hard coughing, or sneezing.

"This is a 17-year-old male with a 2-year history of headaches occurring when weightlifting, hard coughing, or sneezing described as a severe generalized pounding lasting about 10-15 seconds followed by an aching for about an hour without associated symptoms."[12]

Questions—What type of headache does he have? What is the etiology?

Discussion-- Again, he fits the criteria for primary cough headache if secondary causes are excluded.

The MRI of the brain without and with contrast and cervical spine showed 10 mm of tonsillar ectopia with peg-like tonsils and diminished cerebrospinal fluid space in the region of the foramen magnum. The headaches resolved after posterior fossa decompression.

The most common secondary cause of cough headache is Chiari type 1 malformation. Other secondary causes include the following: headache secondary to spontaneous intracranial hypotension; middle cranial fossa or posterior fossa meningiomas; posterior fossa dermoid tumor; medulloblastoma; pinealoma; chromophobe adenoma; brain metastasis or metastases; brain tumor not otherwise specified; midbrain cyst; posterior fossa arachnoid cyst; basilar impression; platybasia; os odontoideum; subdural hematoma; and acute sphenoid sinusitis.

Symptoms from CM1 usually start in the second or third decade of life. In a series of 201 patients with CM1, 13.4% of headaches had onset only during Valsalva maneuver, effort, cough, sneezing, and laughing.[13] Of the entire cohort, the headaches were suboccipital occipital in 29%, diffuse/nonpulsating in 74%, and pulsating in 23% and were worsened by Valsalva maneuver in 85%. Thirty-four percentage had short-lasting headaches and 41% had headaches lasting 3 hours to 3 days. The headaches were severe in 42%, moderate to severe in 50%, and mild in 7%. Migrainous-associated symptoms were reported by 38%. In symptomatic patients who do not have surgery, about 40% of those with cough headaches improved at follow-up.[14]

When a first-time thunderclap weightlifting headache occurs, subarachnoid hemorrhage should certainly be excluded as a cause.[15] Weightlifting can also trigger cervicogenic headache and migraine in some individuals.[16]

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Editor's Notes *(continued)*

Case 3.

"This is a 66 year old white male with occasional mild headaches in the past. He presented with a four month history of headaches occurring one to four times per day, brought on by having a bowel movement, stooping, or getting up from a sitting position. He did not know if the headache was triggered by coughing because he had not coughed at all. The headache was a bifrontal and bitemporal sharp, aching pain with a 7/10 intensity and occasionally a 9/10 intensity with an average duration of one minute and a range of 30 seconds to one hour. About 20 of the headaches had lasted more than one minute, with most in a range of one to two minutes. He had tried ibuprofen and acetaminophen with questionable help. For the prior five days, he had increased his dose of aspirin from 81 mg a day to 325 mg a day. The headache was then different with a constant bifrontotemporal pressure with an intensity of 1/10 but he had not had the brief headaches with activity exacerbation. He had a CT scan of the sinuses on 06/14/04 with essentially negative findings."

There was a past medical history of insulin dependent diabetes with sensory neuropathy and hypertension. Neurological examination was normal except for diminished pinprick distally of both lower extremities and absent deep tendon reflexes diffusely."[17]

Question-What is the diagnosis?

Discussion—The headaches are suggestive of cough headaches.

A MRI scan of the brain was normal except for smooth diffuse dural enhancement around both cerebral convexities and, to a milder degree, in the posterior fossa. A MRI scan of the cervical, thoracic, and lumbar spine revealed degenerative changes but no evidence of extra-arachnoid fluid collections, extradural extravasation of fluid or meningeal diverticula.

A lumbar puncture produced an opening pressure of 11 cm of water. Cerebrospinal fluid analysis was normal except for a protein of 109 mg/dl.

This headache was due to spontaneous intracranial hypotension.

Case 4. A 72 year old female with headaches with sneezing, coughing, or bending over

This is a 72 year old female with a 2 month history of headaches only with sneezing, coughing, or bending over described as all around the head pressure, aching and throbbing with an intensity of 9/10 without nausea, light or noise sensitivity with a duration of 5 seconds. The frequency depends upon how often she does a trigger activity. She has no other headaches.

- Past medical history of hypertension on losartan.
- Neurological exam was normal.
- She declined topiramate
- MRI wwo normal

Questions—What treatments might be effective? What is the prognosis?

Discussion—She fits the criteria for primary cough headache. Treatments which may be effective include indomethacin (25 mg tid titrating up to 50 mg tid as tolerated if needed), acetazolamide, topiramate, naproxen, propranolol, IV DHE, and lumbar puncture with removal of 40 ml of CSF.[18]

In a series of 83 patients, 84% had remission at a mean of 51 months.[10]

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TNS Legislative Update

Dr. Sara Austin, TNS Legislative Chair and Tom Holloway, TNS Lobbyist

TEXAS NEUROLOGY DAY AT THE TEXAS CAPITOL

For the first time, the Texas Neurology Society hosted its very own "Texas Neurology Day" at the Texas Capitol. Neurologists from across Texas at the Texas Medical Association for an issue briefing before heading up to the Capitol to speak with lawmakers and their staffs regarding our legislative agenda. Later, the group was also recognized by Senator Charles Schwertner, MD (R-Georgetown) with a formal resolution in the Texas Senate.

