TEXAS NEUROLOGICAL SOCIETY SUMMER 2017

Broca's Area

The Voice of Texas Neurology

President's Message

Erin Furr-Stimming, MD

Dear Colleagues

I am honored to be your TNS president this year. This has been an interesting year politically and will likely continue to present new challenges for us as physicians. A recently published study in Neurology summarized the data from a national survey that queried over 4000 American Academy of Neurology members. The authors found that burnout is common in all neurology practice settings due to multiple contributing factors. Importantly, however, the best predictor of career satisfaction is the meaning neurologists find in their work. We must remind ourselves of the impact we make in evaluating and treating our patients and remain focused on the true significance of our work.

TNS continues to grow, and in fact, during a recent meeting with the previous AAN President, Terry Casino, MD, we were referenced as the "gold standard" for state neurologic societies. We should be very proud of the TNS, the largest state neurological society in the country with approximately 800 members to date.

"We must continue to remind ourselves of the impact we make in evaluating and treating our patients."

We advocate for our profession with the assistance of Greg Herzog, our lobbyist, who keeps us well informed about pertinent pending bills and important legislative issues. We are nearing the end of the 140-day 85th Texas Legislative Session and healthcare related issues have continued to remain a focal point. TNS' legislative issues have been step therapy/fail first reform, MOC and out-of-network balanced billing. Dr. Sara Austin, TNS Legislative Chair and Greg, have included an in-depth article about these and other issues later in this addition of Brocas. Be sure to check it out and remember, you can track these bills and others online at http://www.capitol.state.tx.us

In addition to advocacy, TNS takes pride in educating the membership both about pertinent neurologic topics and critical economic and regulatory issues. Dr. Stuart Black, TNS Medical Economics Chair, and Kristi Berrier, TNS Medical Economics Advisor, have made significant efforts in conjunction with the Economics Committee to ensure we are familiar with relevant acronyms such as MIPS, MACRA and PQRS to name a few.

TNS has made advances in educating our members during our excellent biannual conferences and increased resident participation remains a priority. Several years ago, we implemented the resident poster sessions, which have been quite successful. This winter, with Dr. Michael Soileau leading the charge as the Program Director, we will invite the residents to present a relevant case prior to each lecture. Thanks to the generous support of our members, we remain financially strong and continue to host the residents free of cost.

Finally, I would like to thank our board members and their dedication to TNS. Most importantly, I would like to acknowledge and thank Ky Camero, the real engine behind TNS. Ky is an amazingly organized, professional and personable colleague and we thoroughly appreciate her dedication to the TNS.

Thank you for your support. We will continue to fight for our profession. I look forward to seeing you in July at the luxurious Hyatt Lost Pines hotel in Bastrop, Texas. Dr. Jetter has planned a great summer conference!

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 14th Annual Summer Conference at Lost Pines, July 28-29. Gina Jetter, program director, Bob Fayle, education committee chair, and the education committee have planned an excellent program.

ST. VITUS

When travelling, I often query whether there were any significant contributors to neurology in that location. I was recently in Europe and made a few stops where there is an abundance of contributors to choose from.

In Prague, the Gothic masterpiece St. Vitus Cathedral is a dominating and impressive site (figure 1). Construction began in 1344.

FIGURE 1. ST. VITUS CATHEDRAL

St. Vitus, of course, is a well-known name in neurology. According to legend, the child Vitus (290-303) from Sicily, son of a Roman senator, drove out a demon (epilepsy?) in a son of the Emperor Diocletian. When he would not sacrifice to the pagan gods in celebration, he was pronounced a sorcerer and put to death. (Epilepsy action. https://www.epilepsy.org.uk/about/st-valentine-epilepsy-patron-saints). St. Vitus is the patron saint of actors, comedians, dancers, and epileptics.

In medieval Europe, there was an epidemic of dancing mania, an unusual choreiform disorder. Most probably had mass hysteria although others may have had epilepsy, ergot poisoning, torsion dystonia, and rarely Syndenham's (Jummani RR, Okun MS. Sydenham chorea. Arch Neurol 2001:58; 311-313).

The Swiss German, Paracelsus (born 1493, died 1541 in Salzburg) was a physician, philosopher, botanist, astrologist, occultist, and founder of toxicology. He gave zinc its name (zincum) and was the first to note that some diseases are due to psychological disorders. In describing the dancer mania, he coined the term "chorea sancti viti" or St. Vitus dance.

In the chapter, "On Saint Vitus Dance" in his last book in 1686, Thomas Sydenham (1624-1689; figure 2), the British Hippocrates and father of chorea (from Latin "choreus" or dance) wrote the following: "This is a kind of convulsion, which attacks boys and girls from the tenth year to the time of puberty. It first shows itself by limping or unsteadiness in one of the legs, which the patient drags. The hand cannot be steady for a moment. It passes from one position to another by a convulsive movement, however much the patient may strive to the contrary. Before he can raise a cup to his lips, he makes as many gesticulations as a mountebank; since he does not move it in a straight line, but has his hand drawn aside by spasms, until by some good fortune he brings it at last to his mouth. He then gulps it off at once, so suddenly and so greedily as to look as if he were trying to amuse the lookers-on."

FIGURE 2. THOMAS SYNDENHAM

Sydenham attributed the cause of chorea as "some humor falling on the nerves, and such irritation causes the spasm." Bouteille, in 1810, reported patients where rheumatism preceded or followed chorea. In 1899, Wesphal, Wasserman, and Malkoff found diplococcus in the pericardial and cerebrospinal fluids of a child who died of rheumatic pericarditis and chorea. In 1956, Taranta and Stollerman established the causal connection between group A beta-hemolytic streptococci and Sydenham chorea.

