TEXAS NEUROLOGICAL SOCIETY WINTER 2016

Broca's Area

The Voice of Texas Neurology

Mark Your Calendar

2016 SUMMER CONFERENCE

July 15-16, 2016

La Cantera
Hill Country Hotel
San Antonio, Texas

President's Message

Robert F. Leroy, MD

The Texas Neurological Society remains strong representing the neurologists in Texas as they care for people with neurological problems. TNS has a stable membership consisting of half the neurologists in Texas. The members are largely in private practice with 1 to 3 members in their groups. They have a mixed acceptance of electronic medical records, CMS quality programs and mid level providers. CME is the main reason that members belong to TNS. TNS is the largest and best organized state neurology society. The Winter Meeting in Austin continues to present a diverse program of neurological topics and is expanding the mix to include medical economics. The Summer Meeting has been growing each year for the past five years. Last summer, the meeting was in Fort Worth sharing the topics with the Southern Headache Society. The Executive Board and associated committees have expanded the role of TNS into the realm of practice management issues developing programs both during the meetings and teleconferences between meetings. In addition, TNS has entered into an advocacy role representing neurological interests in the Texas legislature and communicating with the AAN on a national level. Currently, we are preparing the response for the neurologists to the cannabidiol treatment in refractory epilepsy. There are future issues with balanced billing which are being discussed with the TMA, licensure for neurodiagnostic technicians and Texas standards for stroke center qualifications. TNS wishes to invite members to participate in leadership roles and education roles. Transition of generations is important. Future goals are to involve more of the academic neurologists in the membership especially through the residency programs. We are discussing promoting the TNS meetings to a more regional audience of neurologists. It has been an honor working on the board of the TNS and serving this year as president.

Speaker Ryan and the AAN

By Mike Amery, Esq.
ANN Senior Legislative Counsel, Federal Affairs

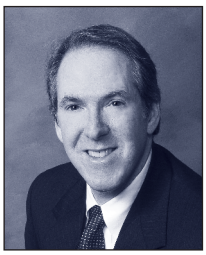
On October 29, 2015 Republicans officially elected Rep. Paul Ryan of Wisconsin as the 54th Speaker of the U.S. House of Representatives. The AAN has had a great relationship with Ryan and his staff for several years.

I attended a fundraising dinner for Ryan on the night before former Speaker John Boehner announced his plans to step down. I really don't think Ryan saw this coming as the discussion surrounded his efforts as chair of the House Ways & Means Committee and all the political intrigue that night revolved around work to get agreement on spending bills and the looming debt ceiling.

Ryan, who has risen through the ranks as a respected policy wonk on budget and health issues, has always understood the issues impacting physicians and was a leading force in the repeal of the SGR prior to it reaching the House floor. AAN members in Wisconsin have the ear of the new speaker. AAN Government Relations Committee member Donn D. Dexter, MD, FAAN, met with Ryan during the August recess and he has frequently met with our Neurology on the Hill advocates.

Another refreshing thing about Ryan is his sense of humor. Ryan knows that I am a native of Minnesota, which makes our next-door neighbors in Wisconsin frequent rivals. No rivalry is bigger between these two states than the NFL's Minnesota Vikings and the Green Bay Packers. Ryan and I were mingling before an event one morning when he said, "Mike, you're from Minnesota, right?" After I said yes he asked, "Do you know what this is?" as he balled his hand in a fist and put it just inches from my nose. I said "No" and he responded, "It's a Minnesota Viking Super Bowl ring." Of course, there were no rings on his fingers, symbolizing the number of championships won by the Vikings.

I'm looking forward to working with Speaker Ryan, even if he is a Green Bay Packer fan.



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 5-7. Reeta Achari, program director, Gary Clark, pediatric director, Bob Fayle, education committee chair, and the education committee have planned an excellent program. And this year, thanks to the efforts of Dr. Black and the Medical Economics Committee, we have a 2 hour Sunday afternoon practice management symposium.

Sir Thomas Browne and Neologisms

What do the following words have in common: ambidextrous, anomalous, approximate, ascetic, carnivorus, coma, computer, electricity, expectoration, gymnastics, ferocious, follicle, hallucination, inconsistent, indigenous, indoctrination, incisor, insecurity, medical, migrant, prairie, suicide, veterinarian, and zoology? Amazingly, they were all coined by Sir Thomas Browne.

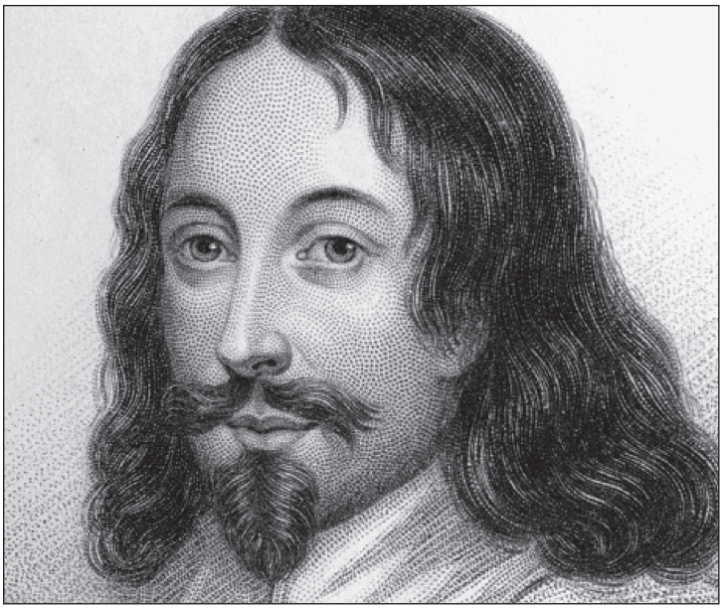
The Oxford English Dictionary credits Sir Thomas Browne (1605-1682; figure 1) with coining 784 new words (Aldersey-Williams H. In Search of Sir Thomas Browne, Norton, 330 p, 2015; Collins J. The cracked archangel. Wall Street Journal, June 26, 2015; Hilton D. Sir Thomas Browne and the Oxford English Dictionary. Available at http://blog.oxforddictionaries.com/2012/08/sir-thomas-browne/; Breathnach CS. Sir Thomas Browne (1605-1682). J R Soc Med. 2005;98(1):33-6).

Born in London and educated at Oxford, Browne practiced medicine in Norwich after studying medicine in Europe. Unlike his contemporary, Sir Thomas Willis, as a physician he made no major discoveries but is remembered from his writing and invention of words which he did on the side. He wrote books on funerary practices through the ages (Urn Burial), gardening and mathematics (The Garden of Cyrus), profession of faith (Religio Medici), a catalog for an imaginary museum of conjectural works of art and literature (Musaeum Clausum), and a compendium of vulgar scientific errors (Pseudodoxia Epidemica). [His books are available at http://penelope.uchicago.edu/]

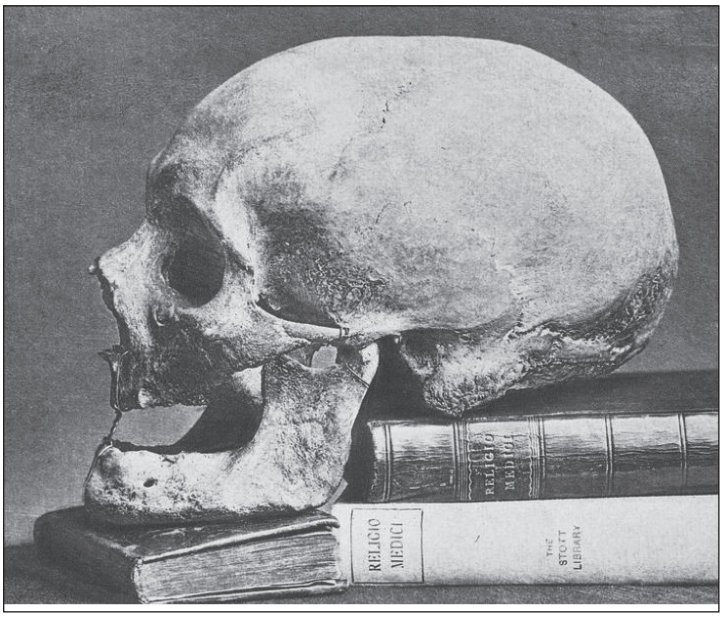
Brown dazzled other writers including Samuel Taylor Coleridge, Virginia Woolf, Herman Melville, and Jorge Luis Borges, and other physicians including William Osler and R.D. Laing. Examples of his famous verse are available on the web (http://www.brainyquote.com/quotes/authors/t/thomas_browne.html; https://www.goodreads.com/author/quotes/53520.Thomas_Browne).

As a bizarre and sad aside, his skull (figure 2) was removed from his coffin by the sexton in 1840, purchased by a local surgeon, and later kept in a local medical museum until being re-interred in 1922. Perhaps presciently, Browne had written in 1658, "But who knows the fate of his bones, or how often he is to be buried? To be gnawed out of our graves, to have our skulls made drinking-bowls, and our bones turned into pipes to delight and sport our enemies, are tragical abominations (Dickey C. The fate of his bones. Cabinet; 2008; available at http://www.cabinetmagazine.org/issues/28/dickey.php).

So the next time you're at the computer using electricity writing a medical note about an ambidextrous patient who is in coma, or reports a history of an hallucination or suicide attempt, think of Sir Thomas.



Sir Thomas Browne (photo: Alamy)



Thomas Browne's skull atop copies of 'Religio Medici.' (Photo: Wellcome Library, London)

Medical Reversals

Some of you may remember Woody Allen's 1973 movie, "Sleeper." Allen plays Miles Monroe, the owner of a Greenwich village health-food store, "The Happy Carrot," who is subjected to cryopreservation without his consent and defrosted 200 years later. The future brings amazing reversals in medical thought.

Two scientists discussing Monroe's recovery:

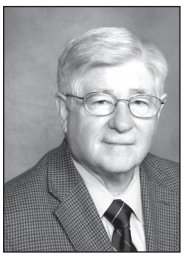
"**Dr. Melik:**	Well, he's fully recovered, except for a few minor kinks.
Dr. Agon:	Has he asked for anything special?
Dr. Melik:	Yes, this morning for breakfast. He requested something called wheat germ, organic honey and tiger's milk.
Dr. Agon:	[laughs] Oh, yes. Those were the charmed substances...That some years ago Were felt to contain life-preserving properties.
Dr. Melik:	You mean there was no deep fat? No steak or cream pies? Or hot fudge?
Dr. Agon:	Those were thought to be unhealthy, precisely the opposite of what we now know to be true.
Dr. Melik:	Incredible."

And

"**Dr. Orva:**	Here. You smoke this, and be sure you get the smoke deep down into your lungs.
Miles Monroe:	I don't smoke.
Dr. Orva:	It's tobacco. It's one of the healthiest things for your body. Now go ahead. You need all the strength you can get."

Forty two years later, with increasingly sophisticated and expensive studies, methodology, and technology, we have so many flip flops in all areas that many patients have less confidence in medical practice. If you want to learn more about why, I recommend that you read a new book by hematologist/oncologist, Vinayak Prasad, and internist, Adam Cifu (Ending Medical Reversal: Improving Outcomes, Saving Lives, Johns Hopkins, 2015) which defines "medical reversal" as the overturning of currently accepted therapy. They offer many examples including medical and surgical treatment and diet, explore why (explain evidenced based medicine and review sources of flawed data), and suggest solutions including changes in medical education and academia. You may not agree with all of the examples of treatments they believe have been reversed which is part of the challenge of dealing with data. They estimate that about 40% of medical practice is either not effective or is harmful.

If you don't have time or don't want to buy the book, you can read the full text online of their two articles which were the basis of the book (Medical Reversal: Why We Must Raise the Bar Before Adopting New Technologies available at http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3238324/; A Decade of Reversal: An Analysis of 146 Contradicted Medical Practices available at http://www.mayoclinicproceedings.org/article/S0025-6196(13)00405-9/abstract).



Start Making Plans Now

By Robert W. Fayle, MD
Chairman, TNS Education Committee

The Winter Meeting of the Texas Neurological Society is scheduled for Friday, February 5 through Sunday, February 7, 2016. This meeting's program director is Reeta Achari, MD, and she has worked diligently to put together a high quality meeting, as those who know her would expect.

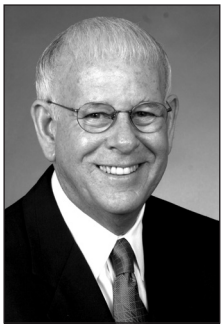
Dr. Clark has also arranged an excellent pediatric session. Both pediatric and adult neurologists will find this section to be of interest. Sleep disorders in children, the transition of the adolescent patient to an adult neurologist, the ethical and legal aspect of medical marijuana, and myths of childhood concussions will all be covered.