Thanks to everyone who participated for helping to make Texas Neurology Day such a success!

SCHOOL SEIZURE ACTION PLAN (SB 1506)

SB 1506 by Senator Bryan Hughes (R-Mineola) and Representative Travis Clardy (R-Nacogdoches) was a TNS priority bill to create a simple, standardized form for students with epilepsy or a history of seizures which outlines important information like triggering stimuli, relevant medications, basic care instructions, and contact information for the child's parent's and treating physician. The legislation builds upon "Sam's Law" (passed by Sen. Hughes & Rep. Clardy in 2021), which requires schools to keep a "Seizure Action Plan" on file with the school nurse for any student with epilepsy or a history of seizures.

This session, TNS' own Dr. Gary Clark and Dr. Daniel Freeman both came to the capitol to testify in support of SB 1506 on behalf of the Texas Neurological Society. Both did a fantastic job of answering lawmakers' questions and addressing the need for standardization in school seizure action plans. The legislation was ultimately passed with overwhelming support in the House and Senate and was signed into law by Governor Abbott on May 27.

BRAIN INSTITUTE OF TEXAS (HB 15/HJR 5)

HB 15 by Representative Senfronia Thompson (D-Houston) would create the Brain Institute of Texas to fund brain research and develop lifesaving medical treatments at Texas institutions of higher education. If passed and ultimately approved by Texas voters, HJR 5 would also dedicate $3 billion over the next 10 years to help Texas universities and medical schools conduct cutting-edge medical research into common neurodegenerative diseases like ALS, Parkinson's disease, and Alzheimer's.

This is the second session TNS has worked with a broad coalition of medical practitioner groups, hospitals, and other stakeholders to get HB 15 to the Governor's desk. Unfortunately, after a strong favorable vote in the Texas House of Representatives, Lt. Governor Dan Patrick refused to refer the bill for further action in the Senate, and the bill ultimately died. Hopefully, the third time is a charm, as we hope to support the creation of the Brain Institute of Texas again in 2025.

EXPANSION OF MEDICAL CANNABIS (HB 1805)

HB 1805 by Representative Stephanie Klick (R-Fort Worth) expands the list of qualifying conditions for the Texas Compassionate Use Program (TCUP), specifically allowing physicians to prescribe low-THC medical cannabis to patients experiencing chronic pain (for which a physician might otherwise prescribe an opioid) and other debilitating medical conditions designated by the Department of State Health Services. The bill would also change the 1% THC by weight limitation to a 10mg of THC per dosage unit.

Like HB 15, Representative Klick's TCUP expansion bill received broad, bipartisan support in the Texas House of Representatives (127-19) before heading to the Texas Senate where Lt. Governor Patrick refused to call the bill for a vote. We expect to continue working with Representative Klick to advance similar legislation next session.

MOBILE STROKE UNITS (HB 2356)

HB 2356 by Representative Ann Johnson creates a new statewide grant program to provide funding for specially equipped ambulances, known as "Mobile Stroke Units," which are capable of treating stroke patients on-scene. These Mobile Stroke Units allow for the rapid diagnosis and treatment of the most common varieties of stroke within minutes of initial contact with a patient, saving valuable time, improving outcomes, and preserving physical function.

TNS member Dr. James Grotta has been the driving force behind bringing the first mobile stroke units to Texas and testified in favor of HB 2356 before the House Committee on Public Health. Unfortunately, the bill was heard late in the session and ultimately failed to receive a vote in the Texas House before the bill deadline. This is another priority TNS plans to pursue when the Texas Legislature reconvenes in 2025.

SCOPE OF PRACTICE

Sometimes, a successful legislative session is defined by the things that don't become law…like troubling scope of practice expansion offered by midlevel practitioner groups. This session, the TNS lobby team worked with the Texas Medical Association and a host of other physician specialty groups to successfully defeat dozens of scope of practice bills pushed by advanced practice nurses, physical therapists, physician assistants, phycologists, podiatrists, chiropractors, acupuncturists and more.

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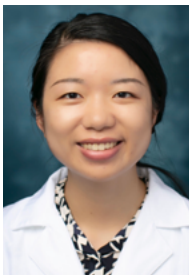


A Sporadic Creutzfeldt-Jakob Disease Case Presenting as Rapidly Progressive Ataxia

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DECLARATION OF INTEREST STATEMENT

Yuanyuan Tan M.D., Jannatul Ferdous M.D., and Katie Hendley M.D report there are no potential competing interest to declare. Written consent form for publication was obtained from patient's family.

ABSTRACT

Sporadic Creutzfeldt-Jakob Disease (sCJD) is the most common form of prion disease and has been closely linked with rapidly progressive dementia. With updated research in animal model phenotypes and the development of real-time quaking-induced conversion (RT-QuIC), any rapid neuropsychiatric deterioration plus positive RT-QuIC would suffice for diagnosis of probable sCJD. For this article, we introduce a 56-year-old female with initial presentation of rapid progressive ataxia and preserved cognition diagnosed with sCJD by RT-QuIC. In our case elevation and fluctuation of multiple autoimmune markers were noted. Early diagnosis of sCJD can help relieve the mental burden of patients and families and avoid unnecessary treatment.

