TEXAS NEUROLOGICAL SOCIETY WINTER 2015

Broca's Area

The Voice of Texas Neurology

President's Message

Kimberly E. Monday, MD

Dear Colleagues,

I wanted to take this opportunity to thank you for your input and assistance in 2014 and provide a quick year-end update.

Thank you to Dr. El-Feky for a phenomenal Summer Conference at La Cantera in San Antonio. Our attendance was at capacity and feedback incredibly positive. We hope to see you soon at the Winter conference February 7th in Austin at the Hilton Hotel. Doug Lewis, M.D. and the education committee have organized a relevant, dense, and first rate three day conference. If you have not registered, please do so at www.texaneurologists.org.

On the socioeconomic front, TNS conducted our first Practice Management Symposium during the July conference. Attendees were coached on ACOs, PQRS, and how to maximize outpatient neurology opportunities. This elective Saturday afternoon conference was well attended and the material well received. Ky Camero and Kristi Berrier followed the Practice Management Symposium with our first Practice Management webinar for Texas neurology staff in September. The second symposium will be held on January 27th and will specifically address ICD-10. This webinar is targeted towards both Texas Neurologists and their office staff. D-day for ICD 10 is set for October 15, 2015. As we all know, ICD 10 coding is more extensive and demanding. The webinars are an excellent way to prepare.

The Texas Legislature begins their session January 13, 2015. I especially want to thank those TNS members who have written checks and/or visited our State and US Senators and Representatives while on summer break. Personal relationships with our politicians are vital to protecting our professional interests. From January to May during legislative years, the TMA sponsors the First Tuesday program. Texas physicians wear their white coats and visit with pre-selected legislators regarding issues affecting scope of practice, tort reform, GME funding, Medicaid parity, provider network requirement among many other topics. The TMA does an excellent job of preparing physicians and accompanying them on office visits so you are not asked for information or material you may not know. Taking a weekday off from work seems financially daunting, but I encourage you to check your malpractice rates in 2003 prior to Proposition 12 compared to your current rates. I ran across an old invoice when cleaning out files recently and my T.M.L.T. rates are 25% of the 2003 rates. Losing that protection would be devastating to all practices.

TNS has been busy with two interim issues in preparation for the upcoming legislative session. In June, several of us testified before the Sunset Commission regarding the state policy allowing chiropractors to provide school sport physicals. As a result, the Commission changed the rules so that a physician must now perform the physicals. In response the Texas Chiropractic Association is gearing up to reverse the ruling during the legislative session.

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Mark Your Calendar

2015 SUMMER CONFERENCE

July 31-Aug 2, 2015

Sheraton Fort Worth
Fort Worth, Texas

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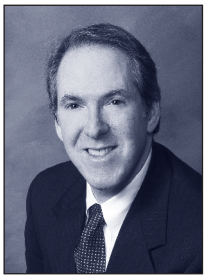
Secondly, the Texas Dental Board changed their scope of practice to allow dentists to diagnosis and treat sleep apnea despite testimony from TNS board members and warnings from the Texas Medical Board. The TMA has graciously taken on this issue and has filed a lawsuit challenging this scope of practice decision judicially. Please remember the TMA's work for us on scope issues and tort reform when it is time to renew your TMA membership and join TEXPAC.

You will be receiving an email survey regarding the Texas Neurological Society after the December holiday season. Please take five minutes to update your demographics and provide feedback. We need your input to ensure that the organization continues to provide value.

As my term as President has come to an end, I would like to thank our volunteer TNS board who spend many hours working on behalf of the society. I would also like to thank our consultants Greg Herzog and Kristi Berrier who have increased our awareness and involvement in Texas issues that affect our profession. A special thanks to Ky Camero who is always insightful, competent, positive, and hard working for Texas Neurologists so we can focus on caring for patients.

I hope everyone has had a healthy and professionally rewarding year and wish you continued success in 2015.



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 Winter Conference in Austin, February 6-8. Doug Lewis, adult program chair; Gary Clark, pediatric program chair; Bob Fayle, education committee chair; and the education committee have planned an excellent program. Be sure to register on or before January 15 to receive your early bird discount.

The Neurology of FDR

Many of you may have been fascinated by Ken Burns' superb 14 hour PBS series, "The Roosevelts" which premiered in September. I was further fascinated on doing a little research into the abundant neurological history of our 32nd president, Franklin Roosevelt.

Did FDR Have Polio?

The documentary reviewed the major impact that developing a paraparesis and being diagnosed with polio had on his life. While campaigning and president, the degree of this disability and his other medical problems were covered up which would not be possible with the modern presidency. Interestingly, in Texas, we have recently elected a new paraplegic governor.

In 1938, FDR founded the March of Dimes (a name coined by vaudeville star Eddie Cantor as a play on the contemporary newsreel series, "The March of Time") to raise money for research which was hugely successful culminating in the approval of the Salk vaccine in 1955 and a change in mission of the organization.

At the age of 39, in 1921, he developed weakness then paralysis of the one leg and then the other, fever of 102 degrees, diffuse achiness, numbness of the legs, and sensitivity to touch of the skin and muscles. Over the next few days, he developed weakness all over including the face and urinary and fecal incontinence. He had no meningismus. A cerebrospinal fluid examination was not performed. He gradually had some recovery. By May, 1923, his arms, face, and neck were normal. Bowel, bladder, and sexual function were normal. The abdominal muscles were weak, the hip flexors were poor, there was paralysis from the waist down, and trace movement of the toes.

Goldman et al, including Texas City neurologist Elisabeth Schmalstieg, (Goldman AS, Schmalstieg EJ, Freeman DH Jr, Goldman DA, Schmalstieg FC Jr. What was the cause of Franklin Delano Roosevelt's paralytic illness? J Med Biogr. 2003;11(4):232-40) analyzed FDRs case in detail and conclude that he probably had Guillain-Barré syndrome. Among the arguments against FDR having polio include polio uncommonly affected adults, caused asymmetric paresis, was not associated with numbness or dysesthesias, usually associated with meningismus (absent in this case), and usually progressed over 3-5 days (while FDR progressed over 10-13 days).

FDR did have fever which can be associated with Guillain-Barré (Dhadke SV, Dhadke VN, Bangar SS, Korade MB. Clinical profile of Guillain Barré syndrome. J Assoc Physicians India. 2013;61(3):168-72). FDRs physicians were polio experts who likely were not familiar with Landry's ascending paralysis (reported in 1859) described again by Guillain, Barré, and Strohl in 1916.

Did FDR Have Essential Tremor?

Starting in 1943, FDR was noted to have an action tremor which affected his handwriting (Lomazow S. The untold neurological disease of Franklin Delano Roosevelt (1882-1945). J Med Biogr.

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2009;17:235-40). His mother, two of his sons, and a daughter also had a tremor. On 8/18/44, Senator Harry Truman observed, "In pouring cream in his tea, he got more cream in the saucer than he did in the cup." In October, 1944, Ambassador Joseph Kennedy noted, "His hands shake violently when he tries to take a drink of water."

Did FDR Have Epilepsy?

Lomazow (Lomazow S. The epilepsy of Franklin Delano Roosevelt. Neurology. 2011;76(7):668-9) reports many observations consistent with a diagnosis of partial complex seizures in 1944 and 1945. In 1944, a reporter, Catledge described a meeting, "When I entered the president's office … he was sitting there with a vague glassy-eyed expression on his face and his mouth hanging open. He would start talking about something, then in midsentence he would stop and his mouth would drop open and he'd sit staring at me in silence … Repeatedly he would lose his train of thought, stop, and stare blankly at me. It was an agonizing experience for me." [Agonizing for him-Catledge met with FDR prior to his nomination to a fourth term. Catledge would soon be the editor of the New York Times. None of this became public. FDR did of course choose a new VP who made momentous decisions as president]

FDR was prescribed phenobarbital between 60 and 90 mg daily from the beginning of April, 1944 allegedly for hypertension.

Did FDR die from a massive cerebral hemorrhage?

On April 12, 1945, while at the Little White House at Warm Springs, Georgia, FDR said, "I have a terrific pain in the back of my head," and collapsed, unconscious, and died 2.5 hours later. He was diagnosed with a massive cerebral hemorrhage. He had a history of uncontrolled hypertension including 230/126 in 1944.

Lomazow (Lomazow S. The untold neurological disease of Franklin Delano Roosevelt (1882-1945). J Med Biogr. 2009 Nov;17(4):235-40) produces interesting evidence to speculate that maybe he really had a right hemisphere metastasis from a melanoma from above his left eyebrow.

FDR and Harvey Cushing?

While a junior at Harvard, James Roosevelt, the son of the governor of New York, became engaged to Betsey Cushing, the daughter of Harvey Cushing, the Moseley Professor of Surgery at Harvard Medical School and surgeon-in-chief at Peter Bent Brigham Hospital, the dominant figure in neurological surgery in the world (Rovit RL, Couldwell WT. No ordinary time, no ordinary men: the relationship between Harvey Cushing and Franklin D. Roosevelt, 1928-1939. J Neurosurg. 2001;95:354-68). The two future father-in-laws exchanged letters. Cushing: "I should have known something of this kind would happen if we came here to Boston to live—that one of my daughters would 'take up' with a Harvard Democrat when there are so many desirable Yale Republicans in that part of the country where we really belong."

In a later letter from FDR: "My better half has made a demand that within three days I give her a complete list of all political and business associates whom I have shaken hands with during the past twenty-five years, in order that they may be invited to your wedding! I want to go on record as expressing my gratitude to you for paying for the event!"

The 1930 wedding reception was held at the Cushing home with 1000 guests including leaders in the worlds of diplomacy, medicine, politics, and society.

In 1936, after Cushing had moved to New Haven, FDR was campaigning and went to Cushing's house for lunch one day with 35 people including Eleanor and Betsey Roosevelt, the governor of Connecticut, the mayor of New Haven, and the attorney general.

Their correspondence over the years in Rovit and Couldwell's article is interesting to read. Cushing lobbied FDR on a variety of medical issues.