For the adult section there are programs scheduled on concussion, chronic traumatic encephalopathy, and Texas concussion law all of which are pertinent to our practices. There will be a very practical presentation on Spine MRI interpretation and discussions on Demyelinating Disease, Stroke, Movement Disorders and Dementia. All the speakers are well known and knowledgeable. This year's meeting looks like another great winter meeting. The poster competition is again open to residents in all the residency programs across the state.

At noon on Friday there will be a "Out of the Box" lecture on the nature of consciousness by Daniel M. Price, PhD, which promises to be very interesting.

The meeting will take place at the Austin Hilton. Please consult the meeting brochure for the registration details and room rates for conference attendees.

We hope to see everyone who are not on call at the meeting which will be current and very educational. We expect to have a good turnout…and a good time!



New Payment Rules For Neurologists

Stuart B. Black, MD
Chair, TNS Medical Economics Committee

Most Texas Neurologists are now aware that the Sustainable Growth Rate formula (SGR) for physician compensation was repealed in April, 2015. Created in 1997 as a part of the Balanced Budget Act, the SGR was designed as an effort to restrict Medicare B spending by comparing the growth in physician Medicare services spending to a growth rate linked to the gross domestic product. The SGR never succeeded because it was not anticipated that the cost of medical care would quickly exceed the growth of the gross domestic product. Since the passage of the SGR, Congress has voted to pass short-term legislation, or "patches" 17 times at an expense of approximately $170 billion dollars to prevent decreases in physician reimbursements for Medicare patients. The 2015 cut in physician reimbursements would have been 21%. On April 14, 2015, one day before CMS was required to begin processing the 21% decreased Medicare reimbursements for physician claims, a new legislation was approved by a 392 to 37 vote in the Senate following an earlier overwhelming 393 to 37 bipartisan vote in the House of Representatives, to replace the SGR. President Obama signed the legislation into law within 24 hours of passage in the Senate. The new law, which is called The Medicare Access and CHIP Reauthorization Act (H.R. 2), or MACRA, not only replaces the SGR but is a legislative advocacy which includes sweeping reforms for the way physicians will be paid for Medicare services. The new law focuses on Medicare's emphasis on value-based payments; which also means it emphasizes transition away from the traditional fee-for-service payment model as we know it today toward a more risk-based, value-incentivized physician payment model. MACRA will eventually create two tracks for physician compensation. One is an Alternative Payment Model (APM) and the other is the Merit-Based Incentive Payment System (MIPS). It is important that physicians understand MACRA as these new Medicare Provisions will map out Medicare payments for the next ten years and beyond. Since indemnity insurance usually follows the lead of Medicare for physician reimbursements, it is likely some form of MACRA will also dictate physician reimbursements from private payers; thus it is predictable that all physician compensation will change whether it is Medicare or private insurance.

Alternative Payment Models (APMs)

MACRA encourages the creation of APMs and provides different incentives for physician participation. Some examples of alternative payment models include: Accountable Care Organizations (ACOs) and other shared savings plans, pay-for-performance plans, patient centered medical homes (PCMH), bundled payment models (which may include carve outs for episodes of care) and capitation (which was unsuccessful in earlier HMO programs). MACRA incorporates Alternative Payment Plans as payment models which follow the recommendations of the Center for Medicare & Medicaid Innovation (CMMI). Under MACRA, to qualify as an APM Medicare participating doctor, the physician must meet annual increasing thresholds which define the percentage of their revenue they receive through an APM. The thresholds are defined under MACRA and require participating physicians to take financial risk for the cost of their medical care of patients. Quality measures must also be reported by use of a certified EHR. Starting in 2019 (just three years away), physicians in APMs must derive at least 25% of their Medicare Part B payments from the APM.

While qualifying APM doctors will be eligible for bonuses stating 2019, partial qualifying APM participants will be assessed under MIPS (the second MACRA alternative discussed below), but will receive credit for the APM work in the form of higher composite scores which will add to income. The timeline for APM requirements is as follows:

2019-2020:	25% of Medicare revenue must be received through APMs For partial APM participants, 20% of revenue must come from Medicare
2021-2022:	50% of Medicare revenue or 50% of all payer revenue along with 25% of Medicare revenue must be received through the APMs For partial APM participants, 40% of revenue must come from Medicare
2023 and beyond:	75% of Medicare revenue or 75% of all-payer revenue along with 25% of Medicare revenue must be received through the APMs For partial APM participants, 50% of revenue must come from Medicare

From January 2016 to December 2019, all physicians will continue to receive the 0.5% increase in Medicare payments which began June 2015. Starting 2019, APM physicians will receive a 5% lump-sum bonus on all Medicare payments for 2019 through 2024. Starting 2026, the APM participating physician will qualify for a 0.75% increase in payments each year. Those not in an APM will receive a 0.25% annual update.

As of the present time, only a relatively small number of Neurologists nationally are involved in some type of APM. Alternative payment models are also extremely expensive to finance, organize and manage. That is the reason why at the current time larger APMs fit the definition of Accountable Care Organizations usually organized by large healthcare systems. However, in many areas of the country, ACOs are not even available for Neurologists to join and when available, Neurology representation in decision making or leadership within the ACO is usually absent. To assist Neurologists around the country, the American Academy of Neurology is investigating the potential opportunities of developing independent APNs centered on neurologic patients and their disorders. The potential opportunities of developing Physician-focused Payment Models (PFPMs) with the focus

on neurological diseases are being addressed by the AAN. PFPMs would need to include access to "big data" and resources for data management and analysis. Financial resources would also need to be indentified to help practices fund the data infrastructure investments to monitor the performance measures necessary to qualify for an APM. There are a multitude of economic and non-economic factors that the AAN is addressing in trying to assist Neurologists across the country toward meeting the APM obligations as well as exploring the potential opportunities needed to develop PFPMs for those practices or groups of practices motivated to become an APM. The final product of any model must demonstrate specific ways it will improve patient care and result in lower health care spending.

Merit – Based Incentive Payment System (MIPS)

For those physicians not in an APM, the second alternative payment track under MACRA is the Merit – Based Incentive Payment System (or MIPS), which starts in 2019. MIPS is essentially a new addition to the current Medicare fee-for-service payment model and is focused on uniting and hopefully improving the three existing quality programs. In addition a fourth performance category is added. The Physician Quality Reporting System (PQRS) will be 30% of MIPS, Value-Based Modifier (VBM) 30%, Meaningful Use (MU) 15% and the new fourth performance category Clinical Practice Improvement Activities 15%. Each year physicians will receive a payment which will be adjusted according to the prior year's performance. MIPS will be the only reporting program for Medicare for those physicians not participating in an APM. According to the Medicare description of MIPS, unlike the current quality reporting system where physician performance and subsequent bonuses are measured against other physicians in similar clinical environments, performance assessment under MIPS would be physician specific and based on a sliding scale. The current VBMs concept of "winners" and "losers," currently exists because someone may even be performing on a higher level. This model would be eliminated. As defined in MIPS, if all the physicians performed at or above their designated performance threshold, no one would get a penalty. In addition, credit would also be given to those physicians who only partially met the performance metrics.

The current PQRS, MU and VBM programs will remain in place until the end of 2018. Under the present separate reporting programs, in 2019 physicians are faced with a potential 2% penalty for not reporting PQRS, 5% penalty for not meeting EHR MU requirements and potential ± 4% penalty for negative adjustments under VBM. Under MIPS, these penalties will be eliminated at the end of 2018 and doctors will be paid on their MIPS adjusted payment rate beginning Jan. 1, 2019. The four performance categories under MIPS will be used to make up a composite performance score (0 – 100) to determine payment adjustments. Physicians will also be given credit if their quality and resource use performance measures improve from year to year.

Performance threshold levels will be determined by either the mean or median of all scores during a performance period. Starting 2019 the positive and negative Medicare payment adjustments will start at 4% and gradually increase to 9% in 2022. For example, physicians who score at the threshold level and earn the predicted composite score will be neutral and receive no payment adjustment; those whose composite score is above the mean will receive a positive payment adjustment on each claim the following year; those whose composite score is below the mean will receive a negative payment adjustment on each claim the following year. In addition, physicians who score the highest composite scores in their respective specialties will be eligible for a positive payment that could be 3Xs the baseline payment adjustment for a year. Thus, if the 2019 positive payment adjustment will be 4%, the highest performers could receive a payment adjustment of 12%. Medicare has indicated that for "exceptional performers", an additional positive payment adjustment of up to 10% is available from 2019 through 2024.

We have all also recently been introduced to the chronic care codes. Chronic care codes will be used under MIPS. The use of these codes would require Medicare to reimburse a physician, under one code, for monthly care management services for individuals with chronic care needs. Since payment would only go to one physician, usually in a patient centered medical home or comparable practice which would need to be certified by a recognized organization, the use of these codes and reimbursement for Neurologists (say for treating MS patients) is still in early stages.

While the programs under MACRA appear to have a number of physician positive features, the big question remains how difficult it will be to become a high or exceptional performer and will the opportunities for bonuses be equally distributed among Neurologists in different practice settings. Specifically will private practice Neurology groups who are not in a hospital provider network have the same opportunities under MACRA as the multidisciplinary hospital based Neurology practices? Will private practice Neurologists in solo practice have the time, resources and financial backing to meet the projected threshold levels?

Funding

Another important question is where is all this money coming from to fund MIPS? As it turns out, the Federal Government has set aside a $500 million annual pool to be used to fund "exceptional performance".

This new financial incentive does not exist within the current Medicare reporting programs. The yearly $500 million is available for physicians in MIPS who reach the highest performance levels from 2019 through 2024. The office of the Secretary of Health and Human Services will determine those physicians who are eligible for the additional awards from the $500 million a year pool. Those physicians participating in an APM would not be eligible for this bonus. The APM physicians would receive a 5% incentive payment from 2019 – 2024. Part of the source of all this additional Government money which will be used to offset the cost of repealing the SGR and introducing MACRA will be a result of cuts in Medicare payments to hospitals and to post-acute providers. But patients will also be affected. In addition to decreased

Continued on page 7

We Bid Farewell to Four of Our Own

Steven L. Linder, MD 1945-2015

Steve L Linder, MD was beloved by both his colleagues and patients. He blazed a trail by becoming one of the first Child Neurologists who specialized in Headache in the State of Texas. Dr. Linder started his path to becoming a physician when he obtained his doctor of medicine degree at the University of Illinois at Chicago College of Medicine. Next, he completed a pediatric internship at Children's Medical Center in Dallas Texas. Finally, he completed his Child Neurology training at University of Texas Health Science Center in Dallas. Dr. Linder also proudly served his country as an Army Pediatrician and obtained the rank of Major by the time his commitment ended. In 1978, Dr. Linder founded Dallas Pediatric Neurology.

A recognized pediatric neurology expert, Dr. Linder was a frequent speaker in the US and internationally on pediatric headaches and the field of pediatric neurology. He authored numerous journal articles to educate pediatricians and neurologists about acute and preventative pediatric headache treatments. He loved to educate and share his knowledge with residents and physicians. He was always willing to spend time one-on-one with adult neurologists to educate them about pediatric headaches. Dr. Linder recognized most teenagers would need to see Adult Neurologists as there are very few Child Neurologists.

At a time when there was very little information available to the public, Dr. Linder produced a series of videos and pamphlets to educate patients and their parents on pediatric headache and migraine. To help his young patients to express themselves, he had his patients "draw their headaches". One way he communicated with his younger children was to use a mouse puppet. The mouse (and Dr. Linder) had a love for sweets and patients would commonly bring M&M chocolate candies to "feed the mouse."

He retired from medical practice in May 2015 shortly before his death. He was survived by his wife Cathy, daughter Heather Linder Blackmar, of Houston, son-in-law, Bruce Blackmar, three grandchildren; Steven Blake Blackmar, Brooke Patricia Blackmar and Brynne Catherine Blackmar

Ninan Mathew, MD 1937-2015

Dr. Ninan Mathew was born on May 21, 1937 in Kerala, India and passed away on July 27, 2015. He did his post-graduate training in neurology at Christian Medical College, Vellore, India and fellowship training in cerebrovascular disease at Baylor College of Medicine where he was a faculty member.

In 1976, Dr. Mathew established the Houston Headache Clinic, the first headache specialty center in Texas which was a major referral and research center for 35 years. In 1984, he started the Dallas Headache Clinic.