INTRODUCTION

Sporadic Creutzfeldt-Jakob Disease (sCJD) is the most common form of prion disease and has been closely linked to rapidly progressive dementia. The 1998 World Health Organization[1], and the 2007 University of California San Francisco criteria[2] both required rapid cognitive decline as a prerequisite for probable sCJD. Since the development of real-time quaking-induced conversion (RT-QuIC) assays in 2010 and its proved high specificity to sCJD[3], the updated 2018 CDC guideline[4] and 2017 international sCJD surveillance[5] broadened the required clinical manifestation from cognitive decline to any progressive neurological syndrome if RT-QuIC is positive. In 2010, 6 sCJD molecular subtypes were identified in mice models based on the amino acid mutation and cleavage site of the prion protein, and some subtypes had initial presentations other than progressive cognitive changes[6]. These updates have greatly increased the sensitivity of the diagnostic process of sCJD. Hereby we introduce a case of a 56-year-old female diagnosed with sCJD presenting progressive ataxia with preserved cognition.

CASE SUMMARY

A 56-year-old Caucasian female started to feel numbness and tingling in her right toe after an injection of vitamin B12. The abnormal sensation ascended to her leg and face, and she felt right-sided weakness shortly after. Involuntary jerking movements of the right arm were also noticed. Symptoms were progressively worse for three months. She gradually lost the ability to ambulate. Three unintentional falls at home prompted her visit to the emergency department. Other complaints include six months of diarrhea, worsening depression and anxiety, tremors, and three days of bilateral blurry and double vision. No report of fever, chills, loss of consciousness, neck stiffness, dyspnea, and tick/insect bite. The patient was a fitness trainer and had no major health conditions except pernicious anemia. The patient had no family history of neurological disease. She could give a full account of her history on admission. Physical exam showed intact mental status, torsional nystagmus in horizontal gaze, opposite-beating nystagmus in vertical gaze, right hemiparesis, right arm spasticity with Hoffmann sign and dystonic hand posture, severe midline and right appendicular ataxia, brisk bilateral patellar reflexes with positive Babinski signs. The lab results were as follows: albumin-cytologic dissociation with positive oligoclonal bands in CSF, negative CSF autoimmune and paraneoplastic panel; elevated serum thyroid peroxidase antibody (TPO Ab) 38 IU/ml with normal TSH and fT4 levels; positive ANA titer and SSA Ab; actin Ab 109 U; GAD65 Ab 137 units/ml in serum but undetectable in CSF; elevated NSE, elevated West Nile IgG but normal IgM in CSF. Other labs were all normal including T. whipplei PCR, ACE meningitis/encephalitis panel in CSF; vitamin levels; celiac disease panel; ceruloplasmin; heavy metals; ANCA; rheumatoid factor; SSB Ab. CSF pathology showed mild lymphocytosis. MRI on admission showed DWI hyperintensities at the left frontal and parasagittal cortex (Figure 1A&B, a finding read by a radiologist as a possible artifact). Serial MRI (Figure 1) demonstrated progressive cortical ribboning in left hemisphere and eventual involvement of right hemisphere and bilateral caudate nucleus. No tumor was found in whole-body CT except an incidental thyroid mass and biopsy showed benign follicular cells. EEG (Figure 2) revealed mild slowing in the left temporal area but no epileptiform discharges at hospitalization week one but progressed to diffuse slowing at week four, and spikes and waves in the right hemisphere at week five.

While awaiting send out lab results, Intravenous Immunoglobulin, high-dose thiamine, two courses of high-dose steroids, and plasmapheresis were given and provided no benefit. The patient developed worsening dysarthria, with more frequent myoclonic jerks and worsening muscle spasms partially responded to Keppra and diazepam. A 5min myoclonic jerking occurred shortly after the third plasmapheresis. Despite increased Keppra, the patient continued to have whole body stiffening with upper extremities flexed at elbows and wrists, straightened legs, and jerking of extremities aggravated by sounds, light touch, and emotion. CSF RT QuIC returned positive with total tau protein 18060 pg/ml and 14-3-3 protein 77478 AU/