In April, 1939, the Harvey Cushing Society (now known as the American Association of Neurological Surgeons) held their annual meeting in New Haven and had a dinner to celebrate Cushing's 70th birthday. FDR could not attend but sent a letter which read in part, "I realize, of course, that in these later years Harvey Cushing has labored under that most severe of all human handicaps— relationship with the President of the United States. His courage and cheerful disposition in the face of this travail proves his eternal greatness."

Cushing died of a heart attack on October 7, 1939. James and Betsey had two children and divorced in 1940. Betsey married John Whitney who was later the ambassador to Great Britain. James Roosevelt had several remarriages and divorces and was a six term United States congressman. Cushing's eldest daughter, Mary, was married for a time to Vincent Astor, one of the richest men in the country. His third daughter, Barbara, married William Paley, the CEO of CBC, in 1947.

Unilateral facial pain

The 2015 Summer Conference will be a joint conference with the Southern Headache Society at the Fort Worth Sheraton July 31-August 2. So a headache case seems appropriate.

Clinical history

This 52 year old female has a 5 year history of facial pain which only occurs at night awakening her from sleep about 2 in the morning. She describes a burning or pressure pain in the right upper teeth with an intensity of 10/10 which then spreads to the entire right face associated with nausea, vomiting once, light and noise sensitivity but no eye redness or tearing, nares congestion or drainage, ptosis or miosis. The pain lasts about 20-30 minutes. During an attack, she feels like she can't lie down and has to get up and move around. The pain may recur a second time within a 2 hour span. The pain may occur daily for 6-8 weeks and

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then go away for about 6-9 months before recurring. She saw a neurologist and was diagnosed with trigeminal neuralgia.

What is the diagnosis?

Episodic cluster headache (at least two cluster periods lasting from 7 days to 1 year (when untreated) and separated by pain-free remission periods of ≥1 month). Cluster headache is misdiagnosed more than 80% of the time when first seeing a physician and even when seeing a neurologist especially in a case like this where there are atypical features (May A, Evans RW. The sexagenarian woman with new-onset cluster headaches. Headache. 2011;51:995-8).

The duration of each headache (untreated, cluster has a duration of 15-180 minutes) nocturnal awakening, and duration of the bouts are all typical of cluster.

In one study, migrainous symptoms of light and noise sensitivity were reported by 70% and vomiting or nausea in more than 20% (Gaul C, Christmann N, Schröder D, et al. Differences in clinical characteristics and frequency of accompanying migraine features in episodic and chronic cluster headache. Cephalalgia. 2012 ;32(7):571-7). Perhaps 14% of cluster patients report an aura including visual and paresthesia (Evans RW, Krymchantowski AV. Cluster and other nonmigraine primary headaches with aura. Headache. 2011;51:604-8). Gaul et al found headaches occurring between 1-6 am in about 75% of patients.

There are two less common features in this case. She does not have associated cranial autonomic symptoms (CAS). In a series of 95 cluster headache patients, 95% had CAS with the following: conjunctival injection and/or lacrimation, 95%; conjunctival injection, 62%; lacrimation, 95%; nasal congestion and/or rhinorrhea, 77%; nasal congestion, 45%; rhinorrhea, 65%; eyelid edema, 21%; and forehead/facial sweating, 57% (Lai TH, Fuh JL, Wang SJ. Cranial autonomic symptoms in migraine: characteristics and comparison with cluster headache. J Neurol Neurosurg Psychiatry. 2009;80:1116-9). Gaul et al's study of 209 consecutive patients with episodic and cluster headache found at least one CAS in 99.5%.

The International Headache Society criteria for cluster require either or both of at least one CAS or a sense or restlessness or agitation. Gaul et al found restlessness in 83% of cases as in this case.

And the distribution of pain? Gaul et al found periorbital pain location reported by more than 75% of patients followed by occipital neck region and orofacial pain. They note that the orofacial localization and some patients reporting toothache-like pain (40%) may lead to unnecessary dental treatments including extractions. Based upon other series, the pain is behind the eye in about 90%, over the temple in 70%, and over the maxilla in 50% (.Nesbitt AD, Goadsby PJ. Cluster headache. BMJ. 2012 Apr 11;344:e2407). The pain is often described as sharp, stabbing, piercing, burning, or pulsating. About 15% report that the pain shifts sides between bouts of attacks and, less often, during a bout, but never during a single attack.

What about the features of trigeminal neuralgia? By International Headache Society criteria, the duration of each paroxysm of pain has a duration of a fraction of a second to 2 minutes. In a prospective series of 158 patients with classical TN, the average age of onset was 52.9 years with 60% females affecting the right side of the face in 56%, left side 41%, and bilateral 3% (Maarbjerg S, Gozalov A, Olesen J, Bendtsen L. Trigeminal Neuralgia – A Prospective Systematic Study of Clinical Characteristics in 158 Patients. Headache. 2014 Sep 18. [Epub ahead of print]. Pain was reported in the following distributions: V1, 4%; V2, 17%; V3, 19%; V1+V2, 10%; V2+V3, 33%; and V1+V2+V3, 13%. Thirteen percent had a more dull persistent pain at the onset of the disorder ("pretrigeminal neuralgia") while 87% had stabbing paroxysmal pain. The paroxysmal pain was rated on average 10/10 by 58% of the patients. Forty nine percent of the cohort reported concomitant persistent pain along with the paroxysmal pain.

Forty percent suffered from more than 10 paroxysms of pain per day. Painful awakening at night because of pain attacks at least occasionally was reported by 49%. Trigger factors were reported by 91% including the following: chewing, 73%; touch, 69%; brushing teeth, 66%; eating, 59%; talking, 58%; and cold wind, 50%. During attacks of pain, 31% experienced ipsilateral autonomic symptoms, most commonly conjunctival tearing or injection. Of the surgery naïve patients, 29% had sensory abnormalities on exam, most commonly hypesthesia confined to the painful area of the face. Most patients (63%) had periods of remission with the average number per year of disease of .44 with 37% having months of remission and 63% experiencing years of remission.

Thank you to our supporters of the 11th Annual Summer Conference

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Mobile Stroke Unit Hits the Road in Houston

By James C. Grotta, MD
Director of Stroke Research, Clinical Innovation and Research Institute, Memorial Hermann Hospital-TMC

The nation's first Mobile Stroke Unit (MSU) was launched in February, 2014 by James Grotta MD and colleagues at the Texas Medical Center in Houston. The concept, pioneered by Marie Curie when she loaded an xray machine on an ambulance during World War I, is logical—to take emergency care to the patient in order to allow accurate diagnosis and urgent treatment earlier when it may do more good. Now 100 years later, instead of a physicist and xray machine, it is a Vascular Neurologist (VN) and CT scanner.

Time is particularly important in acute stroke therapy. Data from primates and rodents show that if recanalization occurs during the first hour after arterial occlusion, cerebral infarction might be completely avoided except in the "core" of distal carotid or proximal middle cerebral artery occlusions. Surprisingly, the initial NINDS tPA study results did not seem to reflect this relationship. But once it was recognized that patients with the most severe strokes presented to the Emergency Department (ED) earlier and stroke severity was factored into the calculation, it became clear that the sooner tPA was given, the better the clinical response to tPA. The same relationship exists with endovascular mechanical reperfusion, and probably efforts to reduce bleeding by blood pressure lowering or coagulopathy reversal after intracerebral hemorrhage.

While the use of tPA is increasing (now 5-10% of all stroke patients, and 10-20% in most stroke centers), treatment usually occurs 2-4.5 hours after symptom onset even if patients or bystanders do the right thing and call 911. In the NINDS study, none of the 302 patients who were randomized within the first 90 minutes actually got treated within the first 60 minutes from onset. Current national databases show that less than 5% of treatments occur in the first hour. The delay is multifactorial and includes delay in calling 911, mobilizing EMS and transporting patients to the right ED, and finally the ED door to needle time which stubbornly averages 50-60 minutes even in our best stroke centers, largely consumed by time to obtain the CT scan and having the decision-maker (usually the neurologist on call) look at it and make a decision after weighing all the variables.

The MSU cuts out the entire door to needle time delay by putting the CT scanner and decision maker on the ambulance. The initial studies conducted in Germany and published over the past 2 years demonstrated anywhere from 25-80 minute time savings, and over 30% treated within the first hour after symptom onset, without excess complications. In 2013, after visiting Drs Fassbender and Audebert who pioneered the MSU concept in Germany, Dr Grotta decided to implement an MSU in Houston.

There were three basic principles that guided how this project would be carried out. First, we understood that no funding was available from UT Medical School, Memorial Hermann Hospital, or the City of Houston to underwrite this project. Second, we needed to operate within the current EMS transportation and triage system which delivered acute stroke patients within 30 minutes' drive from the Texas Medical Center equally to one of 3 Comprehensive Stroke Centers (CSCs). Third, our goal was to determine if the MSU is a practical strategy for speeding stroke care that could have a meaningful impact on stroke outcome nationwide.

To make this determination, we decided to carry out a clinical trial to answer three questions: 1. How much faster could we treat patients and how much better outcome would result? 2. Could we replace the VN on board the MSU by a remote VN using telemedicine (TM)? 3. What would be the balance between the benefits and the costs of implementing and deploying the MSU. The BEST-MSU Study (Benefits of Stroke Treatment Delivered Using a Mobile Stroke Unit Compared to Standard Management by Emergency Medical Services) was approved by the UT-Houston IRB on 11/01/2013 to address these aims and is registered with clinicaltrials.gov (NCT02190500).

We began fundraising in March 2013 eventually raising $1.8M largely from donations by generous Houston citizens and businesses, and grants from pharmaceutical and device manufacturers. Two critical early donors deserving particular recognition were Frazer Limited from Bellaire, Texas who donated the ambulance "box", and James ("Mattress Mack") McIngvale. After purchasing the Neurologica CereTom CT scanner, many steps then went into building, licensing, inspecting, insuring, equipping, and staffing the MSU. This required the 100% effort of a multi-talented Project Manager, Stephanie Parker RN. Frazer modified a standard 12 foot ambulance used by the Houston Fire Department to accommodate the CT scanner. Extensive radiation safety inspection, and licensing by both state and city authorities as an ambulance under the umbrella of Memorial Hermann Life Flight required the cooperation of administrators at UT Medical School and Memorial Hermann. Leadership of the stroke teams at Baylor St Lukes, Memorial Hermann, and The Methodist Hospitals, along with Houston Fire Department Emergency Medical Services (EMS) and the MSU team formed the Houston Mobile Stroke Unit Consortium with a Steering Committee that oversees the entire project.