Dr. Mathew served in many professional organizations including as president of the American Headache Society and International Headache Society and chairman of the Headache Section of the American Academy of Neurology and the American Council for Headache Education. He was one of the founding members of the international research group on cluster headache and chaired international meetings.

Dr. Mathew significantly contributed to medical literature, with more than 200 scientific publications, in journals such as JAMA, Lancet Neurology, Neurology, and Headache. These include the first description of transformation of episodic migraine into chronic daily headache (transformed migraine or chronic migraine); clinical importance of medication overuse in determining the progression of chronic migraine; and the importance of detoxification. He wrote numerous book chapters and was co-author of Handbook of Headache and guest editor of two issues of Neurologic Clinics. He was a member of the editorial board of Headache and Cephalalgia and a frequent peer reviewer for Neurology, Headache, Cephalalgia, and Lancet.

Dr. Mathew was a fellow of the American Academy of Neurology and was on the faculty of the courses offered at the annual meeting of the Academy. He was an invited lecturer on headache in medical meetings and universities in the United States and abroad. He was featured in a 2002 headache cover story in Time Magazine appeared on numerous radio and television programs for the National Migraine Foundation and the American Council for Headache Education.

Dr. Mathew trained a number of physicians from various countries who did fellowships at the Houston Headache Clinic. He participated in clinical trials of new medications for headache therapy and directed more than 100 studies.
Dr. Mathew was recipient of s everal American Headache Society Awards, including the 1976 Harold G. Wolff Lecture Award and the 1994 John R. Graham Distinguished Clinician Award. He received lifetime achievement awards from the Texas Neurological Society in 2012, the Headache Cooperative of New England in 2013, and the American Headache Society in 2014.

Dr. Mathew was involved with various cultural institutions. He was a founding member of the Indian Doctor's Club in Houston and served as its second president. He was president of the India Cultural Center also in Houston and was on the advisory board of the Asia Society Texas Center. He was a founding patron of the Hobby Center for the Performing Arts and the Asian Galleries of the Museum of Fine Arts, Houston. His interests included traveling, antiques, and gardening.

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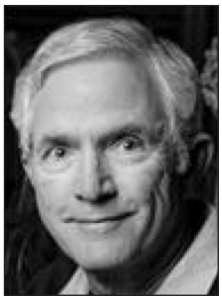
He is survived by his wife of 53 years, Sushila Mathew, 3 children, and 6 grandchildren. or legal changes have the potential to impact every specialty's ability to contract with health plans.

Jean Moure, MD 1931-2015

The daughter of two physicians, Dr. Jean Moure was born in Glasgow, Scotland on July 20, 1931, raised in Northampton, England, and passed away on November 6, 2015 in Carmel, California. She did her neurology residency and fellowship in neurophysiology at Baylor College of Medicine in Houston. Dr. Moure specialized in epilepsy and sleep disorders. She was chief of neurology at Park Plaza Hospital in Houston from 1975-1987 and was also head of the EEG department at MD Anderson. She retired in 1991. She is survived by her husband, neurosurgeon Dr. Antonio Moure, 3 children, and 4 grandchildren.



Jerry Ray Tindel passed away on October 7, 2015

Jerry was born in Alvin, Texas in 1942. His medical life story included a Bachelor degree from the University of Texas School of Pharmacy in 1965, his MD degree from the University of Texas Medical School in Galveston in 1969, internship at University of Indiana followed by Neurology residency at the Mayo Clinic 1970-1973. He then joined the Kelsey-Seybold Clinic in Houston for 2 years. In 1975 he moved to Austin to reunite with Mayo Clinic colleagues and friends to continue building the neurology section of the Austin Diagnostic Clinic where he continued to practice for 33 years. Jerry's patients appreciated and respected his neurology expertise and his calm and caring nature. Jerry was also the director of the Muscular Dystrophy Center at Brackenridge Hospital for 15 years; became Neurology Section Chief at A.D.C. for years and served on the A.D.C. Board of Directors.

Jerry's life outside of neurology included the Eagle Scout Award, service in the Army Reserves, photography, tennis and golf. Most important of all he was a great family man. Jerry and Barbara were happily married for 48 years and enjoyed spending time with their daughters Allison and Chelsea, their husbands and 3 grandchildren.

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New Payment Rules...

payments to hospitals and providers, there will also be changes to Medicare benefits to offset the cost of MACRA. "First dollar" Medigap coverage for new Medicare enrollees will be eliminated for new Medicare enrollees beginning 2020; Medigap plans for those beneficiaries would only be able to cover costs above the Part B deductible. In addition, there will be increases in the percentage of Medicare premiums for both Parts B and D that high income beneficiaries must pay beginning in 2018. Currently, the beneficiary income is determined by the prior year income, which potentially could be an issue if an individual retires and then signs up for Medicare. While retirement may depend upon a lower modest fixed income, the Medicare premium will be determined according to higher income earned the year before when the retiree was employed. Since Medicare premiums are projected to rise considerably, paying for health insurance may be a potential financial burden for the retiree. As an example, it may be that a single senior reporting income of more than $133,500 and married couples with income more than $267,000 will see their share of premiums rise from 50% to 60%. Single Medicare eligible participants reporting income above $160,000 and married couples with income above $320,000 might see premium increases rise from 65% to 80%. Again, however, if indeed income remains based upon the prior year's tax return, it is conceivable that various individuals retiring with lower fixed incomes may find the new Medicare rates unaffordable.

Conclusion

Repeal of the SGR did not just do away with a legislation that had been plaguing physicians for nearly two decades. The law that replaced the SGR contains many provisions that will impact how physicians deliver care now and into the future. There will also be additional modules incorporated within the new legislation as the years go by.

While various potential components of MACRA may have appealing features to some Neurologists, there is still much concern that this new legislation could reward certain Neurologists at the expense of others. Neurology is a relatively small specialty with variances between large group practices in metropolitan areas and solo practitioners in large cities as well as in smaller communities. How will MACRA affect the income of different Neurologists in different models of practice? There is already a national shortage of Neurologists with challenges to future workforce numbers. Given the unknowns and variability's of this new legislation, it is imperative that Neurologists today learn to understand the two components of MACRA, specifically APMs and MIPS. These new reimbursement programs will ultimately determine future physician compensation irrespective of the practice design. We must think of this new legislative advocacy as a long term project and position our career choices within the framework of very different future payment alternatives. Neurologists must begin planning for these reforms well before the initial effects of MACRA become the final reimbursement model.

The Co-Existence of Epileptic Seizures and PNES: Limitations of Outpatient Ambulatory Video-EEG


Sasha Alick, MD

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Video-electroencephalography (video-EEG) is an important diagnostic tool for epilepsy diagnosis. Typically, video-EEG was utilized in people with epilepsy whose seizures do not respond to medical therapy. People with medically refractory epilepsy may be identified as surgical candidates with video-EEG monitoring or their epilepsy may be better characterized for alternative medication or neurostimulation therapies. In pediatric epilepsy, it is also utilized to screen children for any evidence of epileptic seizures before committing them to chronic antiepileptic therapy and its potential complications. Antiepileptic medications are usually maintained during brief evaluations that last 24 hours, but are withdrawn in longer studies lasting longer than 24 hours in order to record seizures. As some patients with medically-refractory epilepsy may have psychogenic nonepileptic seizures (PNES), video-EEG monitoring has also become the gold standard for their diagnosis.

While ambulatory EEG was available for several decades, it recently celebrated a resurgence with the added video capabilities. Clinicians do not have to admit their patients to the hospital for video-EEG monitoring, and patients can undergo the evaluation in the comfort of their homes. During an evaluation, which may last as long as five days, technical staff can check on the integrity of the recording and electrode placement intermittently via the internet. Insurance companies have also embraced ambulatory video-EEG as a way to eliminate the cost of hospitalization. Despite its clear utility, just as with routine standard electroencephalography (EEG), there are still important limitations, even in the diagnostic realm. These shortcomings can be technical, related to inadequate recording due to artifacts, and the inability to examine a patient during or immediately after a seizure. Most clinicians would agree that ambulatory video-EEG would be most useful to record events in people suspected to have PNES. However, as seizure medications cannot be reduced in the outpatient setting, what can be missed is the co-existence of epileptic seizures and PNES. In order to demonstrate these limitations of ambulatory video-EEG evaluations, and their potential consequences, we describe two patients who were admitted in the same week for inpatient video-EEG monitoring to University Hospital in San Antonio. One patient was thought to have PNES by ambulatory video-EEG monitoring, suffering from an exacerbation of seizures after she was taken off her medications, even losing her medical health coverage. The second patient would have been erroneously diagnosed with only PNES had his medications not been withdrawn. Hence, in both patients, discontinuation of antiepileptic medications played a crucial role in achieving the appropriate diagnosis.

CASE ONE:

This is a 62 year-old right-handed woman with a history of epilepsy starting at age seven years old. She denied seizure risk factors, besides being dropped on her head while an infant, with an unclear history of loss of consciousness. There was no family history of seizures or epilepsy. She was born full term in an uneventful pregnancy with good prenatal care. She met all developmental milestones in a timely manner and completed high school in regular classes. During initial consultation, she described her events as beginning an "out of body" feeling, sometimes associated with an inner feeling of tremulousness. She then described shaking of her head and arms with subsequent loss of awareness. The episodes occurred on a daily basis and at times they would cluster. Stress was the most common trigger for her events. She had rare generalized motor seizures, which she described in a similar fashion, except that she would vocalize, fall to the ground, and convulse, occasionally biting her tongue and losing urine continence. She was on phenytoin, clobazam, and levetiracetam when she underwent ambulatory video-EEG monitoring. She had been on valproic acid, phenobarbital, carbamazepine, and topiramate previously. A VNS was implanted in 2001 and the battery was replaced in 2011. She believed the VNS was effective, as her events tend to subside whenever she successfully activated the generator with the magnet. A 72-hour ambulatory video-EEG had captured several events without EEG correlate, suggesting that all her episodes were nonepileptic, and no interictal epileptic discharges were recorded. She claimed to have experienced her typical auras with the feeling of tremulousness but did not develop any of her motor seizures or spells. Nonetheless, her events were considered to be PNES and subsequently her medications were stopped. When her seizures or spells recurred, her antiepileptic medications were restarted with improved control. In the meantime, her Social Security payments were discontinued and she no longer received medical benefits. During her consultation outpatient visit at UTHSCSA, the patient had a spell with flailing head and arm movements, along with screaming and grunting. She became nonverbal, but was able to follow some commands. Decision was made to electively admit the patient for video-EEG to the epilepsy monitoring unit (EMU) with discontinuation of medications.

She was admitted to the EMU at University Hospital for three days. Her VNS was turned off and her AEDs were stopped on the first day of admission. Sleep deprivation, hyperventilation, and intermittent photic stimulation were performed. Her baseline EEG showed a normal posterior background, intermittent right temporal slowing, and paroxysms of right mid-

to posterior temporal spikes. During the first 24 hours she had 3 of her typical auras of "out of body" experience and/or inner tremulousness, all without an EEG correlate. Forty-eight hours into monitoring, she had 6 more of her typical auras with no EEG change and 3 focal dyscognitive seizures (one in the morning and two early in the afternoon). They began with her involuntarily clasping her hands behind her head, head nodding, vocalization (mainly sobbing) and left lower extremity kicking. Subsequent seizures were associated with unresponsiveness and postictal confusion as documented by EMU nurses' examinations. Left arm clonic activity and left version were noted late during the focal dyscognitive seizures. There was an ictal EEG discharge during her seizures, albeit poorly lateralized. Her last seizure was associated with right hemispheric rhythmical activity and spiking, mainly medially. Upon completion of evaluation, she was deemed to have focal epilepsy with right hemispheric onset. The seizures were thought to have a medial parietal or a mesial (interhemispheric) onset. She was ultimately discharged on her admission medications, but at increased doses.