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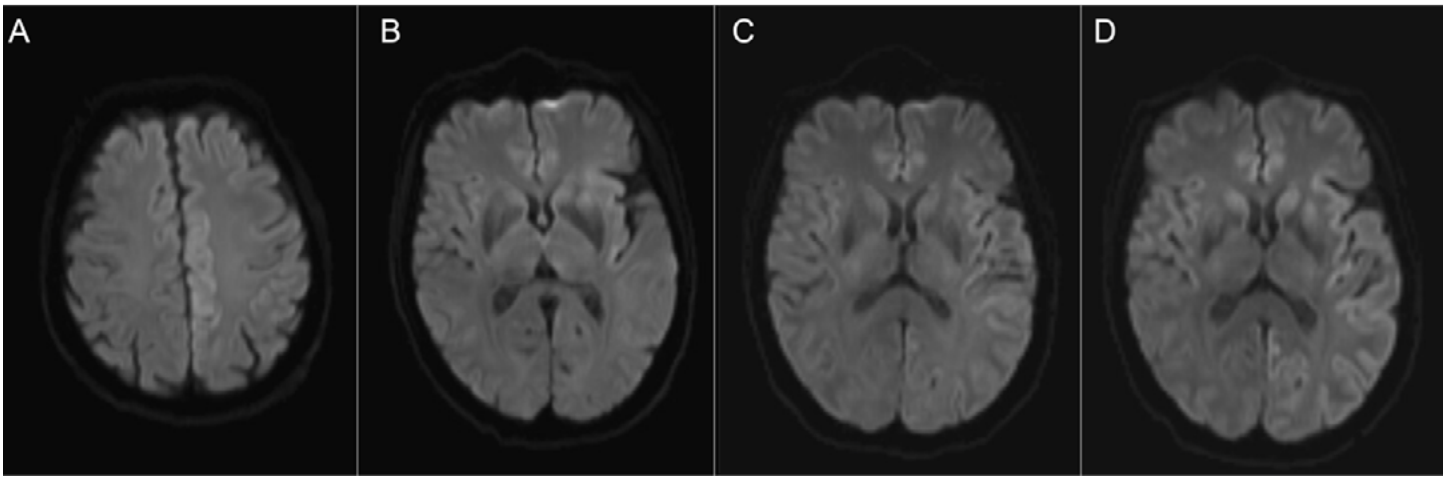

Figure 1: Progressive cortical ribboning: Parasagittal(A) and mild left insular(B) cortical ribboning on hospital day 2. Enlarged left hemisphere and mild right temporal-parietal involvement are shown on hospital day 21(C). Caudate DWI hyperintensities and more prominent right hemisphere cortical ribboning are noted on hospital day 32.

ml. Palliative care was pursued. Patient deceased one week after the diagnosis of sCJD. Autopsy confirmed sporadic CJD MM1 subtype.

DISCUSSION

Our case suggests any rapidly progressive neurological decline without a clear initial diagnosis should raise the concern for sCJD and warrant a CSF RT-Quic assay, as the combination of the two suffices the diagnosis of probably sCJD. Clinical manifestation of sCJD is no longer limited to rapidly progressive dementia but instead is broadened to any neuropsychiatric deterioration. Our patient was initially considered a possible VV2 subtype, which is the second most common phenotype presented as progressive ataxia, according to animal models[6]. Our case also showed intriguing elevations of multiple autoimmune markers. The incidental thyroid mass and elevated TPO Ab initially raised the concern of Hashimoto encephalopathy. Positive CSF oligoclonal bands and elevated protein are more common findings in multiple sclerosis. Elevated GAD65 Ab can be seen in stiff-person syndrome which has similar presentations to sCJD. An autoimmune diagnosis was favored in the beginning due to the history of pernicious anemia and before mentioned labs. With a closer look, it is noted that the levels of the autoimmune antibodies were never too high and had fluctuations. ANA was negative on admission and later became positive. TPO Ab was negative at first and then became elevated for 2 weeks and ultimately down to normal. Such correlation between sCJD and autoimmune markers was never depicted in previously published literature. Apart from the new findings, our case supported some well-established facts about sCJD: DWI cortical ribboning has high sensitivity for sCJD, nonetheless often being missed or misinterpreted; serial EEGs are important as epileptic spikes may not show in the early phase.

As sCJD is a rare and incurable disease, although a timely diagnosis could not offer earlier treatment for a treatable cause, it could possibly relieve the anxiety of patients and families as suffering from "a progressive unknown condition" often takes a great toll on their mentality. Unnecessary medical treatments can be saved, and comfort care can be pursued earlier.

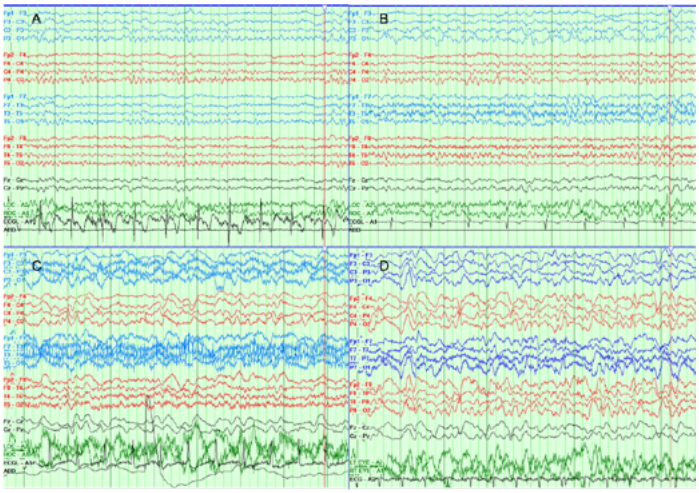

Figure 2: Progressive EEG changes. Mild focal slowing of left temporal region on hospital day 7(A), increased focal slowing of the left temporal region on hospital day 11(B), diffuse slowing on hospital day 27(C), and spikes and waves forms the right hemisphere on hospital day 33(D).

All authors have approved the version to be submitted.