The MSU is staffed by a VN, a Registered Nurse (both with ACLS training), a full time licensed radiology CT tech, and an off-duty EMS paramedic, and also has remote access to a second VN at UT Medical School via TM. In addition to

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standard emergency equipment and the CT scanner, the MSU also has point of care laboratory capability. An MSU office was rented at the UT Professional Building, 6410 Fannin St where we also rent a parking space with power supply for the MSU.

Once the MSU was delivered by Frazer, we developed on-board protocols for all advanced life support scenarios, and carried out extensive in-servicing of EMS personnel in order to integrate the MSU into the daily operations of pre-clinical care in Houston, Bellaire, and West University. A communications system was devised where the MSU would be notified by the dispatch centers of each of these three cities after a 911 call suggestive of a stroke, or if a paramedic requested MSU dispatch after recognizing a stroke patient on-scene. The MSU primarily responds to calls within a 5 mile radius of the MSU offices, but can also rendezvous with EMS squads bringing patients to the TMC from a greater distance. The design is for the MSU to meet up with the "regular" EMS squad without delaying or altering their routine pre-hospital management. The MSU team responds to EMS calls every day from 8 am-6 pm, except designated holiday weeks.

Upon receiving a call, the MSU travels to the site of the call or rendezvous with EMS and evaluates the patient. If the patient meets inclusion criteria (symptom onset within 4.5 hours, and meeting published criteria for tPA treatment pending CT scan and baseline labs), they are enrolled into the study and moved into the MSU. If after CT scan and point of care lab testing on the MSU, the patient still fulfills criteria for tPA according to the on-site VN (the patient is simultaneously evaluated via TM with the remote VN making an independent decision), they are immediately given tPA and transported to one of our 3 CSCs. If the patient doesn't meet tPA criteria, they are managed as per best practice for their diagnosis en route to the CSC. This may include blood pressure lowering or coagulopathy reversal in the case of intracerebral hemorrhage.

The MSU went into service on 05/14/2014. According to a pre-specified plan, the MSU was activated from 8 AM to 6 PM for an 8 week "run-in" phase to test the communication system, rehearse interactions between EMS, the MSU team, and the remote TM VN, test out the case report forms, and confirm projected MSU activity and treatment rates.

During the "run-in" phase, the MSU was dispatched by the dispatch center on 130 occasions, or roughly 2.7/day. The MSU was "disregarded" en-route to the scene for 41 of these dispatches when it was determined by either the first responders or paramedics to not fit study criteria. For another 65 of these dispatches, the MSU team arrived on scene, assessed the patient, and determined the patient did not qualify. These 106 patients were considered "screen failures". They were transported as per EMS routine and no further data were obtained.

Twenty four patients met criteria for enrollment (symptom onset within 4.5 hours, and meeting published criteria for tPA treatment pending CT scan and baseline labs). 11 of these patients were not treated. Four had primary intracerebral hemorrhage and had their blood pressure acutely lowered according to our current standard of care protocol. Three had seizures on board the MSU which were thought to be the cause of their presentation and which were treated on board the MSU. Two patients improved to the point where the MSU staff determined that tPA was not indicated. The time of onset could not be confidently determined in 1 patient, and 1 had a subdural hematoma. 13 patients were treated with tPA on the MSU; 4 (31%) between 0-60 minutes of onset, 4 (31%) between 61-80 minutes from onset, and 5 (38%) between 81-270 minutes of onset. Average on-scene time from MSU arrival to tPA bolus was 24 minutes (range 12-53) in the 13 treated patients. There were no hemorrhagic or other complications and no malfunctions of the CT scanner or MSU. The intravenous infusion pump malfunctioned on one patient, and the i-STAT device malfunctioned because of heat on one occasion. TM assessment of the patient was carried out successfully in all 11 cases in which remote assessment was attempted, and agreement between the remote and on-site VN was 91%. Three of our 13 patients had endovascular treatment with onset to groin times of 224, 140 and 150 minutes.

On 08/19/2014 we began randomizing into the BEST-MSU Study; since then our tPA treatment rate on the MSU continues to average 2-3 patients per week, with over 40% treated within the first 60 minutes of stroke symptom onset. In order to carry out the BEST-MSU study, on 50% of weeks by blocked randomization, the nurse meets the patient without the MSU, determines eligibility by the same criteria, but the patient is transported and managed per current EMS routine. Informed consent is obtained from the patient or next of kin at the CSC after all acute stroke care is complete to obtain follow up data at 1, 3, 6 and 12 months in 248 patients to answer the 3 aims. The Data Management and Health Economics Center is at the UT School of Public Health.

In summary, the Houston Mobile Stroke Unit is in full time operation and should answer important questions about the feasibility of this approach to acute stroke care through the BEST-MSU study. We anticipate it will take 4 -5 years to complete this study. Additional funding is needed and is being sought from granting agencies and donors. Other MSU sites in other cities are in various stages of development to partner with us to answer the aims of the BEST-MSU study more quickly. If we are successful, eventually we envision 4-5 MSUs strategically deployed and embedded within the Houston Fire Department EMS fleet (and proportionate coverage throughout the rest of the country), with telemedicine coverage and 3 persons on board; 2 paramedics and a third person to interact with TM and cross trained to carry out the CT scanning. The costs of building and staffing these MSUs would be amortized by higher reimbursement from payers (insurers) justified by the cost savings resulting from better outcomes and reduced long term care requirements.

James C Grotta, M.D.

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Advocacy Update – Fall 2014

By Sara Austin, TNS Legislative Chair

With advocacy, I have learned if nothing else, you spend an inordinate amount of time preparing for something that will most likely never happen. And if it does happen, it will occur very quickly and without much warning. I had the naïve belief that good policy took years to make and was a work in progress until most people agreed with it, and then it was passed into law. I believe now that a lot of good policy never sees the light of day, and sometimes bad policy is made very quickly, because it's just there at the right time. OK, enough philosophizing. Here is a brief summary of what's going on; federal first.

1. No luck with the SGR fix. There was some talk that the same House members that tried to get the bill passed last spring might give it another go. But no bananas. We still face the cut on March 31st, so that is the next time we will see some work on it.

2. It is highly unlikely that the 'Medicaid bump' will be extended, with or without Neurology included. Not only is the federal government not going to do it, but Texas is not considering it either.

3. Telemedicine is getting some traction this year and there is recognition that state licensure requirements need to acknowledge that many docs who do telemedicine have to be licensed in several different states. There is legislation, the Interstate Compact, that attempts to streamline licensure requirements and both Texas neurologists and the AAN will be advocating for this.

Now on to Texas politics. The TMA had their advocacy meeting in Austin, December 5-6. Greg Herzog, our lobbyist was there with me watching over things for us. The next legislative term starts in January, and there have been lots of committee meetings and appointments being made to get ready.

1. The budget will be bigger than last year, but most people expect that this Republican/Tea Party dominated group of legislators will keep a pretty tight rein on spending.

2. Transparency about medical charges will also come up. We will keep a close eye on that one for you and let you know how it's playing out.

3. Scope of practice is always a big issue and this year the physical therapists are pushing hard to have independent practice. The TMA is against that. The Sunset Commission recommended that the Chiropractors be prohibited from doing UIL physicals (which is a change). It was a narrow vote and the legislature will have to up-hold the ruling – so get ready to contact your representative about that when the time comes.

4. The TMA just filed suit, supported by the TNS, to prohibit new rules from the TX Dental Board that allow dentists to diagnose, test and treat patients for sleep apnea. Thank you TMA for helping us with this one.

5. I think that some 'end of life' issues will come up, but the leadership has said that legislation will not be pulled up for a vote unless there is complete agreement. That is very unlikely to happen, so hopefully this issue will be fairly quiet.

6. I think every physician recognizes the need for more GME funding, and we are lobbying hard for that.

Don't forget that first Tuesdays start in February – please make time to come to one. They are interesting, a lot of fun, and they make a difference. Now's the time to attend some fundraisers for your Congressional member too; show up if you get an invitation. They notice that you are there.

TNS partners with Southern Headache Society on Summer 2015 meeting

On behalf of the TNS Board and the Southern Headache Society (SHS) Board we would like to invite you to this 12th Annual Summer Conference of the TNS and 5th Annual CME meeting of the SHS in Fort Worth on July 31 - August 2, 2015 at the Sheraton Hotel.

The meeting will feature an expanded format with 16 total hours of CME (1/2 day on Friday, full day on Saturday, and 1/2 day on Sunday), with 12 hours devoted to headache and pain. Topics will include; office based procedures for migraine rescue, new classes of migraine medication currently in the pipeline, headache and pain behavioral therapies, and various stimulator devices for cluster and migraine headaches. The headache portion of ICD-10 will be reviewed and shown how it can be a tool for improved patient care.

We look forward to seeing you this summer.

Michael Ready, MD – SHS meeting chair, and Brian Loftus, MD – SHS & TNS meeting chair, Vice President, Southern Headache Society

Expert Opinion

Mild Cognitive Impairment – Neuropsychological and Behavioral Presentation

Francisco I Perez, PhD, ABPP
Clinical Assistant Professor of Neurology, Baylor College of Medicine

The following are three cases that illustrate the most common clinical neuropsychological presentations of Mild Cognitive Impairment (MCI) in a neurology clinic.

Case1:
A 68-year-old married man with a high school education and owner-CEO of a large commercial construction company referred for neuropsychological evaluation by his neurologist who is evaluating concerns of a gradual decline in memory. He worries about word finding problems as well as being able to make decisions with increased distractibility. Admits to a history of being anxious, decreased motivation, at times being disorganized and confused. Reports being depressed lately and not feeling confident. Suffers from sleep apnea and does not use the CPAP. Hypertension is present well controlled with medications. MRI of the brain revealed a moderate degree of atrophy and minimal evidence of periventricular white matter changes. Ventricular size is not out of proportion to the degree of atrophy. MMSE score 29/30. The neuropsychological data reflects continue involvement in his day-to-day activities, continues to work but his son now runs the business, able to drive with no reported problems. His cognitive and intellectual abilities are well preserved. Mild short-term memory problems noted but consistent with age. No language, motor or sensory deficits. Prone to making careless errors associated with distractibility. No executive deficits. Screening for depression revealed evidence for a moderate depression. Neuropsychological data is most consistent with depression. Testing provides a baseline marker.