CASE TWO:

This is a 60 year-old left-handed United States Air Force (USAF) veteran with a history of seizures since the age of 39. Other medical comorbidities include headaches, generalized anxiety, depression, insomnia, hypertension, hyperlipidemia, chronic pain, and osteoarthritis. His sister was treated for epilepsy as a child, which remitted at age 18. He denied any risk factors for epilepsy. He came for an initial consultation with his service dog after being followed for years at his local VA hospital and by community neurologists. Several routine outpatient EEG studies were documented as normal and brain MRI was reported as only revealing a venous varix (vs. possible aneurysm) in area of proximal aspect of anterior branch MCA on the left and an arachnoid cyst in the left Sylvian fissure. He mentioned a previous brain MRI also demonstrated "left temporal lobe atrophy". His first seizure was a generalized tonic-clonic seizure in 1994 with a fall from the stadium bleachers. He was subsequently unconscious in the hospital for longer than 2 weeks. He has no recollection of his clinical course while hospitalized. He was placed on phenytoin at that time, but his seizures were not controlled. He was subsequently discharged from the USAF and has not worked since. He reported daily events characterized by being awake and able to hear, but unable to move. He described behavioral arrest, frequently occurring during conversations, as "freezing." The events have no time preference and tend to last longer than a minute. Additionally, he described 'fugue states' during which he misses blocks of time for 1-2 minutes in duration. These were occurring 2-3 times a week. He states his seizure dog reliably alerts him prior to seizures and that he refrains from driving at those times. He denies ever being involved in a car accident as a result of his frequent seizures. He also described episodes of speech arrest with stuttering and headaches. None of these events were associated with postictal confusion. Stress was a trigger for all his events. He was previously treated with phenobarbital, primidone, lamotrigine, levetiracetam, valproic acid, oxcarbazepine, diazepam, topiramate, phenytoin, and carbamazepine. Topiramate was stopped due to sedation, carbamazepine caused SIADH, and lamotrigine caused a rash. He also took clonazepam for anxiety.

He was admitted to the EMU at UH for characterization of his events for four days. His AEDs were stopped on the first day admission. Sleep deprivation, hyperventilation, and intermittent photic stimulation were performed. His baseline EEG showed a normal posterior background and activation measures produced no abnormalities. During the first 24 hours of monitoring, he had three of his typical staring spells, during which he stopped responding to the nurses and his body became rigid while in a sitting position, which was evident from examthe examination by nursing. After thirty seconds, he recovered, speaking more lucidly than prior to the episodes. There was no EEG correlate with any of the spells. There were no interictal epileptiform discharges during the first 48 hours. On the third night of his stay, his EEG showed frequent generalized spikes, mainly in light sleep, but also during wakefulness. These were at times associated with muscle artifact, although no twitching or myoclonic jerks were visible on video recording. He confirmed that when he was younger and in the early phases of his treatment he had experienced generalized myoclonus. The inpatient psychiatry consultant diagnosed conversion disorder. He was diagnosed with PNES and genetic generalized epilepsy. He was discharged on zonisamide monotherapy with the diagnosis of concomitant PNES and juvenile myoclonic epilepsy. He was to follow-up in both epilepsy and psychiatry clinics.

DISCUSSION:

We describe two patients who were admitted for inpatient video-EEG monitoring for characterization of their seizures or spells. One patient (Case 1) had been diagnosed to have nonepileptic spells based upon a 72-hour ambulatory video-EEG monitoring, and not only lost her insurance coverage and disability, but had been withdrawn from seizure medications. She was found to have only epileptic seizures on her inpatient scalp monitoring after her medications were discontinued, though her first auras were not associated with an EEG correlate. The second patient (Case 2) did not undergo ambulatory video-EEG monitoring but was referred for suspected left temporal epilepsy. Not only was he diagnosed with psychogenic nonepileptic seizures (PNES), but after being off his seizure medications for 72 hours, he was also diagnosed with genetic generalized epilepsy. With an ambulatory video-EEG monitoring, he would have also lost his disability coverage and perhaps he would have even been taken off his seizure medications. Hence, the co-existence of epilepsy with PNES poses a serious challenge to neurologists managing patients with medically refractory epilepsy.

Seizures can be divided into three major categories, epileptic seizures (ES), PNES, or physiologic nonepileptic events. They all present with paroxysmal alterations in motor, sensory, and/or cognitive signs and symptoms. Unlike epilepsy, PNES are not caused by ictal epileptiform activity (LaFrance & Devinsky, 2004). Epileptic seizures are a manifestation of excessive and hypersynchronous discharges in the brain, which can be recorded using EEG (LaFrance et al, 2013). PNES tend to have a psychological etiology, and are not associated with an EEG correlate. Physiologic nonepileptic events are associated with systemic alterations, such as hypoglycemia or cerebral hypoperfusion, as in syncope. PNES is classified with the diagnosis of conversion disorder (functional neurological symptom disorder) with a specific symptom type of attacks or seizures, found un-

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der a new category called somatic symptom and related disorders in the current DSM-5 (DSM-5, 2013). PNES are commonly seen at epilepsy centers, where they represent approximately 20% of patients referred for refractory seizures (Benbadis et al, 2000). Epilepsy is a recognized risk factor for the development of PNES. The combination of epilepsy and PNES occurs in about 10% of patients with PNES. The proportion of patients with a dual diagnosis of PNES/ES has been reported to vary from 10% to over 50%, likely due to relatively "loose" criteria for coexisting epilepsy and a number of normal variants overread as interictal epileptic discharges (IEDs) on EEG (Benbadis et al, 2001). Even numbers generated by inpatient video-EEG monitoring evaluations may underestimate the true prevalence of this co-morbidity, as some patients may demonstrate only epileptic seizures after medication withdrawal. The correct diagnosis and proper management of patients with non-epileptic seizures decreases mean medical costs by 84% in the six months following diagnosis (LaFrance & Benbadis, 2006).

The main advantages of ambulatory EEG is the lower cost when compared to long-term in hospital monitoring and the ability to record subjects in their typical living environment, where they may be exposed to their usual seizure triggers, i.e. family and daily life stressors. Ambulatory EEG allows for the detection of IEDs in up to 25% of cases when routine EEG and prolonged sleep deprived EEG examinations are normal (Waterhouse et al, 2003). It also permits the detection of seizures, which are not identified or reported by the patient, making pertinent treatment changes feasible. Another benefit is the opportunity to record the natural sleep cycle and evaluating the circadian rhythm of seizures, especially for those which tend to occur during nighttime.

The advantages of inpatient video-EEG monitoring in an epilepsy monitoring unit (EMU) over ambulatory video-EEG monitoring are more significant. Inpatient video-EEG monitoring allows the identification and immediate correction of technical problems related to EEG recording, such as electrode artifact, equipment malfunction, and movement or myogenic artifact. Furthermore, patients can be clinically evaluated by EMU nurses during or after an event, which helps to determine whether cognitive changes are occurring even in the absence of EEG changes. Physical interactions between nursing and inpatients can also help elucidate functional deficits. Finally, it is also not possible to safely record patients off medications unless they are in an EMU setting. As some AEDs may decrease or even suppress interictal epileptic discharges, their reduction or discontinuation can enhance the sensitivity of inpatient video-EEG studies. Despite the increasing trend in utilizing ambulatory EEG due to convenience and reduced cost, continuous video-EEG monitoring in an epilepsy monitoring unit remains the gold standard for diagnosis, particularly in people presenting with multiple seizure or spell types, which should raise the suspicion for coexisting epilepsy and PNES events.

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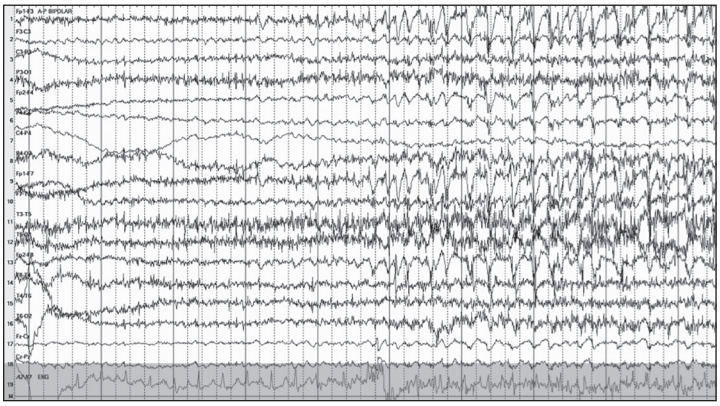
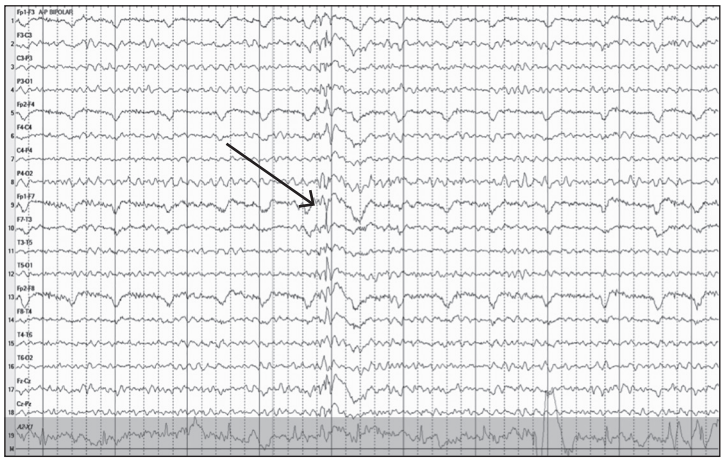
http://manuals.medtronic.com/manuals/search?region=US&cfn=37602&manualType=MRI+Technical+Manual

Figure 1. Non-lateralized focal onset seizure recorded on day 2 in Case #1



Sensitivity=20uV, LFF=1.6Hz, HFF=70Hz

Figure 2. Generalized spike-and-wave complexes recorded on day 3 in case #2



Sensitivity=7uV, LFF=1.6Hz, HFF=70 Hz

Compassionate Use in Texas: Cannabidiol for Intractable Epilepsy

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"Emily", A Composite Case

"Emily", a 15-year old female with an established diagnosis of juvenile myoclonic epilepsy (JME), returns to your office for follow-up with her mother. You established her diagnosis two years prior after her first generalized tonic-clonic seizure lasting 1-2 minutes. JME was suspected with a history revealing onset 6 months before that time of morning myoclonic jerks, a decline over the past year in school performance, and a maternal uncle with onset of convulsive seizures reported in his teens. EEG confirmed the diagnosis with brief interictal bursts of 4-5 Hz generalized polyspike and slow wave and capture of multiple myoclonic seizures. She is currently seizure-free on valproic acid, her third antiepileptic medication. She previously failed lamotrigine with breakthrough generalized tonic-clonic seizures at a therapeutic serum level, followed by zonisamide which was discontinued due to cognitive side effects at a low dose. She has been stable without further breakthrough seizures on a moderate dose of valproic acid (750 mg twice daily) for over a year, and a recent EEG demonstrated no seizures and rare fragmentary spikes. Her grades are back to her previous performance.

Today, Emily complains of weight gain, which she attributes to valproic acid after reviewing its side effect profile. She is concerned about its long list of potential adverse affects, specifically weight gain, birth defects, liver failure, osteoporosis, and hair loss. She reports having discovered "the amazing effect" of cannabis oil for "so many" patients with epilepsy, and she wants your advice about starting cannabis and coming off of her valproic acid. She states "I prefer natural methods over drugs" because of "all the side effects [with pharmaceuticals]". Her mother is mostly quiet, but when asked how she feels she states that she certainly wants Emily to remain seizure-free and believes "she is mature enough to decide…what goes into her body."

Many Texas neurologists, particularly those seeing a large percentage of patients with epilepsy, may have patients of their own whose recent questions and concerns are echoed in those of Emily. The Texas Compassionate Use Act, designed to provide access to cannabidiol-rich cannabis extracts for patients with medically-intractable epilepsy, was passed as Texas Senate Bill 339 (SB-339) and House Bill 892 (HB-392) and signed into law by Governor Abbott this year. With the bill in force since September, Texas neurologists should prepare themselves to advise their patients on the medical evidence for the effectiveness of cannabis-derived treatments of their conditions. In this rapidly changing intersection of medicine and law, it is often difficult for the individual practitioner to feel fully informed and thus prepared to responsibly advise their patients. To that end, we have prepared this review of the most recent clinical evidence for cannabinoid treatments in epilepsy and changes to expect from the law.

Clinical Evidence: Cannabinoids for Epilepsy

Marijuana is chemically complex, containing approximately 60 pharmacologically active compounds. Whereas its psychoactive properties come primarily from delta-9-tetrahydrocannbinol (THC), cannabidiol (CBD) appears to have little effect on cognition and has gained recent interest for its potential antiepileptic effects (Katona, 2015). Recently, the American Academy of Neurology conducted a systematic review of the evidence for various marijuana-derived treatments in all neurologic conditions, identifying thirty-four studies meeting their strict inclusion criteria, including eight class I studies. Such a review was admittedly complicated by the diversity of treatments and conditions falling under consideration, but refreshingly comprehensive. The group did identify quality evidence for efficacy of some treatments examined (namely, oral cannabis extract and nabiximoles) for multiple symptoms (spasticity, central or spasm-related pain, and urinary dysfunction) in multiple sclerosis. The evidence outside of multiple sclerosis, however, is much less encouraging: oral cannabis extract is probably ineffective in treating levodopa-induced dyskinesias in Parkinson's disease, and results in Huntington's disease, Tourette syndrome, and epilepsy are insufficient to determine its efficacy (Koppel et al., 2014).