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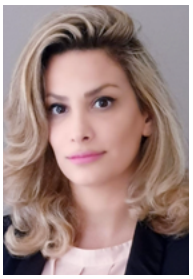
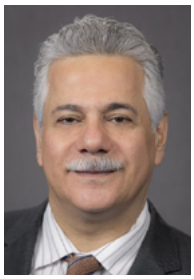



Multiple myeloma and myopathy

Saja Asaad, MD and Aziz Shaibani, MD

Nerve and Muscle Center of Texas

A 54-year-old healthy physician presented with generalized fatigue, muscle pain and weakness started 2 weeks after a mild COVID 19 infection. She developed difficulty swallowing liquids and solids, shortness of breath on exertion and difficulty climbing stair. She reported no fever, speech difficulty, skin rash or chewing fatigability. She lost 10 pounds.

Examination revealed 4/5 strength of the proximal arms and legs muscles and 5/5 distal strength. She had normal neurological examination otherwise.

Laboratory values: CK level was 180 IU/Liter. SR was 85 mm/hour. Serum Calcium was 11.5 mg/dl. IFPE revealed monoclonal gammopathy of IgD type.

Electrodiagnostic study: normal sensory and motor nerve conduction study in the ams and legs. Needle EMG revealed 30% short duration polyphasic early recruited motor unit potentials in the iliopsoas, deltoids and biceps muscles with no fibrillation. Thoracic paraspinal muscles were normal.

The pain and stiffness improved with prednisone 40 mg a day.

Because of hypercalcemia, elevated SR and monoclonal protein, she had skeletal survey which showed several lytic bone lesions. Bone marrow aspiration revealed 25% atypical plasma cells.

Abdominal fat aspiration: negative Congo red staining for amyloid.

Due to the proximal weakness, she was referred for a muscle biopsy.

The following are possible cause(s) of myopathy in a multiple myeloma patient:

A. Amyloid myopathy
B. Sporadic adult onset nemaline myopathy
C. Inflammatory myopathy
D. None congophilic light chain deposition myopathy
E. All of the above

The correct answer is E

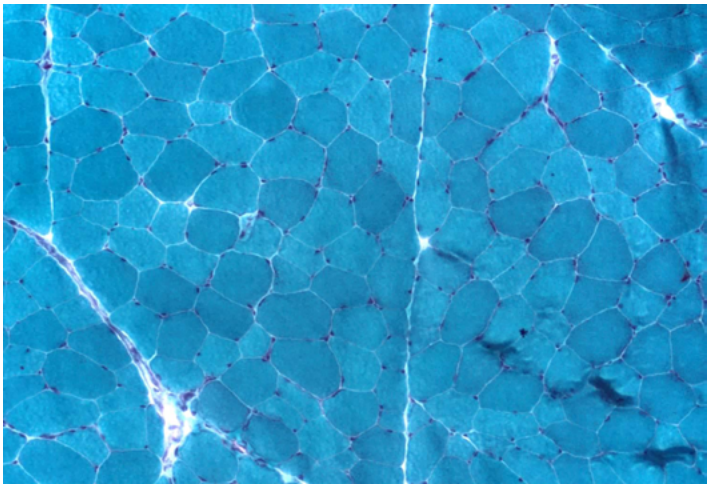
Left biceps muscle biopsy revealed finding shown in the following photos:

WHAT IS THE DIAGNOSIS:

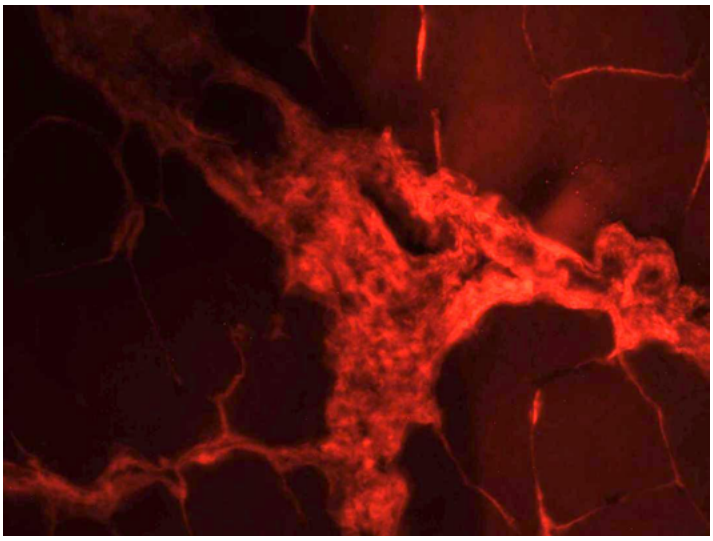
A. Amyloid myopathy
B. Sporadic adult onset nemaline myopathy
C. Inflammatory myopathy
D. None congophilic light chain deposition myopathy
E. All of the above

The correct answer is A.

The top photo : Modified Gomori Trichrome stain. 400x. normal. No Nemaline rods.



Modified Gomori Trichrome stain. 400x. normal. No Nemaline rods



Congo red-stained sections are viewed under fluorescence illumination with Texas red filtration: bright red appearance represents amyloid deposits in the endomysium and vascular wall.

The lower photo: Congo red-stained sections are viewed under fluorescence illumination with Texas red filtration: bright red appearance represents amyloid deposits in the endomysium and vascular wall.

DISCUSSION:

This is a case of amyloid myopathy as the presenting

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feature of multiple myeloma.