Case 2:
A 71-year-old married man with an MBA worked as a CPA and is now retired. Presents to the neurologist with concerns of a gradual decline of memory over the years worse the last few years. Has resorted to taking notes and does well with this. Also worries about finding words and decreased ability to process information. Wife thinks he is doing well except "not being able to remember very well." Father had Parkinson's. Good health. No prescription medication. Continues to be active and socially involved. He is more careful while driving. Sleeps well. Denies depression. Neuropsychological data documents well preserved intelligence and cognition within the context of significant verbal short-term memory problems. Mild word finding problems and decreased processing of information were noted. Mild executive deficits were documented associated with impulsivity and decreased cognitive flexibility. No sensory or motor deficits. Depression screening was normal. MRI showed significant periatrial microvascular white matter changes, visibly not only on FLAIR, but on T1 as well. No evidence of hydrocephalus or of hippocampal atrophy. PET did not show an AD pattern. Neuropsychological data is most consistent with Amnestic Multiple Domain MCI. Suspect a vascular etiology. Testing provides a baseline marker.

Case 3
An 82-year-old divorced woman with two years of college living in an independent living facility presents to the neurologist on 12/2012 with concerns about memory loss first noticed "within the last year." Worked as church secretary for over 20 years. Hypertension is under good control with medications. Good health otherwise. Sleeps well. She has four grown children who look after her. No history of mental health issues. She gave up driving on her own. No family history of dementia. On Aricept. Daughter concerned about recent memory loss. MRI showed enlarged ventricles. Suspected hydrocephalus but it was ruled out. Neuropsychological data indicated normal intelligence and well preserved cognitive abilities. Processing speed of information was superior. Mild to moderate short-term memory problems documented. Decreased hearing but no other sensory or motor deficits noted. Depression screening was normal. Neuropsychological data is most consistent with Amnestic MCI Single Domain. Testing provides a baseline marker.

Referred again for repeat assessment on 10/2014 because of worsening short-term-memory. She is now 88. Continues to live at the independent living facility. No significant changes in her health. Sleeps well. Neuropsychological interview indicates increased problems relating her recent history. Family reports seeing increased memory problems and concerns about her consistency in taking her medications. They report that she eats food that has gone bad and has gotten sick. She buys things that she does not need and has already bought. She is buying memory supplements. Her children have noted increased irritability and problems managing her life. Neuropsychological data documents in general a mild cognitive decline but a very significant decline in her short-term-memory from a Memory Quotient of 106 to 77. Increased language problems were documented. Increased proneness to making errors was found. Screening for depression was normal. Neuropsychological data documents changes in cognition and memory over a two-year interval with increased problems in managing day-to-day life. The data suggests progression to a degenerative mild-to-moderate dementia when compared with baseline testing.

Learning Objectives:

1. Prevalence of MCI.
2. Cognitive subtypes of MCI.
3. Prevalence and incidence of MCI subtypes.
4. The progression of cognitive subtypes of MCI.
5. Predictors of Progression from MCI to Dementia.
6. Risk factors for MCI and Dementia
7. Behavioral-psychological disturbances in MCI
8. Management of MCI.

Prevalence of MCI:
Some degree of cognitive decline is normal with age. In MCI, cognitive changes are more substantial than those seen in normal aging, but not severe enough to cause major lifestyle changes. Although there can be progression along the continuum from normal aging to MCI to dementia, this does not always occur. A general estimate of the prevalence of MCI is between 10 and 15% of adults over the age of 65 (1). Not everyone who has MCI will develop dementia. Some

studies indicate that as many as 40% of individuals who first meet criteria for MCI return to normal within a year. Mood fluctuations and medication effects can mimic MCI. About 10-15% of individuals with MCI will progress to dementia within the first year, and about 50% will develop dementia within 5 years. This is a higher incidence rate than seen for individuals without MCI, but it is worth noting that MCI does not always progress to dementia (2).

Cognitive subtypes of MCI:
Several subtypes of MCI have been identified. They differ in the type and in the number of cognitive abilities that are impaired. The amnestic MCI describes individuals with significant deficits in memory that either occur in isolation (Amnestic MCI Single Domain) or can be accompanied by deficits in other cognitive domains (Amnestic MCI Multiple Domain). In nonamnestic forms of MCI, individuals exhibit normal performance on memory tasks but show significant deficits on one other cognitive domain such as language, executive functioning, or visualspatial abilities (Nonamnestic MCI Single Domain), or impairment in two or more of such cognitive domains (Nonamnestic MCI Multiple Domain). It has been suggested that these four clinical subtypes may have different underlying etiologies, one of which is an impending dementia or Alzheimer's disease (3).

Prevalence and incidence of MCI subtypes:
Findings from population-based studies suggest that more than twice as many people suffer from Nonamnestic Single Domain MCI than Amnestic Single Domain MCI. Multiple Domains, either the nonamnestic or amnestic form, is thought to be the next most frequent form, while Amnestic MCI Single Domain is the least common (4). However, in a clinical setting, the amnestic form of MCI tends to be seen more frequently. A decline in the ability to remember motivates people to seek medical assistance. Other domains of MCI without memory concerns may not be referred or may not be as concerned (2).

The progression of cognitive subtypes of MCI:
A meta-analysis of 51 clinical and population-based studies showed that people who develop Alzheimer's disease exhibit cognitive deficits in a range of cognitive functions three or more years prior to the onset of a clinically diagnosable dementia, including global cognitive deficits, and impairment in episodic memory, executive functioning, speed of information processing, language abilities, attention, and visuospatial abilities (6). Research both from clinical and population-based samples show that individuals with amnestic MCI have a high risk of progressing to a full dementia over subsequent years. Three quarters of the people with Amnestic MCI Multiple Domain had reached full dementia syndrome over a three-year period, compared to only 4.7% of people with no cognitive impairment and one-third of persons with Amnestic MCI Single Domain. This accounted for a twenty-fold higher risk of developing dementia, especially Alzheimer's disease, in people with amnestic MCI Multiple Domain compared to people with no cognitive impairment. The dementia risk associated with amnestic MCI Single Domain was lower, but still significant, with a nine-fold risk of dementia compared to cognitively normal elderly. Nonamnestic MCI Single Domain did not have an increased risk of developing dementia or Alzheimer's disease compared to people with normal cognitive functioning (5).

The most typical outcome of MCI depends on the subtype. MCI can be seen as a very early, preclinical phase of different forms of dementia. The different subtypes of MCI may reflect the earliest signs of different forms of dementia. These are the most likely outcomes of different subtypes of MCI (6):

- Amnestic MCI Single Domain – Alzheimer's Disease; Depression.
- Nonamnestic MCI Single Domain – Frontotemporal dementia.
- Amnestic MCI Multiple Domains – Alzheimer's; Vascular dementia; Depression.
- Nonamnestic MCI Multiple Domains – Lewy Body disease; Vascular dementia.

Predictors of Progression from MCI to Dementia:

1. Greater cognitive impairment, and faster rate of cognitive decline.
2. Multiple-domain MCI versus single-domain MCI.
3. More and faster atrophy of medial temporal lobe regions including hippocampus.
4. Apolipoprotein E e-4 allele presence.
5. Positive evidence of amyloid on PET imaging and/or higher ratio of tau protein relative to a particular amyloid protein (AB42) in cerebrospinal fluid.
6. Other cerebrovascular risk factors such as high blood pressure and diabetes.

Risk Factors for MCI and Dementia:
Not much is know about MCI risk factors. It is likely that the risk factors for MCI are quite similar to those for dementia. There is no way to determine for sure who will develop MCI. There are both modifiable and non-modifiable risk factors.

Non-Modifiable Risk Factors:

1. Rates of MCI and dementia increase with age.
2. Most studies find no effects of sex on MCI.
3. More people with MCI than older adults without cognitive impairment have the APOE e4allele. However, since MCI is associated with various etiologies, there is likely not a special genetic risk factor for MCI (7).
4. TBI has been associated with increased risk of dementia. A few studies also document increased risk for MCI (8).

Modifiable Risk Factors:

1. The higher the educational level the lower the risk for MCI or dementia (9).
2. Older adults who are cognitively engaged are less likely to develop MCI and dementia.
3. Vascular and metabolic factors.
4. Apathy and depression.

Behavioral- psychological disturbances in MCI:
Apathy and depression are the most common neuropsychiatric symptoms in MCI, followed by anxiety, agitation, irritability,

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nighttime behaviors, and appetite disturbances (10). Symptoms related to psychosis, such as hallucinations and delusions, are not common in MCI. Data from a clinical study on memory clinic outpatients showed that although apathy was associated with an increased risk of progressing to Alzheimer's disease in patients with MCI, patients with concurrent depression had less of risk of Alzheimer's disease (10).

Management of MCI:

To date there are no approved drugs for the management of MCI. A number of clinical trials have demonstrated no convincing effects for delaying conversion to dementia. Various memory intervention programs have been established (11). They combine education, counseling, as well as practice of the cognitive and behavioral strategies taught in the program. The emphasis is in developing procedural memory and habit strategies to develop routines and structured habits. Procedural memory learning remains very stable for longer periods of time in MCI and dementia. Initial research studies found that the program was successful in changing everyday memory behavior in MCI (11). The Mayo Clinic has developed the Healthy Action to Benefit Independence & Thinking (HABIT). Some preliminary data has demonstrated efficacy of the program in increasing quality of life in people with MCI as well as their partner who also participates in the program.

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Obituary

WILLIAM J. RILEY, III, MD, PHD (1930-2014)

Founding Member of TNS

WILLIAM J. RILEY, III, MD, PHD, FAAN, FACP passed away July 21, 2014 in Houston, Texas. He was a loving husband and father, caring doctor, tireless promoter of Catholic vocations through Serra Club International, a mentor, teacher, educator to all, a cattle rancher, and, above all, a man whose greatest desire was to bring honor and glory to God. He excelled as a student at UC Berkeley, University of Chicago, and University of Minnesota. He served his country as a Staff Sergeant in the U.S. Air Force. He served as Chief of Neurology at St. Luke's Hospital, as a faculty member at Baylor College of Medicine and the University of Texas at Houston, and held many positions in the Harris County Medical Society and the Texas Medical Association.