A recent systematic Cochrane review, specifically focusing on efficacy and safety of cannabinoids for epilepsy, found similarly inconclusive evidence (Gloss and Vickrey, 2014). None of four randomized controlled trials identified addressed a modern primary outcome of efficacy (proportion of patients with seizure freedom at either one year or three times the longest seizure-free interval), though two studies (Cunha et al., 1980; Mechoulam and Carlini, 1978) did find efficacy by some measure. Across all four trials, there were no reported adverse effects from short-term (one to six months) exposure (in 48 patients total), and the Cochrane review authors conclude that the available data informs the secondary outcome of safety, indicating that cannabidiol may be safe for treatment. This conclusion is cautionary, however, in that no data about long-term administration is provided by these results. Due mostly to the limited number of patients and high risk of bias, "no reliable conclusions can be drawn at present regarding the efficacy of cannabinoids as a treatment for epilepsy" (Gloss and Vickrey, 2014).

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With patient access secured in some other states by more "comprehensive" medical marijuana statutes, however, much interest surrounds the emerging experience there. Pediatric neurologists at Mattel Children's Hospital in Los Angeles (Hussain et al., 2015) collected data from an online survey capturing responses from 117 individual patients' caregivers, and reported an overall efficacy of seizure reduction in 85% (100 patients). A group at Children's Hospital Colorado, in contrast, employed a more rigorous open-label chart review that required at least 2 points of follow-up contact for inclusion, defined efficacy as >50% seizure reduction reported by parents, and limited confounding factors by providers agreeing not to change other anitepileptic medications during treatment (Press et al., 2015). Under these conditions, the investigators found efficacy in only 33% of their 75 patients, with 57% of patients reporting any improvement in seizure frequency and 33% reporting other improvements (in behavior, language, or motor skills). Interestingly, with 45% of their patients having relocated to Colorado to obtain treatment (as the Colorado law requires residency for access), the strongest predictive factor for reported efficacy was relocation to obtain treatment, with an odds ratio of 3.16 compared to families with established care in the state. As the authors of both studies acknowledge, the risk of bias in such designs remains high, and definitive evidence of efficacy of treatment in any particular epilepsy condition awaits results from double blind, placebo-controlled trials.

Even as we await licensure of the first Texas dispensary, the first multicenter randomized trials of CBD in epilepsy continue. Results are completed and forthcoming shortly for treatment in Dravet syndrome, and enrollment has completed for treatment in Lennox-Gastaut syndrome. Within the year, the first Class I evidence addressing efficacy of CBD in epilepsy should be published.

The Law: SB-339/HB-392

SB-339 directs the Department of Public Safety (DPS) to establish rules and standards for the oversight of low-THC cannabis dispensaries in the state. These dispensaries will provide cannabis extracts with at least 10% by weight cannabidiol and at most 0.5% by weight tetrahydrocannabinol. The highlights of the bill's definitions of eligible patients, physicians, dispensaries and its timeline are presented in the Box. In keeping with a focus on providing cannabidiol only to intractable epilepsy patients, the bill specifically authorizes only those practitioners board-certified or board-eligible in epilepsy or neurophysiology to provide prescriptions. There is no ambiguity in the language to allow for alternate indications (akin to "off-label" prescriptions) or prescriptions by general neurologists or other specialists (Eltife, 2015).

The bill is unique compared to legislation in many other states in two important ways: it provides a means of access only for patients with medically-intractable epilepsy, and it requires a prescription, instead of a recommendation, from providers. Texas is not the first state to use the language of "prescription" in medical marijuana legislation, with such language first appearing in an initiative passed in Arizona in 1996. The legality of such prescriptions, however, remains dubious. There is legislation in each federal congress this year (H.R.1774 and S.683) that aims to exclude cannabidiol from the definition of marijuana and reclassify marijuana according to federal law. If this legislation fails, however, marijuana, THC, and CBD will remain listed on Schedule I, whereby they are defined to have "no currently accepted medical use in treatment," and prescriptions for them will remain in violation of federal law (Snyder, 2015).

Highlights of SB-339 Patients, Physicians, Dispensaries

- ***Patients*** – "intractable epilepsy": two or more appropriately chosen and maximally titrated antiepileptic medications have failed to control seizures.
- ***Physicians*** – board-certified/board-eligible in epilepsy, or board-certified in neurophysiology. The physician:
  - Certifies the patient has
    - intractable epilepsy,
    - does not have "other treatment options… available or appropriate", and
    - a potential benefit of treatment that reasonably balances the risk of medical use of low-THC cannabis.
  - Registers a low-TCH cannabis prescription, including:
    - Physician name, patient name, patient date of birth
    - Dosage and dosage form, and
    - Total amount to be dispensed.
  - Maintains a treatment plan, including:
    - Dosage, dosage form, and planned dura tion
    - Monitoring plan for patient symptoms, and
    - Monitoring plan for tolerance or adverse reactions.
  - For minors (<18 years), a second qualified physician documents concurrence in the medical record.
- ***Dispensary*** – licensed by DPS to:
  - Cultivate, process, and/or dispense "low-THC cannabis": <=0.5% THC, >=10% CBD, and
  - Verify prescriptions and track dispensed quantities.
- December 1, 2015: registry established, rules for dispensaries released.
- January 1, 2018: at least 3 dispensaries shall be licensed.

Emily's Case: Some Final Thoughts

So how best to advise patients, such as Emily, who express interest in CBD? Emily's case, specifically, is somewhat unclear as to whether she qualifies for CBD treatment by the statute's language – "maximally titrated" includes failure due to adverse effects, but at what point do "minor" adverse effects such as weight gain constitute treatment failure? Perhaps

more importantly, the evidence available for cannabidiol in intractable epilepsy suggests that its utility may be highest in, or even limited to, particular syndromes such as Lennox-Gastaut or Dravet syndrome (Press et al., 2015); no data has addressed JME specifically. Thus, compassionate education about the medical evidence, the law, and the alternative approved treatments (including medications, vagus nerve stimulation, and diet therapies) is certainly the first most appropriate step.

Even if Emily were to adamantly request and qualify for CBD, however, the law has not quite yet made it available. Until such time as a DPS-certified dispensary is operational, marijuana and any of its derivatives remain prohibited by both state and federal statute. We will be watching and considering this situation as it continues to evolve, before deciding whether participation in Texas' experiment in limited compassionate access is in the best interest of our patients. We advise other Texas epileptologists and neurophysiologists to consider the same.

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Dr. Eddie Patton **(Houston)**

Selected as a member of the highly competitive 2016 AAN Emerging Leaders Forum. This forum is designed to identify, orient, and cultivate talented, motivated young individuals into possible future Academy leaders.

Advocacy Review 2015

Sara Austin, Chair, TNS Legislative Committee and Greg Herzog, TNS Lobbyist

As we close this year, I want to provide the TNS membership with a look back at the past Texas Legislative Session and a glimpse forward at issues expected to challenge our specialty in 2016.

The past year has provided TNS with several public policy challenges and opportunities. With scope of practice, TNS has been engaged on several fronts. During the legislative session, TNS worked to keep chiropractors from issuing handicap placards and also joined TMA in a lawsuit against the Texas Dental Board to bar dentists from diagnosis and independent treatment of sleep apnea patients. TNS also worked with a new group of healthcare providers, the Neuro-diagnostic Technologists (NDTs), who were seeking licensure. Even though, the legislation creating licensure for NDTs failed this session, we expect it to return next session and will continue to work with them.

Legislation that allowed for the prescription and use of low-THC cannabinoid oils for the treatment of epilepsy was an issue that TNS focused on during this session as well. TNS and organized medicine believes more research is needed on this topic and we are watching the ongoing rule-making process closely.

TNS attempted to include our specialty in a proposed extension of the now expired "primary care incentive' money in the Medicaid program. As you may know, neurology was excluded in the original definition of primary care set by CMS, but, TNS successfully amended the definition to include our specialty when the Texas Legislature considered extending this money. Success! Unfortunately, the entire proposal was cut in the final version of the state budget and no new physician reimbursement increases were included.

TNS was a leader in the effort to remove the so-called "occupations fee" from licensure renewal. Each payer of licensure fees will see a $200 reduction every year from this point forward.

Looking forward, TNS will be monitoring several rule-making proposals and the interim charges recently issued by the Texas legislative leadership. Additionally, we will monitor the Sunset review of the Texas Medical Board (TMB) as this review potentially impacts the entire set of licensure, discipline, and scope of practice rules governed by TMB.

TNS will also be watching the suggested changes to "surprise medical bills" or balanced billing for out-of-network patients. This issue has garnered national attention and has primarily been focused on hospital-based physician specialties. However, legislative or legal changes have the potential to impact every specialty's ability to contract with health plans. TMA and other groups will heavily discuss this topic in the coming weeks and months.

As always, continue to reach out to the state and national representatives and senators. Those contacts are valid to your profession and the house of medicine.

Teenager's Sleep Pattern and School Performance

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Introduction

Over the past 25 years, despite the increased of evidence based knowledge regarding the impact of sleep disorders on behavior, cognition and quality of life, the exact function of sleep still remains elusive.

Sleep patterns of teenagers have been extensively studied and have revealed considerable variations between school nights and non-school nights of rest. Total sleep time tends to be less on school nights when compared with non-school nights. Bedtime and wake times appear to be influenced by external factors such as school start times and changes during puberty to later sleep onset time resulting in a diminished total sleep time and daytime sleepiness.

What is a normal sleep pattern?

Sleep is classified into the two types: NREM (Non-rapid eye movement) sleep and REM (rapid-eyes-movement) sleep. Cycling through all of the sleep stages for an adequate amount of time is essential to achieving a good night's sleep and adequate functioning the next day. These sleep stages are defined by distinct polysomnographic features of electroencephalographic patterns, eye movements and muscle tone.

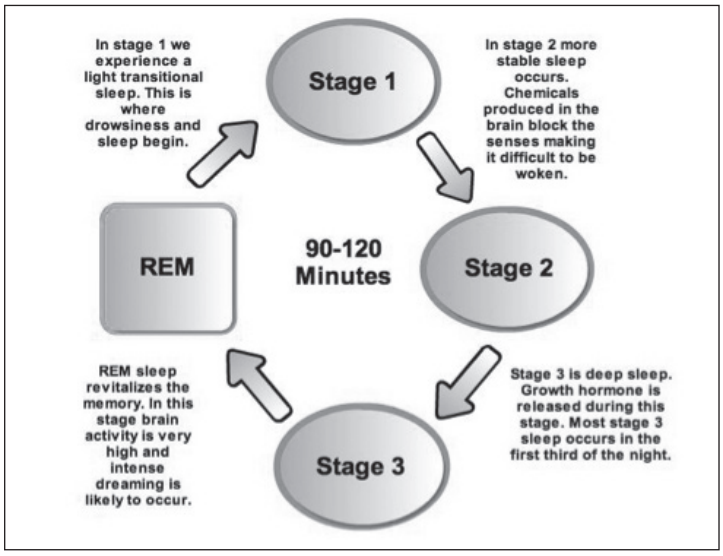

Fig. 1. NREM sleep and REM sleep cycles.

Non-REM sleep is characterized by distinct EEG patterns including sleep spindles, K complexes and slow (delta) activity. The respiratory and cardiovascular parameters are relatively regular. In contrast REM sleep, is characterized by asynchronized cortical activity with a high brain metabolic rate, dreaming, lack of normal thermoregulation, and irregular respiratory and cardiac rhythms. The hallmark features of REM sleep include absent of skeletal muscle tone with the exception of the diaphragm, middle ear and erectile muscle, and episodic burst of rapid eye movement.

Why is sleep important?

There are many theories concerning the need for sleep. However, what we know has primarily evolved from research studies conducted in animals and humans examining the impact of sleep deprivation on the physiological and neurobehavioral systems. During sleep important body functions and brain activity occurs to create new pathways for learning and memory. Insufficient sleep alters activity in some parts of the brain that may interfere with the ability of making decisions, alertness, solving problems, controlling emotions and behavior, and coping with change.

What is considered an appropriate sleep for teenagers?