The preceding COVID 19 infection was incidental.

Immunoglobulin D multiple myeloma (IgD MM) accounts for almost 2% of all myeloma cases. It is associated with an increased frequency of undetectable or small monoclonal (M)-protein levels on electrophoresis; osteolytic lesions; extramedullary involvement; amyloidosis; a lambda (lambda) light chain predilection; renal failure; hypercalcemia; and, often, advanced disease at diagnosis. (1). Skeletal myopathy is a very rare extra medullary manifestation of multiple myeloma. It is usually caused by amyloid myopathy (2,5). However, sporadic late onset nemaline myopathy should also be considered (3,4). None amyloid light chain deposition myopathy (LCDM)(6), paraneoplastic polymyositis and chemotherapy induced myopathy are other possibilities.

LCDM is a category of monoclonal immunoglobulin deposition in the muscle and is characterized by tissue deposition of non-amyloid monoclonal immunoglobulin light chains in granular form, which are not stained by Congo red.

SLONM is a rare, acquired, late-onset muscle disorder with subacute progressive proximal legs and arms weakness, distal weakness , respiratory failure and dysphagia. It can be associated with a monoclonal gammopathy of unknown significance (MGUS). Histopathologically, nemaline rods can be identified on modified Gomori's trichrome staining as subsarcolemmal or intermyofibrillar dark red clusters. Using electron microscopy, the rods appear as electron dense bodies. Rods also represent the histopathological hallmark of the inherited congenital nemaline myopathies, which are currently known to be caused by 11 different genes. SLONM with MGUS is a paraneoplastic syndrome that usually responds with chemotherapy with or without autologous stem cell transplantation (7).

LESSONS FROM THIS CASE:

- Monoclonal gammopathy should always be considered seriously in particular in patients with myopathy and high serum calcium.
- Negative abdominal fat aspiration does not exclude amyloid myopathy.
- In this case, the patient was excluded from clinical trials based on muscle biopsy finding of amyloidosis.
- Response to steroids is not specific.

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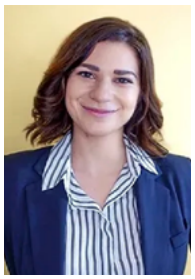
Chi-Ying R. Lin, MD, MPH


Sheng-Han Kuo, MD


Miriam King, MS

Broadway for Ataxia: a creative program originated during COVID-19 pandemic

Chi-Ying R. Lin, MD, MPH[1], Nadia Amokrane, BA[2], Miriam King, MS[2], Haidyn Emmerich[2], Sheng-Han Kuo, MD[2]*

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ABSTRACT

The cerebellar ataxia is rare compared to common neurological disorders, such as Parkinson's disease and essential tremor, and people with cerebellar ataxia is considered the minority of among the movement disorders. The unmet needs in cerebellar ataxia, including lack of awareness, decreased social connection, and reluctance to sustain physical therapy and exercise abound and continue to expand. These unmet needs, together with physical limitations and mental burden, have greatly influenced the self-perception and support systems in cerebellar ataxia. During the COVID-19 era, a theatrical art-based program delivered by performance art actors and singers in New York City was established. The curriculum design was facilitated by multidisciplinary efforts with physical therapy and speech-language training. Post-implementation, we have found camaraderie among the ataxia community, wellness of participants, and neurology trainees' training experiences have been greatly enhanced. Most importantly, the positive outcomes, including altruism, have formed a reciprocal virtual cycle. This program can thus serve as a model to combine art and music to enrich the lives of people with neurological disorders.

It is another one of those clinic visits, we had just newly diagnosed a woman with cerebellar ataxia and we reinforced the need for exercise and physical therapy for her.[1-3] She was quite honest about not being able to pursue it because "*It is just not my thing.*" She felt she needed to cut down the family and social gatherings because of progressive speech and walking difficulty. She felt "*the spice of life is just not the same as before*."

It is estimated that more than 150,000 people in the United States have cerebellar ataxia.[4] While ongoing clinical trials have shed light on the symptomatic and disease-modifying therapy,[5, 6] unmet needs in ataxia still abound. Due to physical limitations, people with cerebellar ataxia have reduced family bonding and social connections. Together with other cognitive and affective symptoms, particularly depression,[7, 8] the self-perception of people with cerebellar ataxia has been tremendously impacted. What has made the situation worse is the "awareness" of ataxia – compared to other more well-known neurological disorders such as Parkinson's disease, the majority of the general public have never heard of "ataxia" and many healthcare professionals could not fully define what the constellation of symptoms of "ataxia" looks like. The lack of awareness has led to the dearth of support systems at the individual, organizational, and governmental levels. Many people with ataxia are mistaken as "being drunk all the time." As movement disorders neurologists and cerebellar ataxia specialists, we have been pondering and looking for solutions to cope with this condition and alleviate the suffering of the ataxia community.