Dr. Riley was a co-founder of the Texas Neurological Society in 1974, president 2002-2003, and recipient of the Lifetime Achievement Award in 2005. Dr. Riley was an always energetic and dynamic advocate for physicians.

Dr. Riley will continue to be loved by his wife Margit, his former wife Joan M. Weismann Riley, as well as his children, Sean and Ginny Riley, Kevan and Sarah Riley, Megan and Steve Turner, Janeen and Kevin Carter, Michael and Jeanie Riley, Britta Riley, Shane and Magda Riley, Tim and Amy Riley, and honorary son Majid Al-Sudairy. Dr. Riley was also blessed with grandchildren Nicholas and Bonnie Riley, Kendall Turner, Zachary Carter, Patrick Riley, and Addison Riley, and his first great grandson.

Dr. Riley's great intellect, humor, and good nature will be terribly missed among his TNS, HCMS, TMA colleagues and friends and by former residents.

Case Study for REM

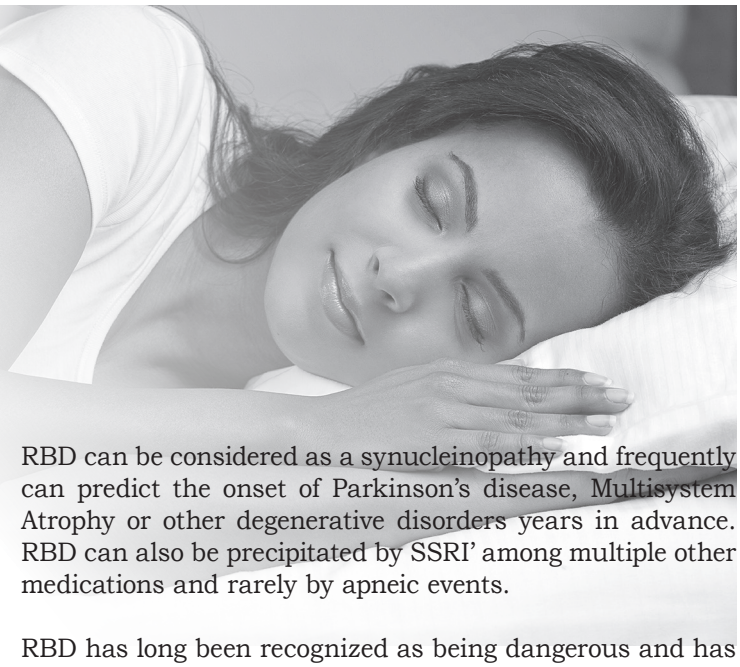
By: Robert Fayle, MD

The Rapid Eye Movement (REM) parasomnias usually demonstrate an aberration in the control or maintenance of the features of REM sleep. Salient feature of REM sleep include an asynchronous, rapid EEG pattern, a loss of muscle tone and reduction to loss of the EMG signals except for the extra-ocular muscles and the diaphragm. When awakened from REM sleep, patients often report dreaming with dreams including physical activity: a video segment, not a static photographic dream. When these features, a variety of REM disorders occur. The following case is an example.

RD is a 57 man who was referred for possible polysomnography (PSG) for suspected sleep apnea. He had been noted by his wife to snore and occasionally to have complete apneic events. He has infrequent nocturia. He reports mild daytime sleepiness with as Epworth Sleepiness Scale (ESS) of 10 (NL<9). Both he and his wife denied sleep walking or periodic limb movements. He had been on as stable dose of sertraline for more than two years. For several years, he had begun to have episodes in the second half of the night time sleep period in which he would sit up and shout. He would flail about and punch with his arms as though he were fighting off an assailant. His eye remained closed in these episodes and he did not get out of bed or walk about. In one of these episodes, he flung himself out of bed, landing on the vertex of the head. He awoke immediately, aware of excruciating neck pain, but there was no weakness of the limbs and no sensory loss. EMS was called and he was taken to an ER, where multiple fractures of the C2 vertebra were found. He underwent a reconstructive surgery, and the bony fragments were stabilized and he had an uneventful post-operative course.

On examination, there were no neurological deficits. There was no evidence of radiculopathy or myelopathy. The examination was remarkable crowding of the upper airway (Mallampati scale ¾), a healing transverse incision from the anterior cervical neurosurgical approach.

A PSG was done, and he did have moderate sleep apnea with moderate O2 desatruations. During the PSG he also has brief bursts of phasic EMG activity in the limb leads and one full blown dream enactment in which he sat up in bed and began to flail his arms. The dream enactment lasted less than a minute and ended spontaneously. He had fragmentary memory of the episode the following morning. He underwent CPAP titration and was started on home CPAP. On the CPAP follow-up, he has been adherent with the machine and his symptoms of the sleep apnea have improved. The history and the PSG findings were also compatible with RBD. He was started on temazepam 15 mg which has eliminated the RBD, although he still has frequent but non-violent dreaming.



RBD can be considered as a synucleinopathy and frequently can predict the onset of Parkinson's disease, Multisystem Atrophy or other degenerative disorders years in advance. RBD can also be precipitated by SSRI' among multiple other medications and rarely by apneic events.

RBD has long been recognized as being dangerous and has been associated with sleep related injuries. This case demonstrates the danger of the disorder. There is often injury to bedmates who are sleeping in proximity to the patient. The potential for possibly severe trauma to the patient of to the bedmate underscores the importance for neurologists to get appropriate history when confronted with patients having movement activity during sleep.

Practice Management Webinar — Mark your Calendar —

The Texas Neurological Society would like to invite all **TNS members and their office staff** to participate in the TNS free webinar series. The second webinar in the "Practice Management for Staff" series will take place on **Tuesday, January 27th at 12:00 pm (Noon).** The topic and speaker is listed below. If you are interested in participating in this free webinar, please email Ky Camero at ky.camero@texmed.org. We encourage all TNS members and office staff to participate!

TOPIC

ICD-10 Preparation: Is Everyone Ready?

What needs to be done to prepare from a business perspective

Speaker: Bryan Soronson, MPA, FACMPE, CRA
Senior Administrator
University of Maryland Department of Neurology

AAN Update

Free CME and MOC Programs for AAN Members

NEW Beginning January 1, 2015, the following online learning programs will be included FREE* with AAN membership:

- NeuroPISM: Provides 20 CME per module; designed to help neurologists meet the ABPN performance in practice clinical component requirement for Maintenance of Certification
- NeuroSAE®: Provides 8 self-assessment CME per exam; designed to help neurologists meet the ABPN self-assessment requirement for Maintenance of Certification
- NeuroLearnSM: Provides 1–2 CME per course; designed to address relevant clinical and practice topics

Visit www.aan.com/view/MOC for more information

Value-based Payment Modifier

The value-based payment modifier (VBPM) fulfills an Affordable Care Act (ACA) mandate that, by 2015, the Centers for Medicare & Medicaid Services (CMS) apply a value modifier to payments under the Medicare Physician Fee Schedule. Both cost and quality data are included in calculating the value-based payments. Watch the AAN video series on the Value-Based Payment Modifier and reading your QRUR.

2015 Medicare Physician Fee Schedule

Download the national relative value units (RVUs) and dollar values for CPT codes used by neurologists.

New CPT Codes Available January 1

Report new codes and receive payments for chronic care management services and advance care planning starting January 1.

Capitol Hill Report

Learn about legislative action and how the Academy ensures that the voice of neurology is heard onCapitol Hill. The Academy's Center for Health Policy staff in Washington, DC, provides bi-weekly updates on advocacy for neurology and neurologic concerns through Capitol Hill Report.

https://www.aan.com/public-policy/capitol-hill-report/

Attend Breakthroughs in Neurology January 23–25 in Phoenix, AZ

This innovative experience combines the "best of" clinical highlights, scientific breakthroughs, and other hot topics from the past year and delivers them in a concise three-day weekend. Attendees can choose from a variety of topic-intensive tracks providing the latest updates on several key areas of clinical neurology.

https://www.aan.com/conferences/breakthroughs-in-neurology/

TNS Winter Meeting

February 6, 7, and 8, 2015

It is time to register for the TNS Winter Meeting. The meeting will be at the Austin Hilton. This year's meeting promises to be another of the excellent TNS meetings. Douglas Lewis, MD and Gary Clark, MD, the program directors for the adult and pediatric meetings, have put together an excellent menu of speakers on a wide variety of topics that should provide a good learning experience for everyone. As always, there are ethics topics for both the Pediatric Neurology session and the Adult Neurology session. The cost is low and the setting is central.

Dr. Clark has arranged for timely presentations for anyone seeing children with neuromuscular disorders and epilepsy.

The adult sessions are also going to be very interesting. There is something for everyone. Topics include neuropsychiatric testing, sleep, movement disorders, neuromuscular, neuro-ophthalmology, stroke, skull base surgery, neurosurgery update, neurological infectious disease, MS and Drug Delivery systems.

This meeting will also give us the chance to greet the new Dean of the Dell Medical School. Dr. Johnston is a neurologist with a long experience in patient care, teaching and stroke research. He will moderate the Friday afternoon session. We welcome Dr. Johnston to Texas and to the TNS.

Residents from the neurology programs across the state have been invited to attend and to submit posters. It is important to meet our young colleagues, and we welcome them to become active in our society

The TNS meetings are a great chance for education, CME credits, re-connecting with friends and colleagues and plain enjoyment of the meeting.

2015 WINTER CONFERENCE

Austin Hilton Hotel
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Register today at
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Expert Opinion

End of Life Decision Making and Texas Law: A Review for the Neurologists

Joseph S. Kass, MD, JD,
Associate Professor of Neurology, Psychiatry, and Medical Ethics
Baylor College of Medicine

Whether involved in the chronic care of patients with neurodegenerative diseases or the acute care of patients suffering intracerebral catastrophes, neurologists must often guide patients and/or their family members through the end-of-life decision-making process. To feel comfortable delving into such discussions, neurologists should be familiar with the legal framework that supports end-of-life decision-making. This article is meant to serve as primer to aid neurologists in understanding Texas law related to end-of-life decision-making. This article is not meant to replace consultation with an ethics committee and/or legal counsel.