The National Sleep Foundation released recommendations in February 2015 that school-aged adolescents (14 to17 years) should obtain at least 8 to10 hours of sleep per night. However, on average the amount of sleep that teenagers achieve is about 7 hours, particularly on school nights. Thus, teenagers are constantly coping with "sleep debt" during the school year. The amount of sleep reported by adolescents varias across countries and regions; but overall patterns of later sleep timing and diminished sleep across adolescents is reported by most investigators. If this sleep is cumulative, subjective and objective evidence of increased daytime sleepiness should appear.

The most recent US poll of sleep patterns in adolescents was reported by the National Sleep Foundation in 2006. This data showed that on average, adolescents get about seven and one-half hours of sleep on school nights. However, the amount of sleep varies by grade, with teenagers tending to get less sleep, as they get older.

This is of particular concern because chronic sleep deprivations, also known as sleep loss, insufficient or deficient sleep, leads to a myriad of health deficits. Disrupted sleep-wake cycles and sleep restriction contribute to significant negative effects on the renal, cardiovascular, thermoregulatory, digestive, and endocrine systems. For example, sleep loss can contribute to insulin resistance and the development of metabolic abnormalities, obesity, and diabetes mellitus.

Furthermore, inadequate sleep has also been associated with mental health and safety deficits. Sleep deprived teenegers have less interest to participate in physical activity or sports. They are more likely to be depressed, anxious, irritable, defiant, and impulsive than teeneager who achieve optimal sleep amounts. They are at increased risk for suicidal ideation, substance use, as well as motor vehicle accidents related to drowsy driving.

Sleep restriction has been linked to cognitive and behavioral problems that adversely impact academic performance and functioning. For example, teenagers who are chronically sleep deprived have worse academic performance. Teenagers achieving inadequate amounts of sleep have increased absenteeism and tardiness, decreased ability to learn and retain material, and diminished ability to actively participate in the classroom and perform decision-making tasks.

How does puberty alters sleep-wake cycle?

There are changes in the biological clock or circadian rhythms of teenagers. At about the time of puberty onset, most teenagers begin to experience a sleep-wake "phase delay" (later sleep onset and wake up time), manifested as a shift of up to 2 hours relative to sleep-wake cycles in middle childhood.

The onset of sleep is triggered by the release of melatonin, a natural body hormone. Toward dawn, melatonin shuts off and cortisol increases and also core body temperature rises, signaling the individual to wake up. Two biological changes in sleep regulation are thought to occur during puberty. First, there is a delayed timing of nocturnal melatonin secretion, that parallels a shift in circadian phase preference. Therefore, teenagers have a biological tendency to fall asleep later in the evening and to wake up later in the morning. Additionally, sleep drive is altered across adolescence. Even those teenager, who have experienced sleep deprivation (and therefore accumulated a sleep debt) tend to feel more alert in the evening, thus making it more difficult to go to bed at a time that parents might consider a reasonable hour. There is a further "mismatch" in that early school start times for adolescents and teens that do not allow them to achieve their biological need to have a later out-of-bed/wake time and achieve an adequate amount of sleep for optimal daytime functioning.

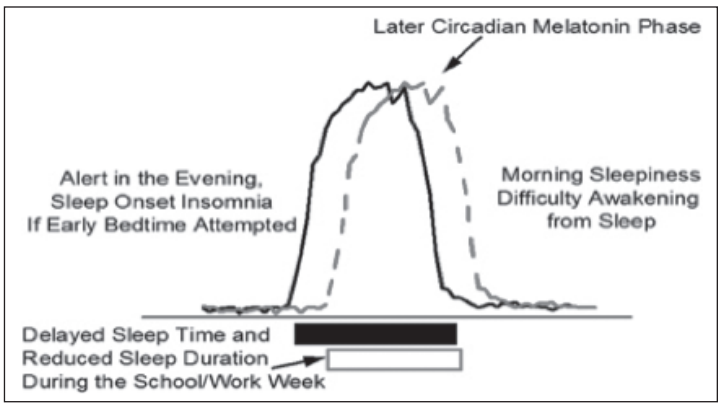

Fig. 2. Delayed sleep phase.

Are there other factors that contribute to sleep deprivation in adolescents?

There are others reasons why teenagers do not get enough sleep. For example, caffeine consupmtion is increasing among adolescents to fight against the daytime sleepiness, resulting from not getting enough sleep. More worrisone is the increasing consumption of energy drinks and "super caffeinated" products like caffeine pills and gums to promote alertness. Daytime and evening caffeine consumption may further disrupt nighttime sleep. The ability to achieve an appropriate sleep onset time and adequate amounts of sleep may be further impaired by after school activities (part-time work), socializing and electronic devices. Erratic sleep schedules, principally during non-school nights, in an attempt to compensate for the lack of sleep during school nights, seems to be a good idea, but can even make worse the situations. These behaviors impaired the ability to develop appropriate bedtime/sleep time and out of bed time/wake schedules that promote healthy and adequate sleep for teens. Later school start times, even as little as 30 minutes have been associated with improved academic performance and reduced vehicle accidents among teens.

What sleep disorder should be evaluated in a sleepy adolescent?

Among adolescents and teenagers, common sleep problems include sleep disordered breathing, insomnia, and hypersomnolence. They may also experience other sleep disorders such as restless leg syndrome and parasomnias such as sleepwalking. Sleep problems occur very frequently in this age group. His important that all adolescents and teenagers be screened for sleep problems including questions regarding nighttime sleep, daytime sleepiness, and snoring.

How to get a good nights sleep?

According to leading sleep researchers, there are techniques that may be implemented in order to decrease the common sleep problems such as keep a regular sleep-wake schedule that allows for a developmentally appropriate amount of sleep; avoid caffeine beverages four to six hours before bed and minimize daytime use; avoid alcohol and heavy meals before sleep; practice regular exercise; minimize noise, light and excessive hot or cold temperatures during sleep; establish regular bed time and go to bed at the same time each night; early morning bright light exposure may help to promote an earlier bedtime/sleep time.

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The Neuropsychology Of Multiple Sclerosis

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Introduction:

Multiple Sclerosis (MS) is the most common cause of neurological disability in the young and middle-aged. Most patients with MS experience a decline in cognitive abilities, even in the early stages of the disease when there is no or minimal physical symptoms and/or disability. Neuropsychological investigations have suggested a prevalence rate of 50% to as much as 60% of cognitive problems in MS.

Cognitive dysfunction in MS is difficult to identify on routine neurological examination because language skills and intellectual function are usually well preserved. Understanding the neuropsychology of MS facilitates the management of the disease for the neurologist as well as the patient. The following are three cases that illustrate the most common clinical neuropsychological presentation in MS.

Case 1:

A 57-year-old left handed white male with a college degree in business and CFO of an investment company presents with a history of MS. He was first diagnosed with relapsing-remitting MS since 1996. Initially treated with Avonex and for a number of years he had been on Tysabri infusion. He had remained very stable with no significant physical symptoms except fatigue. His initial MRI demonstrated a few white matter lesions. His medical history includes hbp and high cholesterol managed with medication. His neurologist first referred him for neuropsychological evaluation in 2006. At that time his main concerns were mild cognitive problems, primarily with memory and processing information. The results of that assessment identified very mild cognitive difficulties and significant depression that was felt to exacerbate his cognitive problems. A brief course of cognitive behavioral therapy was done and improved his depression with some reduction in his cognitive concerns. He was referred again for neuropsychological evaluation on January 2015. His most recent MRI demonstrated no active lesions but now a few more lesions as well as some spinal cord lesions. The burden of the disease had increased. His concerns now included increased cognitive problems that were impacting his ability to perform his job. He was increasingly making minor mistakes, not meeting deadlines because of working slower and was concerned about his verbal communication skills with increased word finding problems. The results indicated a mild decline in cognition since 2006 with increased problems with concentration, memory, word fluency and the most significant decline was in speed of processing information. His depression is now moderate and in need of treatment. Because of the nature of his work, he can't afford making mistakes and he is now applying for long-term disability. This is a contributing factor to his depression.

Case 2:

Her neurologist refers a 67-year-old right-handed white female who is now retired from working in graphics marketing and has a college degree for neuropsychological evaluation. She is concerned about increasing cognitive problem related primarily to word finding difficulties. No history of dementia in the family. In 2014 she had an MRI of the brain and cervical spine. The findings included confluent areas of white matter disease around the ventricles particularly around the posterior horns. Subcortical white matter disease was also noted with changes noted in the pons. MRI of the cervical spine showed increased signal in the cord of about 1cm around C4 slightly off to the right. The neurologist noted her presentation as "complicated and involved" and suspected a demyelinating disease such as MS. No disease modifying treatment was initiated. The patient reports a history of right-sided weakness beginning about 15 years previously that has progressed slowly and mildly. She now has a right foot drop. Fatigue has been a problem for years and she has noticed increased sensitivity to heat. She believes her memory in general remains good. There is a history of chronic depression treated with psychotropic medication by a psychiatrist. Reports that her depression has been quite stable over the last few years. No other significant health issues noted. The neuropsychological findings were normal and consistent with her lifelong abilities with no decreased word fluency. No cognitive impairment was noted. Her depression was found to be well under control. Patient was reassured on follow up.

Case 3:

His neurologist for neuropsychological evaluation on November 2012 initially referred a 44-year-old, left-handed, white male with a one-year college education. He was first diagnosed with Relapsing-Remitting MS in 2007. Initially he developed some problems with his balance. He was started On Gilenya, Provigil, clonazepam, citalopram, and Vyvanse. At the time he worked as project manager for a cellular phone company. He was concerned about working slower and his employer was accommodating him. Fatigue was a major concern at the time. History of episodes of optic neuritis as well as having joint and muscle pain. He acknowledged some depression with no prior history. The results of the 2012 evaluation demonstrated some reduction in processing speed of information and mild attention problems. There was a significant depression in need of more aggressive treatment Results were discussed with him at the time. The pattern of neurocognitive testing suggested a frontal-subcortical pattern of mild deficits. His neurologist treated his depression with Lexapro. Referred again for re-assessment on February 2015. Now concerns of increasing word finding problems and memory problems that has been impacting his work efficiency. He has been making mistakes, slower in getting his work done increasing his stress. He perceives his employment in jeopardy. He continued on Gilenya and Provigil. MRI has

remained stable. He had been experiencing increased pain and had increased spasticity. He was started on baclofen. A pain management physician started him on morphine. The results of the second evaluation demonstrated increased problems with processing information; attention problems were noted impacting his memory efficiency. In spite of his accommodations at work performing his job had become increasingly more stressful. He is applying for long-term disability.

LEARNING OBJECTIVES:

1. Prevalence of cognitive impairment in MS.
2. Evaluating cognitive impairment in MS.
3. The nature of the cognitive deficits in MS.
4. Correlations between cognitive impairment and brain pathology.
5. Disorders of mood and behavior in MS.
6. Management of cognitive and psychosocial disorders.
7. The role of the neuropsychologist in MS management.

Prevalence and course of cognitive impairment in MS:

Neuropsychological studies of community-based MS patients found 43% with cognitive deficits. This figure rises to 60% in the sample from specialized clinics (1). Cognitive impairment (CI) in MS tends to be subtle and difficult to detect. CI is a major cause of disability in many MS patients with minimal or no physical disability. The form or degree of CI does not correlate with the disease course. Longitudinal studies have documented an increase of CI from 26% to 56% over a ten-year interval (2). CI can present early in the disease course. Individual variation in test performance is noted due in part to the heterogeneous distribution of MS lesions in the CNS.

Evaluating cognitive impairment in MS:

The Minimal Assessment of Cognitive Function in MS is a recommended neuropsychological test battery (3). The 90-minute battery includes:

1. Controlled Oral Word Association Test.
2. Judgment of Line Orientation Test.
3. California Verbal Learning Test.
4. Brief Visuospatial Memory Test.
5. Paced Auditory Serial Addition Test.
6. Symbol Digit Modalities Test.
7. Sorting Test from the Delis-Kaplan Executive Function System.

This battery assesses cognitive domains impacted by MS. These include language, spatial processing, new learning and memory, processing speed of information and working memory as well as executive functions. In complex cases as well as those with concerns about applying for disability intellectual, personality as well as validity testing should be administered.

The nature of Cognitive deficits in MS:

Multiple domains of memory are impacted with MS. Deficits in working, semantic, and episodic memory has been replicated. MS patients also have difficulties in acquiring and retrieving new information. Procedural memory is unaffected. A significant problem is decreased attention and decreased processing of information. Decreased word fluency as well as executive dysfunction are common. Problem solving difficulties are common as well as problems with planning and sequencing tasks. They have difficulties with organizational skills and follow through. Considerable individual variation exists in performance on neuropsychological testing in MS due to the widespread and idiosyncratic nature of the lesions present in a given individual.