COVID-19 PANDEMICS: CHALLENGES AND OPPORTUNITIES

The opportunity unexpectedly came when Coronavirus Disease-19 (COVID-19) was fiercely hitting New York City. Followed by the city's stay-at-home order was the shutdown of business venues, including Broadway, the city's theater district. Times Square became empty. We thought of many actors and singers who were suddenly out of work had spare time. It came to our mind that Broadway actors have spent their lifetime practicing and perfecting movement and voice controls, which may provide a unique perspective for people living with cerebellar ataxia. Therefore, we created this program, *Broadway for Ataxia*. The first step we took was to recruit Broadway talents who were interested in working with people with cerebellar ataxia. To our pleasant surprise, we received 87 applications, from which we selected the final 5 as our current instructors based on their experience, empathy, and willingness to explore whether theatrical arts can improve neurological symptoms. These instructors have talents in diverse domains, including but not limited to choreography, physical comedy, dancing, vocal performance, and piano. Our mission is to enrich the lives of people with ataxia through theatrical art-based sessions to bring out fun, joy. and inspiration. To empower instructors' ability to combine their theatrical talents with neurological knowledge, we applied a multidisciplinary approach, incorporating neurological physical therapy and speech-language pathology training into their coaching curriculum. Our overarching goal is to enable people with cerebellar ataxia to "*live with ataxia, but not defined by the disease*."

SELF-CHALLENGE: OUT OF THE COMFORT ZONE

People have been told that in order to make a difference, it is necessary to step out of their comfort zones. Those who live with cerebellar ataxia can further attest to this fact. One participant that used to be quite athletic before developing ataxia was skeptical about joining the program, as he was not a theater fan. However, he soon proved that it is not

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necessary to be a Broadway lover to thrive in the program. He has exerted his potential with a positive attitude through the program; instead of focusing on activities that he can no longer do such as bicycling and skiing, he focuses on activities that he never thought he could do, such as singing and choreography, which has greatly helped him keep "*different parts of the brain moving*." Likewise, another participant shared that he especially loves the improvised sessions, which always surprise and challenge him out of his comfort zone without any safety concerns. With the instructors' meticulous and individualized coaching, participants feel they have learned how to improve the mind-and-body connection. We have been inspired by their commitment and optimism to live well with cerebellar ataxia, and this is what motivates us to continue expanding the program.

REKINDLING THE JOY AND FUN WITH FAMILY

Perhaps one of the most inspiring moments for us has been when participants said they are now able to regain the enjoyment in bonding with their families. One participant told us: "*It's nice to have something we can do together again because my husband and I used to do a lot, but not anymore due to ataxia. We are also helping one another with vocal exercises in Broadway for Ataxia.*" Another participant practices the songs with his wife when they are driving. Most importantly, their spouses noticed a glow in their faces after each session because they found joy from it. For example, "*physical and speech therapy could be dry, but not anymore when we are doing Broadway for Ataxia.*" In addition to the individual session, we also provided monthly group sessions to promote peer, family, and friends' participation.

ATAXIA AWARENESS AND COMMUNITY CAMARADERIE

Compared to Parkinson's disease, the word "ataxia" is unfamiliar to the general public. The husband of one participant told us when he mentioned his wife has a neurological condition called ataxia, "*they look at me like I am speaking a foreign language; and it should not be a foreign language, as ataxia is happening to a lot of people in different ways*." After joining the program, our participants were able to share their stories through short video narratives, showing how limb coordination, gait, and speech are affected by cerebellar ataxia. Our participants enthusiastically share their videos with their networks to fundraise for *Broadway for Ataxia*. Among this, *altruism* has beautifully emerged: we observe people donating money to fund other people with cerebellar ataxia, which further broadens the community engagement and awareness. People who participate in this program also further use social media to tell their own stories and share how they are inspired by Broadway talent. In addition, we have established a specific program to create accessible sessions for underserved populations that better represent the diversity of people affected by ataxia. We have been deeply touched and proud to have established this platform to build up community camaraderie, a core mission for Broadway for Ataxia.

CONCLUSION AND FUTURE DIRECTIONS

Broadway for Ataxia has become a mutually inspiring program among Broadway performers, people with cerebellar ataxia, and healthcare providers. We established this program with the hope to rekindle the joy and inspiration in people with cerebellar ataxia, yet it turns out we have been inspired by the progress that these participants have made. Based on the current success, we aim to expand these services to pediatric populations, especially Friedreich ataxia, the most common pediatric cerebellar ataxia. In the future, we look forward to working with other art therapy organizations, forming an alliance to provide more mind-and-body healing for those with neurological diseases.

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LIFETIME ACHIEVEMENT AWARD WINNER

E. STEVE ROACH, MD

Contributed by Audrey C. Brumback, MD, PhD

E. Steve Roach is Professor of Neurology and Associate Chair for Clinical Operations of Department of Neurology at The University of Texas Dell Medical School in Austin. He is also the Chief of Neurology at Dell Children's Hospital. After finishing medical school at the University of Tennessee, Roach completed residency training in pediatrics and neurology at Wake Forest University School of Medicine. He remained on the faculty, overseeing the adult consultation service for several years before becoming Chief of Child Neurology at the University of Texas Southwestern Medical Center in Dallas.