Planning for Care at the End of Life: Advanced Directives

In the United States, the courts and legislatures have established the right of patients and patient surrogates (when the patient lacks decision-making capacity) to refuse and withdraw life-sustaining treatment. If a patient possesses decision-making capacity, the patient makes his or her own treatment decisions. However, Texas law, through the Texas Advance Directives Act ("TADA"), recognizes that illness and injury can rob patients of decision-making capacity and therefore allows Texans three types of advance directives to help communicate treatment preferences in the event that decision-making capacity is lost: (1) Directive to Physicians (TADA Section 166.033); (2) Out of Hospital DNR Order (TADA Section 166.081); and (3) Medical Power of Attorney (TADA Section 166.151).

The Directive to Physicians, commonly called a "living will," allows a person with intact decision making capacity to express in writing a choice about the type of treatment he or she desires at the end of life should decision making capacity be lost. Importantly, the Directive to Physicians takes effect only when the person lacks decision-making capacity and suffers from either a terminal or irreversible condition. A terminal condition is an "incurable condition caused by injury, disease, or illness that according to reasonable medical judgment will produce death within six months, even with available life-sustaining treatment provided in accordance with the prevailing standard of medical care" (TADA Section 166.002). An example of a terminal patient is one suffering from a fast growing, aggressive brain tumor who will die regardless of heroic measures to sustain circulation and respiration. An irreversible condition is a "condition, injury, or illness: (a) that may be treated but is never cured or eliminated; (b) that leaves a person unable to care for or make decision for the person's own self; and (c) that, without life-sustaining treatment provided in accordance with the prevailing standard of care, is fatal" (TADA Section 166.002). Examples of irreversible conditions are patients in vegetative or minimally conscious states and those suffering from advanced dementia or amyotrophic lateral sclerosis.

Although an individual must have decision-making capacity to execute the Directive to Physicians, he or she does not need capacity to revoke it. Furthermore, the Directive to Physicians form does not need to be notarized, but two qualified witnesses must sign it (see TADA Section 166.003 for details about witness qualifications). A physician may be protected from legal liability by following the Directive but may face legal penalty for not following it (see below for a legally sanctioned approach not to follow the Directive).

A Directive to Physicians has important limitations. Although helpful in providing guidance to surrogate decision makers and the healthcare team, a Directive cannot anticipate all nuances of clinical care. Additionally, healthy individuals may overestimate the burdens of disease and disability when drawing up their Directive to Physicians. The Directive to Physicians requesting withdrawal or withholding of life-sustaining treatment does not apply to pregnant women, since a person may not withdraw or withhold life-sustaining treatment from a pregnant woman (TADA Section 166.049).

The Out-of-Hospital DNR is the second type of advance directive established in TADA for patients with terminal or irreversible conditions. An Out-of-Hospital DNR directs health care professionals, for example paramedics, acting outside a hospital not to initiate CPR and other resuscitative measures. Either a physician acting under a Directive to Physicians or a surrogate decision maker can execute an Out-of-Hospital DNR on behalf of the patient. The biggest problem with this type of advance directive is that the document may not be available when needed. However, TADA allows for a DNR identification device such as a DNR bracelet or necklace to serve as "conclusive evidence" of a valid Out-of-Hospital DNR (TADA Section 166.090). The form can be tricky to fill out properly, as it requires signatures in multiple places of multiple people.

A Medical Power of Attorney is the third type of advance directive allowed under TADA. In a Medical Power of Attorney, an individual with capacity designates an agent to make deci-

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sions on his or her behalf in the event the individual loses medical decision-making capacity. The Power of Attorney comes into effect any time an individual looses medical decision-making capacity, even if he or she is not suffering from a terminal or irreversible condition. The agent acting under the Power of Attorney cannot be the individual's healthcare or residential care provider or an employee of these enterprises (unless a relative of the individual). Furthermore, the agent is unable to consent for voluntary inpatient mental health services, electroconvulsive therapy, psychosurgery, abortion, or omission of care primarily intended to provide comfort. Furthermore, the agent must make health care decisions "(1) according to the agent's knowledge of the principal's wishes, including the principal's religious and moral beliefs; or (2) if the agent does not know the principal's wishes, according to the agent's assessment of the principal's bet interests" (TADA Section 166.152).

The Patient Who Lacks Both Capacity and an Advance Directive

Neurologists are often called to manage end-of-life care situations in which a patient lacks decision-making capacity and has neither a Directive to Physicians nor a designated Power of Attorney. TADA provides for a hierarchy of surrogates who may make end-of-life decisions for the terminally or irreversibly ill patient. Importantly, the TADA hierarchy of surrogate decision maker applies only to qualified patients who are terminal or irreversible, not to routine medical treatment decisions. The Texas Consent to Medical Treatment Act (Health and Safety Code Chapter 313) addresses the issue of consent for medical treatment unrelated to a decision to withhold or withdraw life-sustaining treatment from a terminal or irreversible patient and establishes a hierarchy of surrogate decision-makers, similar to, but not identical to TADA.

Under TADA "if the patient does not have a legal guardian or an agent under a medical power of attorney, the attending physician and one person, if available, from one of the following categories, in the following priority, may make a treatment decision that may include a decision to withhold or withdraw life-sustaining treatment: (1) the patient's spouse; (2) the patient's reasonably available adult children; (3) the patient's parents; or (4) the patient's nearest living relative" (TADA Section 166.039). Importantly, the surrogate decision maker must make the decision "based on knowledge of what the patient would desire, if known" (TADA Section 166.039). The treatment decision must be documented in the patient's chart and signed by the attending physician. If the patient lacks a legal guardian, a Power of Attorney, or a surrogate from the list above, the patient's attending physician may make a treatment decision to withhold or withdraw care with the concurrence of "another physician who is not involved in the treatment of the patient or who is a representative of an ethics or medical committee of the health care facility in which the person is a patient" (TADA Section 166.039).

Conflict Over the Goals of Care at the End of Life

Neurologists are no strangers to conflict with surrogate decision makers at the end of life. The decisions surrogates are asked to make are fraught with difficulty and may result in a difference of opinion between the surrogate decision maker and the healthcare team. TADA Section 166.046 provides a mechanism for the attending physician not to honor a directive or treatment decision if the physician believes the decision is medically inappropriate. TADA spells out a specific mechanism that must be followed to allow the physician to disregard the decision-maker's decision. First, an ethics or medical committee must review the attending physician's decision and must agree with the attending physician's decision. However, the patient or surrogate must be provided 48 hours notice and the right to attend the ethics/medical committee meeting. Either the patient or surrogate must receive a written explanation of the decision reached. The decision must also be recorded in the medical chart. There should be a reasonable effort to transfer the patient if the attending, patient, or surrogate does not agree with the committee's decision. If the attending physician refuses to carry out a request for life-sustaining treatment and the committee supports this refusal, then treatment can be discontinued on the 10th day after the written decision is given to the patient or surrogate. A judge can extend this 10-day period if there is a reasonable likelihood of finding an alternate provider.

Defining Death

TADA applies to patients who are alive and suffering from either a terminal or irreversible condition. However, TADA does not cover patients who have already died. The Texas Health and Safety Code Sec. 671.001 defines death by two means: (a)"irreversible cessation of the person's spontaneous respiratory and circulatory functions" or (b)"irreversible cessation of all spontaneous brain function." In patients who are artificially supported and suffer death due to cessation of brain function, "[d]eath must be pronounced before artificial means of supporting a person's respiratory and circulatory functions are terminated. " Thus, when a physician, using "ordinary standards of medical practice," declares the patient to have suffered an "irreversible cessation of spontaneous brain function," the patient is dead. After the declaration of death, there should be no controversy about withdrawal of "support," since the patient is legally deceased. There is no "code status" discussion once the person is pronounced dead. The hospital may follow its policies regarding the handling of a dead patient.

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Multiple Sclerosis Mimics: Case Presentations

John A. Lincoln, MD, PhD

Abstract

Magnetic resonance imaging (MRI) is a valuable diagnostic tool in multiple sclerosis (MS), currently allowing the identification of patients likely to develop disease even after the first clinical symptom. However, MRI, once an important adjunct tool to a comprehensive neurological evaluation, is currently often used as the primary tool in evaluating patients with non-specific or otherwise vague clinical presentations. Especially in this setting, the presence of atypical T2 hyperintense abnormalities can lead to misdiagnosis. This article describes two cases that were initially improperly diagnosed as MS and reviews imaging findings that should raise suspicion for MS mimics.

Introduction

The diagnostic tools available to neurologists have advanced considerably over time. Imaging technology is one tool that allows us to visualize abnormalities associated with chronic disease even at very early stages. Magnetic resonance imaging (MRI) has profoundly decreased the average time needed to diagnose multiple sclerosis (MS). Revised consensus criteria allows for the diagnosis of MS even after the first clinical symptom, given that an asymptomatic active lesion (ie. enhancement following administration of gadopentetate) is seen on baseline MRI (1). A crucial but often overlooked caveat in utilizing MRI for diagnostic purposes is that often "typical" T2 hyperintense MS lesions are at least 3 mm in size, oriented perpendicular to the callosal plane and are present in specific sites of the brain or spinal cord (figure 1) (2). Additionally, it is important to remember that prior to making the diagnosis of MS, there should be no better explanation for the patient's clinical symptom (3).

Neurologists often evaluate patients who present with waxing and waning symptoms without clear objective exam findings. In addition, patients are often referred by primary care physicians having already had brain imaging for vague symptoms. Imaging findings of non-specific T2 hyperintensities are often misinterpreted as evidence of a demyelinating process and change in these abnormalities a surrogate for chronicity, possibly leading to misdiagnosis. Therefore, it is important to remember that though imaging remains a very useful tool, multiple sclerosis remains a clinical diagnosis.

This article describes two cases with waxing and waning clinical symptoms that were incorrectly diagnosed as MS and reviews findings on imaging that should raise concern for MS mimics.