Correlations between cognitive impairment and brain pathology:

MRI measures have been correlated with CI in MS. These include whole brain atrophy (4,5), cortical volume (6), lesion load (7), and diffusion anisotropy (8). Total lesion area is predictive of dysfunction in memory, abstract and conceptual reasoning, language and visuospatial skills. Another study found a correlation with lesion burden in frontal and non-frontal regions and attention, memory, planning, problem solving, and conceptual reasoning (9). Volumes of white matter (10) and T2 hyperintense lesions in deep gray matter nuclei (11) also correlated with overall cognitive functioning. T1 and T2 lesion volumes are significantly higher in patients with CI than those without CI. Nelson (12) has demonstrated that cortical lesions play an important role in CI. However, the results suggested that lesions that remain contained within the cortical ribbon do not play a more important role than ones extending into the adjacent white matter and furthermore, the size of the cortical lesion, and not the tissue-specific location, may better explain their correlation with CI. Benedict (6) has shown that frontal cortex atrophy predicts CI in MS. Benedict (13) has also shown that the main predictors of cognitive changes over 7 years are baseline diffuse brain damage and progressive central brain atrophy over the 2 years after MS diagnosis. Benedict (14) has also demonstrated a correlation between third ventricle with and CI in MS. Benedict (15) has evaluated the role of cortical atrophy and personality changes in MS. Preliminary findings suggest that cortical atrophy in MS is associated with adverse impact on personality in MS.

Disorders of Mood and Behavior:

The lifetime prevalence of major depression in MS is about 50%. This is higher than in any other neurological disorder. The basic phenomenology of MS depression overlaps with that found in primary depression. Suicide rates in MS are up to 7 times higher than rates in the general population. The lifetime prevalence of bipolar affective disorder is twice the prevalence in the general population.

Common emotional reactions to MS diagnosis include:

- Fear and apprehension.
- Denial
- Anger
- Grieving
- Depression
- Guilt.

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Highlighting Unique Features Of The Pediatric Concussion

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Brief overview of Concussion (or mild Traumatic Brain Injury)

According to the Centers for Disease Control, there are at least 1.7 million traumatic brain injuries (TBIs) in the United States each year, the majority of which are mild injuries.[1] The Texas Traumatic Brain Injury Council Report (2014) estimates that 146,000 Texans sustain a TBI every year, a majority of which are mild.[2] With over half of the TBIs each year occurring in individuals between the ages of 0 and 24 years (especially 0 to 4 and 15-19 years of age),1 brain injury is a prominent pediatric concern.

Concussion is a *mild TBI*, defined in the most recent Consensus Statement on Concussion in Sport as a "complex pathophysiological process affecting the brain, induced by biomechanical forces" leading to "rapid onset of short-lived impairment of neurological function that resolves spontaneously; Concussion results in a "graded set of clinical symptoms" that resolve over time.[3] The concussive injury produces a neurometabolic cascade resulting in ionic imbalances, glutamate release, energy crisis, oxidative stress, inflammation, cytoskeletal damage, altered neurotransmission, and axonal injury, all of which contribute to the diffuse and variable clinical signs and symptoms of concussion.[4] Cerebral blood flow has also been shown to be reduced following sports-related concussion in children.[5] While there is no current evidence of significant variability in the acute physiological changes of concussion by age at the time of injury, *the potential disruption to the ongoing underlying processes of neurodevelopment, such as synaptic pruning and myelination, are unique to the pediatric concussion.*

What are the main causes of concussions in children?

Case: An 8 y/o girl falls off of monkey bars and hits her head on the ground, having brief loss of consciousness. She was taken to the ER where her head CT was normal. At clinic follow-up 2 weeks later, parents report she had headache, irritability, and increased sleep for about 1 week but now is back to baseline with no deficits and normal neurological exam.

While most literature, including the current American Academy of Neurology (AAN) concussion guideline, focuses specifically on sports-related concussion (SRC), *it is important to recognize SRC is not the most common etiology of concussion in younger children.* In children 0-4 years of age, falls (ex. from playground equipment) are the primary mechanism of injury, accounting for approximately 70% of emergency room visits for TBI.1 In a study examining the etiology of concussion children 8 -19 years of age presenting to the emergency department, only 38% of injuries occurred during organized team sports and almost two-thirds of concussion in children 8-13 years of age resulted from individual or leisure activities such as riding a bicycle.[6] Unfortunately, no studies have yet evaluated different mechanisms of injury and effects on pathophysiological changes or outcome following injury.

As SRC are more common in older children, these injuries are more likely to be seen by adult neurologists. The epidemiology and rate of SRC are similar in high school and collegiate athletes, with football and soccer posing the highest risk of concussion, greatest during a game rather than practice.[7] While males have the highest overall rate of concussion, females actually have a higher risk of concussion in gender-comparable sports.7

What are the symptoms of concussion in children?

Case: A 4 year old male climbs up on the kitchen table then falls off, hitting the back of his head. Mom reports that he is still playful, running around the house, but is acting clingy and whining more, has become a picky eater, and had nighttime enuresis 3 nights of the past 5 nights since his injury.

Given the diffuse metabolic changes, the symptoms of concussion are not-specific but classically evaluated within 4 domains: physical, cognitive, mood, and sleep. While there are multiple graded post-concussive symptom scales, there is little evidence of the validity of these measures specifically in the pediatric population.[8] Younger children are often unable to express specific symptoms, therefore evaluation of concussion relies on parent observed behavioral changes (ex. more clingy, changes in play or eating habits). Additionally, younger children may present with atypical somatic symptoms, such as increased nighttime enuresis, belly pain, and nightmares, not included in many concussion symptom scales. Using a modified Rivermead concussion checklist, parents of preschool children (age 2 – 5 years) reported fewer concussive symptoms than parents of older children (6-12 years) following concussion.[9] *When evaluating pediatric concussion, it is essential to explore behavioral changes and evaluate for signs and symptoms beyond those defined in post-concussive symptom scales.*

What is the expected course of recovery?

Case: A 12 year old football player was involved in helmet to helmet contact without loss of consciousness. He presents to neurology clinic 1 month later and is still complaining of daily headache, fatigue, and difficulty with concentration.

While young adults typically recover within 10 days from SRC, *children sustaining concussion have a longer course of recovery, with symptom resolution occurring in most children within 3 months.*[10, 11] Mechanical factors (weaker neck muscles and larger head to body ratio) and/or neurobiological factors (varying stages of brain development) may contribute to the prolonged post-concussive symptoms in children compared to adults.4 Additionally, several studies indicate that compared to collegiate athletes, high school athletes have longer duration

of symptoms, more severe cognitive deficits, and more difficulty with balancing following SRC.[12, 13] As there are no objective measures of recovery from concussion, relying on patient and parent symptom report, it is important to recognize and clinically evaluate other factors potentially contributing to prolonged symptoms, including underlying premorbid conditions as well as child and parent coping strategies.

WHAT IS THE MANAGEMENT OF CONCUSSION IN CHILDREN?

Case: A 14 year old female with a history of concussion sustained 3 months ago presents for evaluation of her second concussion sustained in gymnastics. She is a straight A student in only honors and AP classes and a competitive gymnast. Her pediatrician recommended that she rest (no texting, TV, computer, reading, or any form of physical exertion) and not to return to school or gymnastics until all of her symptoms resolved. She has been home resting for the past 2 months due to persistent daily headache. She presents to Neurology Clinic as her symptoms are now worsening, with increased headache and fatigue, new irritability and moodiness, and worsening sleep.

Removal from play: In the immediate period following concussion, *children are uniquely potentially susceptible to catastrophic cerebral swelling following brain injury* with either a single hit or successive impacts (second impact syndrome (SIS)). The diagnosis of SIS is controversial as the incidence overall is low and evidence limited to case reports and series; however, current literature indicates that children and adolescents are uniquely at risk for massive potentially fatal cerebral swelling in the acute period following concussion.15 More specific predictors, such as underlying metabolic or genetic factors, identifying which specific child is at risk for SIS are unknown therefore clinical caution is imperative in the immediate period following a pediatric concussion. Any concern for concussion in a child should prompt immediate removal from play and evaluation by a medical provider with expertise in brain injury before a child is returned to sports.

Beyond the immediate removal from play, the cornerstones of concussion management include physical and cognitive rest. Based on the neurologic energy crisis following TBI,4 limiting physical and cognitive activity theoretically lessens neurometabolic demands following concussion. Initial studies of animal models of TBI suggested that voluntary exercise in the acute period following brain injury lead to worse outcomes; however, subsequent studies explored the timing of exercise following injury and indicate that exercise in the subacute period promotes recovery and improves outcome in animal models of mild TBI.14 To date there are no basic science studies evaluating potential effects of cognitive exertion on recovery. Despite limited evidence, current guidelines recommend both physical and cognitive rest following concussion and symptoms resolution prior to return to sport.3

Physical rest: Practice recommendations with the AAN guidelines not a child should not return to sports until cleared by a provider,16 noting that a more conservative management approach should be considered in younger athletes. However, specifics on the level of physical activity during the period of recovery is not defined and varies by practitioner. Children are typically very active and unmonitored, rigid restriction of physical activity (such as a child forced to sit in the library during recess or a competitive athlete limited to walking associated with only daily activities) is often clinically seen to be associated with persistent or even increasing symptoms. In addition to physiological changes associated with inactivity, psychological consequences of prolonged activity restriction are related to worsened outcomes following concussion17. In adolescents with persistent concussive symptoms (> 4 weeks following injury), active exercise rehabilitation has been shown to reduce post-concussive symptoms and improve outcome.18 Further evidence is needed to better address the precise timing and level of exercise in the subacute phase of recovery and individual variability is expected; noting the potential negative effects of both early overactivity as well as prolonged inactivity, it is important to monitor the level of physical exertion and encourage progression of daily, safe activity as tolerated. Once a child is symptom-free (or based on clinical judgment of concussion resolution for complex or prolonged recoveries), the "gradual return to play" (6 steps progressing from limited activity to return to contact sports / game play3), is recommended; however, providers of children with concussion often modify the steps (such as recommended 2-3 days at each "level" of activity) to allow additional time for assessment of a child's tolerance of increased exertion. Furthermore, it is common practice to assure that a child is able to attend and tolerate school full-time without new accommodations prior to returning to sports, as education is paramount.

Cognitive rest: As children are primarily in an educational setting, undergoing active cognitive stimulation, the effects of cognitive exertion and timing of returning to school is also critical to the management of the pediatric concussion patient (Figure 1). Similar to physical rest, there is little evidence supporting or defining "cognitive rest." In a cohort of children and young adults with SRC, prolonged symptom duration was only associated with the highest level of reported daily cognitive activity whereas the clinical course of recovery was similar among all other estimated levels of cognitive exertion.19 A recent randomized controlled trial compared the course of recovery in children presenting to the ED with concussion receiving recommendations for either strict rest (no cognitive or physical exertion) for 5 following days following injury or unusual care (typically 1-2 days of rest with gradual return to school and physical activity). This study found that children with concussion undergoing strict rest reported more daily symptoms and had a longer time to symptom resolution.20 Children are unlikely to be entirely symptom-free before returning to school; therefore, it is critical to provide individualized accommodations to best support the child's optimal function. For providers unfamiliar with educational considerations and accommodations, the American Academy of Pediatrics clinical report "Returning to Learning" and Center for Disease Control Acute Concussion Evaluation Care Plan both provide additional information to guide practitioners managing children with concussion.21, 22 The balance of rest and activity, both physical and cognitive, is individualized and requires flexibility to adjust recommendations based on each child's tolerance of increasing levels of exertion as well as consideration of other factors potentially affecting recovery.

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Neuropsychology...

Emotional Stress of MS include:

- The unpredictability factor.
- The invisible symptom factor.
- Making decisions about treatment.
- Life issues including employment and disability.
- Changes in sexuality
- Changes in family life
- Personality and behavior changes associated with brain changes.

Management of cognitive and psychosocial disorders:

There is no accepted medical treatment for the management of cognitive disorders in MS. Stimulants has been used with no consistent benefit. Side effects can be significant. The best approach includes lifestyle changes and behavioral adaptation including cognitive rehabilitation as well as developing compensatory strategies to manage cognitive problems. Stuifbergen (16) at UT-Austin has found that a computer assisted cognitive rehabilitation program holds promise for enhancing cognitive functions in MS. Currently at UT research is being done to explore systematic ways in which cognition can be improved in MS by brain exercises and developing compensatory behavioral and cognitive strategies. Developing and increasing cognitive reserve is an objective of the current research in MS. Depression and other psychiatric problems are managed using conventional psychiatric approaches. These include appropriate medication management and cognitive behavioral therapy. Developing self-efficacy has been found to improve the quality of life in persons with MS. Group psycho-educational programs can facilitate improvements in quality of life. Successful living with MS includes staying involved, appraising MS with realism and flexibility, maintaining strong bonds with family and friends and keeping a sense of purpose.