Key early mentors were stroke neurologist James F. Toole and epilepsy expert J. Kiffin Penry. With a stroke neurologist as a mentor and extensive experience with adult stroke patients, Roach was uniquely positioned to see stroke in children as more than the curiosity it was considered. His seminal 1988 first edition of *Pediatric Cerebrovascular Disorders* explored the etiology and pathophysiology of cerebrovascular disease in children. The book's comprehensive approach defined the scope of the problem, and pediatric stroke has since become an active topic for research and clinical care. The book's third edition appeared in 2011. Roach was also the lead author of *Toole's Cerebrovascular Disorders*, the sixth and final edition of his mentor's 1967 stroke textbook, written as a tribute with fellow Toole protégé Jose Biller. Steve led the creation of the American Heart and Stroke Association's influential 2008 scientific statement on the diagnosis and management of stroke in infants and children, the first evidence-based and consensus-based recommendations concerning stroke in children.

In his 12 years as the head of child neurology at The Ohio State University and Nationwide Children's Hospital in Columbus, Steve built almost from scratch a powerhouse pediatric neuroscience program that has for several years been ranked in the top ten in the US News survey. The program developed the first pediatric pseudotumor cerebri clinic, an exceptional multidisciplinary pediatric stroke program, and the first dedicated infantile spasms clinic in the United States. Members of the program oversaw the development and FDA approval of the first disease-modifying gene therapy for spinal muscular atrophy, technology that can likely be employed for gene therapy of other genetic diseases.

Roach has also made several important contributions to the study of genetic disorders, on several occasions attending family reunions around Texas with a resident in tow to investigate genetic disorders. The DNA specimens collected during these field trips led to the linkage of paroxysmal kinesigenic dystonia to chromosome 16 and contributed to the linkage of *TSC2* to chromosome 16. He was the first to demonstrate a correlation between the severity of neurological impairment due to tuberous sclerosis complex (TSC) and the extent of brain abnormalities on imaging. He led the creation of the first consensus clinical diagnostic criteria for TSC as well as consensus guidelines for ongoing surveillance for complications.

Roach has served several neurology organizations with distinction. He was elected to the executive committees of both the American Neurological Association and the Child Neurology Society. He served a two-year term as Chair of the Child Neurology Section of the American Academy of Neurology as well as a two-year term as President of the Child Neurology Society. In recognition of his many contributions to the field of child neurology, Roach received the 2015 Hower Award, the Child Neurology Society's highest honor.

In 2021 Roach finished a nine-year stint as the Editor-in-Chief of *Pediatric Neurology*. During this time, both the number of manuscript submissions and the journal's impact factor more than doubled. He was previously an associate editor of *JAMA Neurology* and currently serves on several other editorial boards. Never idle for very long, in 2022 Steve became the founding editor of the *Annals of the Child Neurology Society*, a clinical companion journal to *Annals of Neurology*.

In 2018, Dr. Roach became the Associate Chair for Clinical Operations of the Neurology Department at Dell Medical School and Chief of Neurology at Dell Children's. Among the initial achievements in Austin is the creation of a rapidly growing joint program between the children's hospital and the medical school, The University of Texas Health Austin Pediatric Neurosciences at Dell Children's.

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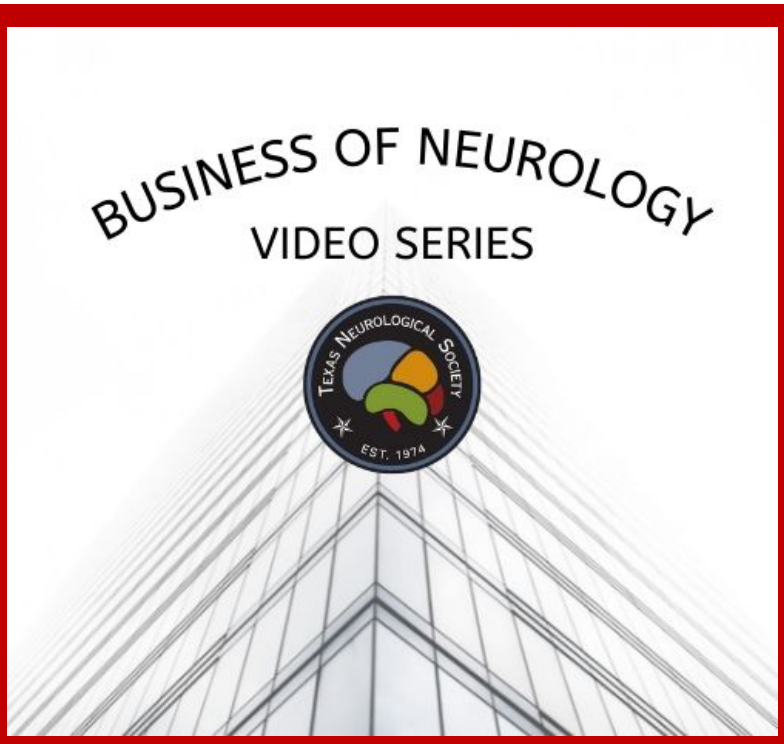

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TNS COMMITTEES

Women Neurologists Section Come network at conferences! This section gets together one evening at the close of a day's general session for the purpose of connection, support and mentoring. It is the best time to meet fellow TNS women neurologists. Get involved today!

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Grants Committee TNS gives back to its membership through the grants committee. Annually, committee members review submitted applications to determine which one/ones achieve the goal of improving and supporting neurological practices, education, and disease awareness and prevention. Continuing to further neurology is what this committee is designed to do.

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