Case 1 –

Case 1 Presentation –

AB is a 52 year-old, right-handed Caucasian male with history of poorly controlled hypertension, who presented to his primary care physician with new stabbing headache involving the temples. The headaches occurred infrequently but persisted hours when present without exacerbating or relieving features. In addition, he complained of fatigue and "heat intolerance" described as a worsening headache, fatigue and issues with memory when exposed to increased ambient temperatures.

He had sustained trauma to the head on two occasions, one that occurred five years earlier after a severe motor vehicle accident resulting in prolonged hospitalization with associated amnestic event. He works as general contractor

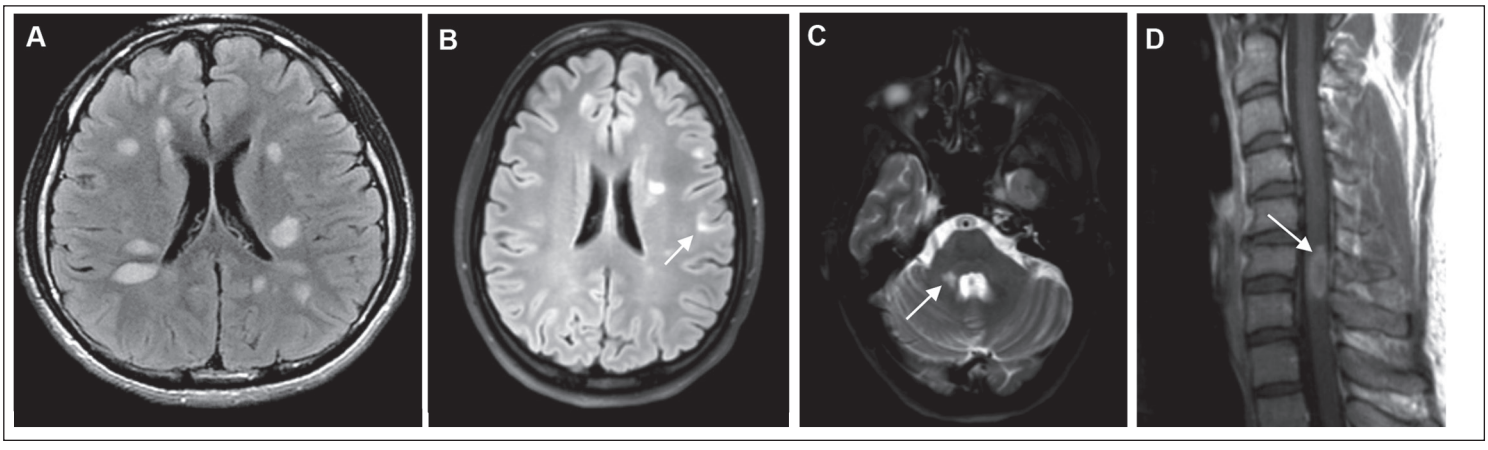

Figure 1: Brain and cervical spine MRI showing a "typical" appearance and location of MS lesions. (A) T2-fluid-attenuated, inversion recovery (FLAIR) sequence showing typical periventricular lesions are seen each measuring greater than 3 mm in diameter, generally ovoid in appearance and oriented perpendicular to callosal plane corresponding to the course of venules. (B) T2-FLAIR sequence showing juxta-cortical lesion involving cortico-cortical connecting fibers. (C) T2 sequence showing lesion involving the right middle cerebellar peduncle (infratentorial lesion). (D) Post-contrast T1 sequence showing enhancing cord lesion

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and complained that over the past one to two years has been having more issues because of "memory difficulty".

His general physical exam was normal. He scored 28 out of 30 in the Montreal Cognitive Assessment with errors only in serial sevens. There were no deficits noted on cranial nerve exam, formal motor or sensory testing. There were no extrapyramidal signs. Deep tendon reflexes were normal throughout and plantar responses were flexor bilaterally.

Given the initial presentation of headache, fatigue and memory deficit, his primary care physician obtained MRI of the brain (figure 2). Imaging showed numerous discrete T2 hyperintense foci located throughout the deep white matter oriented perpendicular to the ventricular plane. There were two T2 hyperintensities in the body of the corpus callosum though none involved the callososeptal interface. There were no clear infratentorial, juxtacortical or periventricular hyperintensities and few T1 hypointense foci present. There were no intramedullary signal changes within the spinal cord. Comparison to a brain MRI performed two years prior to the initial evaluation showed no interval change in T2 load. Despite the atypical appearance but based primarily on analysis of imaging, both his primary care physician and another neurologist told AB that he had MS.

His cerebrospinal fluid (CSF) showed no white blood cells and normal protein. There were no oligoclonal bands (OCBs), increased IgG synthesis or index and myelin basic protein within normal range.

Case 1 Discussion –

Traumatic brain injury (TBI) can result in either focal brain or diffuse axonal damage (4,5). Focal injuries can occur as a result of an object striking the head or the brain striking the interior of the skull often causing extradural, subdural or intraparenchymal hematomas. Diffuse axonal injury (DAI) often occurs as a result of rapid acceleration-deceleration leading to damage along white matter connections (4–6) with focal T2 hyperintensities observed on MRI. Damage commonly involves the axolemma and neurofilament resulting in disruption of axonal transport (7). In addition, secondary damage can occur due to ischemia, inflammation and Wallarian-type degeneration (7).

Clinically, both headache and cognitive deficits have often been reported after TBI with disorders of cognition involving areas of attention and memory integration (8). Though AB's headaches are likely unrelated to concussion, the abnormalities on imaging are consistent with that previously reported.

Multiple sclerosis is often considered part of the differential diagnosis in patient's presenting with waxing and waning symptoms. However, it is important to remember that a waxing and waning characteristic is not unique to MS. AB's clinical history provides important clues that might suggest alternate diagnoses including his previous repeated closed head injuries. In addition, though there were discrete T2 hyperintensities on brain MRI, the location of abnormalities in the deep white matter but not juxtacortical, infratentorial or spinal cord regions did not fit consensus MS diagnostic criteria (1). Finally, though not necessary for diagnosis, the lack of lesions within the cord, negative spinal fluid analysis and stable longitudinal brain MRI suggested the diagnosis of static encephalopathy likely due to diffuse axonal injury.

Case 2 –

Case 2 Presentation –

JP is a 34 year-old, right-handed African-American female who has carried the diagnosis of MS for three years. In 2011, she presented with headache, painful vision loss of the left eye and enhancement of the optic nerve on MRI. In 2012, despite having been on a disease modifying therapy (DMT) approved to treat MS, she experienced three discrete episodes of lower extremity weakness and gait imbalance each associated with severe pressure-like headache and persisting for nearly one month. Each episode improved after treatment with intravenous methylprednisolone followed by prolonged oral prednisone taper. She presented to hospital in 2014 with a one-month history of progressively worsening confusion with visual hallucinations and an inability to move her legs.

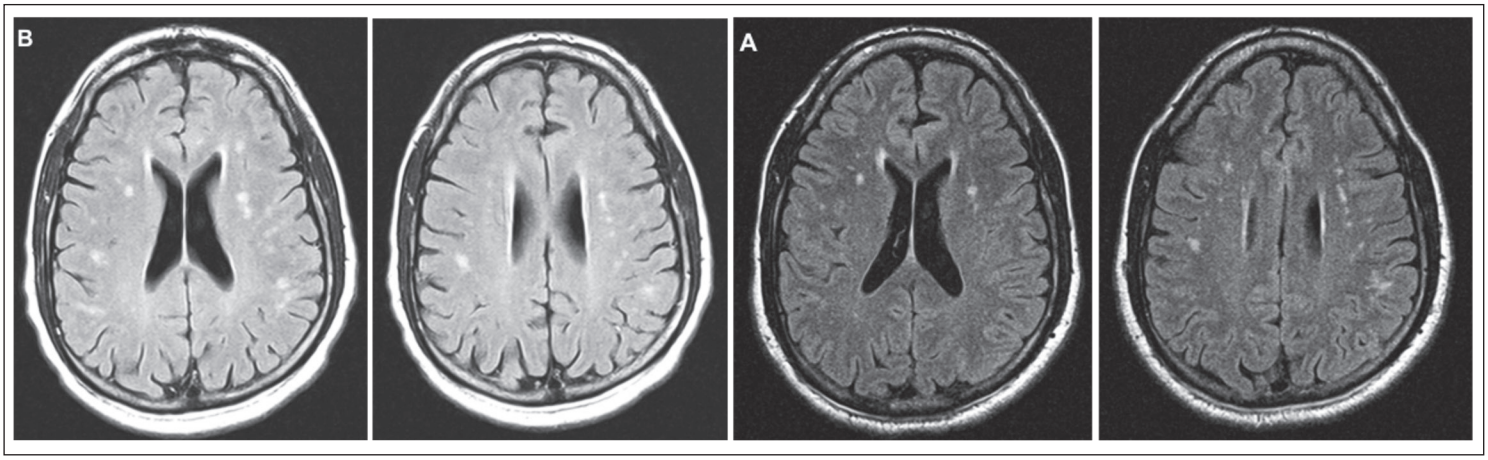

Figure 2. (A) T2-fluid-attenuated, inversion recovery (FLAIR) sequence for AB showing multiple somewhat discrete T2 hyperintensities located primarily in the deep white matter. (B) Comparison T2-FLAIR sequence from scan performed three years prior to initial clinical visit showing stable appearance of T2 abnormalities.

Other than the diagnosis of MS, JP had no other medical problems and, at least prior to the most recent hospitalization, had not complained of difficulty breathing, chronic cough or sore throat, swollen lymph nodes or abdominal pain.

Her general physical exam was normal. She was awake but not oriented and had fluent nonsensical speech. There were no deficits noted on cranial nerve exam. She had spontaneous and full movements of her arms but no spontaneous or stimulus-induced movements of the legs. Deep tendon reflexes in the arms were problematic to obtain given her mental state but clearly hyperactive in the legs bilaterally with spontaneous extensor plantar responses bilaterally.