The role of the neuropsychologist in MS management:

Neurologist working with MS patients should be aware of the many psychosocial issues associated with MS so appropriate assessment and interventions can be facilitated. The neuropsychologist plays a very important role in the multidisciplinary management of MS. The objectives of good health care management are to empower the patient by developing self-efficacy and facilitate improvement in the quality of life. Patients who are confident in their ability to communicate with their health care team tend to do better and manage their symptoms and life more efficiently. In addition to objectively identify cognitive problems that a patient may have, the neuropsychologist can provide guidance and counseling to the patient and family. In my experience one of the major concerns facing MS patients is developing cognitive impairment. This can impact work and decision-making. Neuropsychologists have unique set of skills to help patients deal with these issues.

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Mary Vanderlick, MD, Program Director

Join us in San Antonio Texas at the La Cantera resort July 15 and 16, 2016 for the TNS Summer Conference.

Topics include neuromuscular disorders, paraneoplastic and autoimmune encephalopathies and neuro-otology. Practical use of genetic technologies in neurologic testing plus the future of solo or group practice in the new health care environment will be interesting topics related to the not too distant future.

Speakers are experts in their fields from Texas and the United States.

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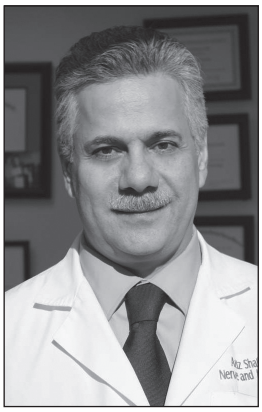

Texas Neurological Society has grown to more than 700 members, which attests to the quality of its leadership. It is the largest and most active state neurological society in the USA. However, we still strive to capture the remaining 700 neurologists in Texas who are missing a whole lot of fun and learning.

Here is what They are missing:

- High quality educational programs at reduced registration. We have two CME meetings per year: our Annual Winter Conference is held each February in Austin; our Summer Conference is held each July in Central Texas.
- TNS has a strong relationship with the American Academy of Neurology, tracking federal and state issues. Advocacy at the State Level, which is conducted through a part time lobbyist, has impacted the relevant state laws. The chiropractors have not been allowed to do EMG/NCV, dentists have not been allowed to do sleep studies, and more.
- Through our partnership with Texas Medical Association, we have been watching for regulations and legislation that affect neurology.
- Our newsletter Broca's Area is published 2 times a year, and includes legislative, socioeconomic, and society news. And our web page, www.texasneurologist.org, includes meeting information and news of interest, as well as a complete listing of TNS members and their office phone numbers. Your practice website can be linked to the TNS website.

I would like to invite you to renew or join our membership. Please complete and submit the membership application on the TNS website. Dues are only $75 per year.

If you have a question or want to discuss TNS issues, please do not hesitate to call me 24/7 at (713)795-0033.

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Highlights...

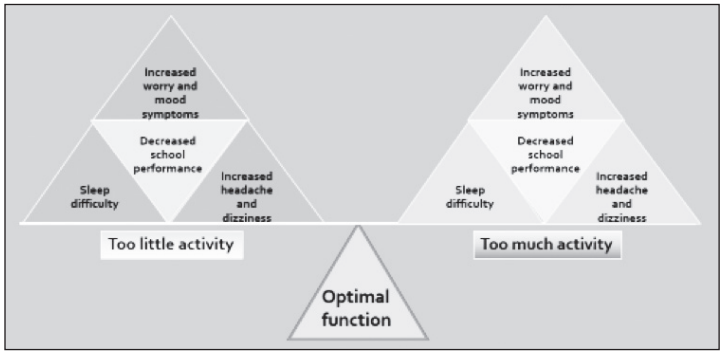

Figure 1: The Balance of Cognitive Exertion Level in Children with Concussion

Conclusion:
While concussion in children is in many ways similar to concussion in adults, there are important differences to recognize when evaluating the pediatric concussion patient. The unique features of pediatric concussions combined with the lack of objective measures to measure concussion contribute to the necessity for an individualized approach in the management of this population.

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An Atypical Presentation of Spontaneous Intracranial Hypotension

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Introduction

Spontaneous intracranial hypotension (SIH) is a postural headache syndrome without preceding major head trauma or dural laceration, associated with low cerebrospinal fluids (CSF) volumes and opening pressures.5,2 The headache is typically provoked by upright position, and relieved by lying flat. 2 This case demonstrates an atypical presentation of spontaneous intracranial hypotension and the dilemma of treatment.

Case Report

A 56 year old woman with past medical history of mechanical mitral valve on warfarin presented to the neurology clinic with a 10 month history of headaches. She described her headaches as pressure like, usually starting bifrontally and radiating to temporo occipital regions. Her headaches were worse with activity and associated with nausea and photophobia. She denied any noticeable positional component to her headaches. She had no relief with various over the counter medications and had minimal relief with sumatriptan, prescribed by her primary care physician.

After being seen in the neurology clinic for her daily headaches, the patient was prescribed topiramate, which was moderately helpful in controlling her pain. An MRI Brain was ordered to further evaluate this new-onset headache. The MRI Brain revealed deformity of the midbrain compatible with brainstem sagging (Figure 3), very small bilateral frontal parietal subdural collections (Figure 2), and extensive supratentorial and infratentorial dural enhancement bilaterally (Figure 1). All these findings were compatible with intracranial hypotension.

The patient denied any prior history of lumbar puncture or trauma. As she had minimal relief with topiramate and caffeine, the option of epidural blood patch was discussed with the patient. Her medical history, however, made this treatment option more difficult. She had been on warfarin for a number of years for her mechanical mitral valve and had history of complications with bridging with enoxaparin for procedures in the past.

After discussion with her cardiologist and referral to a hematologist, the patient elected to proceed with hospital admission for an epidural blood patch. Per the hematologist's directions, the patient was taken off of warfarin and started on heparin. Once her coagulation studies had normalized, the patient underwent an epidural blood patch with 12 ml of blood. She tolerated the procedure well without complications. She denied a headache after the procedure but unfortunately has continued to have a mild headache since.

Discussion

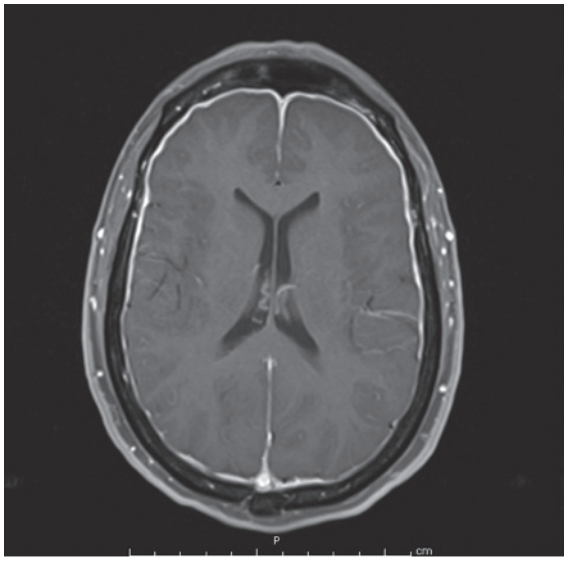
SIH is characterized by the presence or worsening of preexisting headaches after a change from supine to standing position and improvement or resolution of the pain after acquiring the recumbent position.4 The syndrome develops because of CSF leakage, usually in the absence of any easily identifiable cause.5 Although an orthostatic headache is the typical presentation, other headache patterns may occur and some patients may not have a postural component to their headache.1 Patients may also present with posterior neck pain or stiffness, nausea, vomiting, dizziness, tinnitus, hyperacusis, deafness, diplopia, nystagmus, ataxia, and visual loss.1,4,5

The most common finding on MRI of the brain is diffuse pachymeningeal gadolinium enhancement.1 Other MRI abnormalities include subdural fluid collections, downward displacement of the cranial contents, engorgement of the venous structures, and pituitary hyperemia.1,4
Initial management of SIH includes analgesics, strict bed rest for several days, and liberal fluid and caffeine intake. If conservative measures fail in alleviating the pain within one to two weeks, a non-directed lumbar epidural blood patch (EBP) is recommended.1 EBP is done by infusing 10–20 ml of autologous blood into the epidural space. Although the exact mechanism of EPB is not known, the proposed mechanisms of action are related to the volume replacement by compression of the dura and to sealing of the dural defect.5,3

The efficacy of each blood patch is thought to be about 30% and many patients may need more than one treatment.5 For patients with clinically typical SIH, with or without brain MRI confirmation of the diagnosis, Amoozegar, et al. recommends up to three non-directed blood patches at least five days apart before proceeding with further tests to localize the leak.1

In this particular patient, an MRI brain was a valuable tool in the diagnosis of SIH, given her atypical presentation. This case was complicated by the patient's cardiac problems and prior response to enoxaparin. The question arises as to whether treatment with EBP would have been indicated in this patient, given her complicated history and potential need for future EBPs. Tests to localize the leak may have been beneficial in preventing further EBPs, however, these scans are not often rewarding and would have presented additional risks to her. In an attempt to provide relief and as conservative measures had failed in alleviating this patient's symptoms, I believe the right measures were taken in the management of this patient's SIH. She will be monitored during follow up visits to assess for clinical improvement.

Special thanks to Dr. Jack Fraim for his input to this article.



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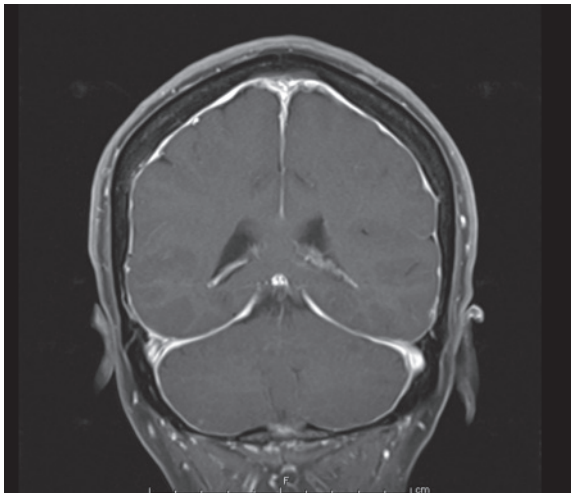

B

Figure 1: MRI T1 weighted post contrast image demonstrating diffuse dural enhancement. a) axial image, b) coronal image.

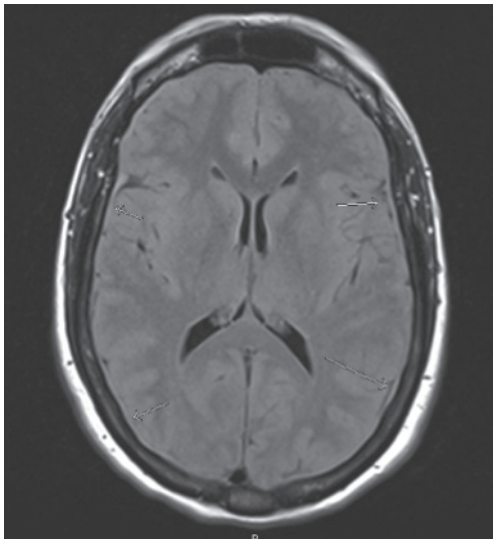

Figure 2: MRI FLAIR axial image showing bilateral frontal subdural collections (arrows).

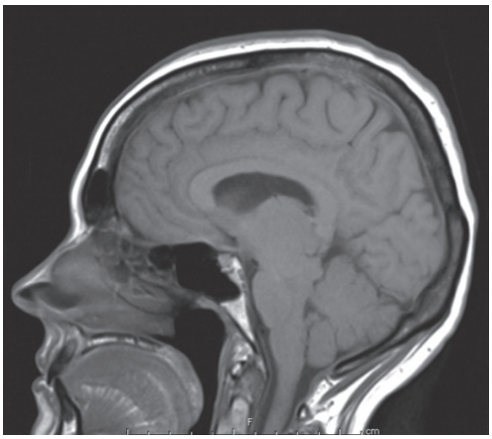

Figure 3: MRI T1 weighted sagittal image showing downward descent of brainstem.

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