An initial brain MRI from 2011 and subsequent image obtain during her hospitalization in 2014 are shown in figure 3. Images show several non-discrete T2 hyperintense

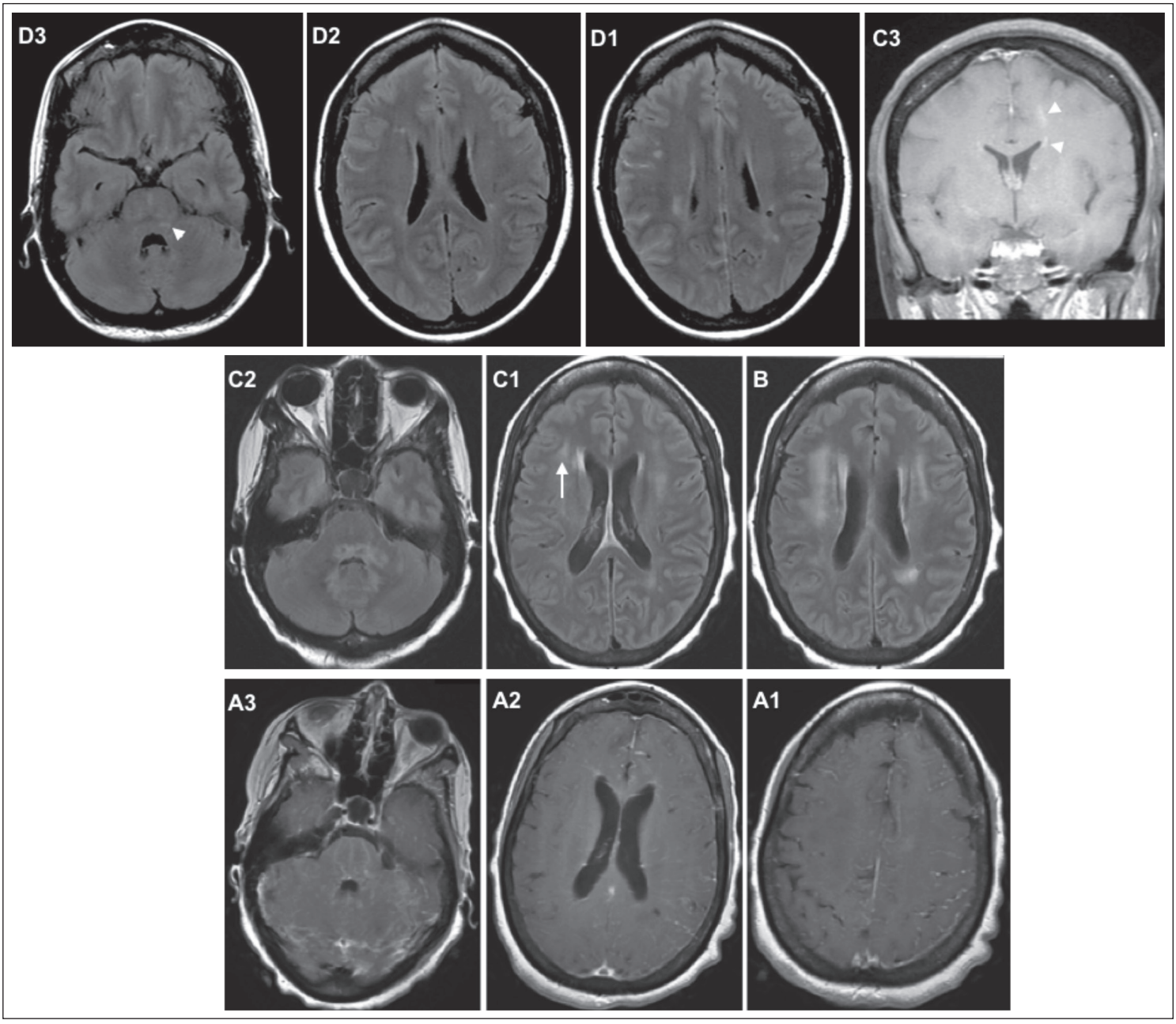

Figure 3. (A1-3) Baseline MRI, T2-fluid-attenuated, inversion recovery (FLAIR) image, for SD showing multiple hyperintense areas involving the brainstem (white arrowhead), adjacent to the lateral ventricles and deep white matter (B) two of which enhanced after administration of gadopentetate (white arrowheads). (C1-3) MRI, T2-FLAIR image, performed 3 years later for JP showing increased T2 load suggesting chronicity. Note that the majority of T2 hyperintensities are located in the deep white matter and are not discrete lesions. Additionally, a lesion near the cortex (white arrow) fails to involve the cortico-cortical connecting fibers as commonly seen in MS. (D1-3) Gadolinium-enhanced T1 image, showing waspy pattern on enhancement involving the pons as well as leptomeningeal involvement.

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periventricular, subcortical and deep white matter abnormalities. The scan from 2014 shows interval worsening of T2 lesion burden with involvement of the posterior pons and left middle cerebellar peduncle. In addition, the 2014 MRI showed diffuse leptomeningeal enhancement.

JP had CSF analysis on several occasions. She persistently had between 9 and 15 white blood cells, predominantly leukocytes, with elevated protein, no OCBs and normal IgG index and synthesis rate. Testing for various infectious etiologies, including Treponema pallidum, Borrelia burgdorferi and varicella and west nile virus PCR were negative. CSF cytology failed to show abnormally appearing lymphocytes. Both serum and CSF angiotensin converting enzyme (ACE) levels were normal on multiple occasions.

JP was treated with high-dose intravenous methylprednisolone with improvement but not resolution of the encephalopathy. She underwent biopsy of the right frontal lobe including the dura that showed non-necrotizing granulomatous inflammation predominantly involving the leptomeninges with normal neurologic cytoarchitecture, consistent with the diagnosis of neurologic sarcoidosis. She had whole body PET imaging that failed to reveal involvement of any other organ system.

Case 2 Discussion –

Central nervous system sarcoidosis is rare, occurring in only between 5% to 16% of persons with sarcoidosis (9,10). The pathophysiology of sarcoidosis is thought to be related to an abnormal immune response mediated in large part by resident cells, such as macrophages, capable of releasing tumor necrosis factor-alpha (11–13). Though isolated parenchymal involvement can be seen, typically neurologic sarcoidosis is thought to develop from granulomatous inflammatory meningitis that then extends into the brain parenchyma (14–16).

Clinical manifestations of neurologic sarcoidosis occur only in about half of cases, often presenting with cranial neuropathies, headache, peripheral neuropathy and less commonly encephalopathy (10,16–20). Cranial neuropathies are the most common manifestation with the facial nerve most often involved (10). The optic nerve is also affected with damage related to granulomatous infiltration or compression with resultant atrophy and often permanent vision loss (18). Headache is a common symptom with causes including meningitis, mass lesion and hydrocephalus (18,21). Finally, various neuropsychiatric symptoms, including depression, psychosis or encephalopathy can be seen with neurologic sarcoidosis (21,22).

JP's initial presentation was consistent with a demyelinating rather than granulomatous condition and, despite the negative CSF results, it was reasonable to consider the diagnosis of MS. Given compelling data on the benefit of early therapy for MS (see review by Thrower, (23)), starting treatment with a DMT for the presumed diagnosis of MS was also initially appropriate.

Indeed, in this case the MRI might have technically satisfied revised MS consensus criteria (1). However, when scrutinizing lesions adjacent to the ventricles, it is apparent that the majority are not discrete and are parallel rather than perpendicular to the callosal plane. The somewhat atypical appearance of her initial and especially subsequent brain MRIs raised the strong possibility of an MS mimic, with brain biopsy suggestive of neurologic sarcoidosis.

Conclusions

There are several publications detailing the many disorders that can mimic clinical and imaging characteristics associated with MS and a complete review of MS mimics is beyond the scope of this article (3,24,25). It is important to recognize that for the majority of patients presenting with classic clinical symptoms and signs, MRI and ancillary testing suggesting MS that this will ultimately prove to be the correct diagnosis. However, when clinical presentation and imaging are not entirely typical, one should always consider the potential for alternate diagnoses that can mimic MS.

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Member News:

Dr. Ninan T. Mathew received his third Lifetime Achievement Award at the American Headache Society's annual scientific meeting in Los Angeles on June 27, 2014.

Legislative Update

By Greg Herzog

The 2014 General Election has concluded and for the first time in over a decade, the Texas Legislature will convene with new leadership in every position elected at a statewide level. Governor-elect Greg Abbott; Lieutenant Governor-elect Dan Patrick; and Attorney General-elect Ken Paxton will be major players in healthcare policy this session. Additionally, the Senate Committee on Health and Human Services will have a new Chairman, Charles Schwertner, MD (R-Georgetown). Sen. Schwertner replaces Sen. Jane Nelson who is tapped to lead the Sen. Finance Committee.

Now that the election cycle is over, legislators have begun pre-filing bills for next session. Already legislation affecting healthcare is being filed. You can search and track any legislation at www.capitol.state.tx.us

Texas enters the 84th Legislative Session in great fiscal shape. The Texas economy and oil and gas production have left state coffers flush. Big challenges, however, await state leaders. Transportation and water issues will require significant effort and funding. Also, the Texas Supreme Court is reviewing the constitutionality of how the state funds its public education system. Complicating these needs are campaign promises of the newly elected; google this article for more information: http://www.myfoxhouston.com/story/27297216/post-election-tax-relief-in-the-works

Healthcare issues at the Texas Legislature only seem to be mounting. Among the many health related issues expected to be discussed:

- Requiring Physicians to Post Prices/Estimates for patients (TMA Led Proposal): http://www.texmed.org/Template.aspx?id=30971&terms=bonnen
- Attempts to Ban Out of Network Balanced Billing and require physicians to attend mediation: http://www.tahp.org/press-releases/305-tahp-call-for-reforms-on-health-care-billing
- Changing End-of-Life standards: http://www.kvue.com/story/news/state/2014/11/17/fort-worth-end-of-life-case-may-weigh-on-legislature/19163363/
- Ubiquitous Scope of Practice Attacks including the Sunset Commission's call to remove Chiropractor's ability to perform school physicals: http://www.chirotexas.org/index.php?option=com_content&view=article&id=492:sunset-commission-votes-to-recommend-that-chiropractors-be-removed-from-performing-school-physicals&catid=20:site-content
- Residency Program slots updated at long last? Will this be the year that state funds residency slots to match the growth of population?

TNS leadership will be monitoring these and other bills for impact.

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