DOPPLER

While walking through Salzburg, I came upon the house where Christian Doppler (1803-1853) was born and next door to where Mozart's family had lived. In 1842, while on the faculty of the Prague Polytechnic, Doppler published "On the coloured light of the binary stars and some other stars of heaven" where he postulated what would later be known as the Doppler effect.

Fig. 1

Fig. 2

In 1941, the Austrian neurologist, Karl Dussik, collaborating with his physicist

brother, Friedreich, were the first to ultrasonically image human intracranial ventricles. Starting in the late 1960s, Strandness and colleagues at the University of Washington conducted research on Doppler ultrasound to diagnose vascular disease and later developed technologies to use duplex imaging in conjunction with B-mode scanning to view vascular structures in real-time (Zierler, R. Eugene (2002). "D. Eugene Strandness, Jr, MD, 1928–2002". Journal of Ultrasound. 21 (11): 1323–1325).

FREUD

Sigmund Freud lived at Berggasse 19 in Vienna for 47 years until he was forced to flee the Nazis and moved to London. The building has been his museum since 1971.

Freud, of course, was a neurologist first publishing on basic neuroscience research, and books on aphasia and paralysis in children (Galbis-Reig D. Sigmund Freud, MD: forgotten contributions to neurology, neuropathology, and anesthesia. The internet journal of neurology 2003; 3(1)).

Researching the action of cocaine, he started using it himself for experimentation in 1884 which may have led to abuse which stopped in 1896 (Markel H. Sigmund Freud, William Halsted, and the Miracle Drug Cocaine. Pantheon, 2012). He sent a letter to his fiancé and promised that she would be unable to resist the advances of "a big, wild man who has cocaine in his body."

On a related topic, in the opening scene of the second novel featuring Sherlock Holmes published by Conan Doyle in 1890, "The Sign of the Four," Holmes describes the cocaine which he was injecting himself as "a seven-per-cent solution." Holmes cocaine addiction was the basis for the fantastical 1974 novel by Meyer and the 1976 movie, "The Seven-Per-Cent Solution," where Watson takes Holmes to Vienna for Freud to cure the addiction with hypnosis. (Fun film starring Robert Duvall as Watson, Alan Arkin as Freud, Laurence Olivier as Moriarty, and Vanessa Redgrave as Lola Devereaux.)

He had a 6 month travel grant to study with Charcot in Paris in 1885-1886. Charcot's lectures and demonstrations on hysteria changed Freud's life and psychiatry.
In 1895, Roth in Moscow coined the term "meralgia paresthetica," which was also described by Bernhardt in Berlin in 1878.

In 1895, Freud also provided a description: "…For years, I have been familiar with that little malady, having already observed it in five to seven cases. …Aged 39, and for the last 7 years at least, I have been feeling Bernhardt's sensory disturbance on the outer surface of my right thigh. No etiologic derivation has ever been obvious to me; among the etiologic factors adduced by Bernhardt (enteric typhoid, lead poisoning, use of cold showers) only the last mentioned applies to me, but I would not insist on its significance. …I have gained the impression that the affection has a rather high incidence. … Dr Josef Breuer, who I consulted some time ago as an expert regarding my own paresthesiae, was likewise aware of the symptom and drew my attention to the superficial course of the external femoral cutaneous nerve (closely adjacent to the anterior superior spine of the iliac bone, lying between two strands of the external inguinal ligament). He suggested a possibly damaging effect on the nerve due to the pressure exerted by tight clothes around the waist." (Freud S. Ueber die Bernhardt'sche Sensibilitatsstorung am Oberschenkel. Neurolog Centralbl 1895;14:491-2; Schiller F. Sigmund Freud's meralgia paresthetica. Neurology. 1985;35(4):557-8).

JENDRASSIK

Ernö Jendrassik was born in Transylvania then spent the remainder of his life in Budapest starting with medical school (Pásztor E. Ernö Jendrassik (1858-1921). J Neurol. 2004 Mar;251(3):366-7). The Jendrassik maneuver, described in 1885, is still widely used by neurologists. He observed that the deep tendon reflex is increased if other muscles are contracting at the same time. He had the patient perform a muscular activity such as squeezing a hand or pulling clasped fingers. The maneuver can also be performed by making one or two fists or pressing a button with the thumb (Stam J. Jendrassik's maneuver. In Koehler PJ, Gruyn GW, Pearce JMS. Neurological Eponyms. Oxford, New York, 2000, pp 143-147). If the patient focuses their attention on the one leg, the knee jerk in that leg becomes more active.

Welcome New Members!

(02/05/2016- 02/23/2017)

Farida Abid, MD
Irfan Ali, MD
Shannon Dicarlo, MD
Simon Keyyal, MD
Anuranjita Nayak, MD
Monica Proud, MD
Sarah Risen, MD
Rebecca Schultz, PhD, RN, CPNP
Padma Kumar
David Larson, PNP
Yu-Tze Ng, MD
Samiya Ahmad, MD
Gary Bobele, MD
Yaman Eksioglu, MD, PhD
Melissa Svoboda, MD
Guojun Zhang, MD
Vickie Farmer, BSN, PA-C
Subhashie WIjemanne-Sarathkumara, AP
Amy Hicks, FNP-C
Rony Ninan, MD
Vasishta Patel, MD
Walter Wechan, MD
D. Michael Chachere, MD
Jerome Lisk, MD
Nirav Shah
Nancy Opperman, FNP
Jordan Harborth, FNP-C
Sabina Miranda, DO, MPH
Ariana Minton
Ryan Patrick
Justin Meuse, MD
Freedom Perkins, MD
Martin Rossi, MD
Laleh Abedin
Kimberly Kosatka, NP
Annette Wilson, PA-C
Sarah Gibbons, DO
Marylyn Kajs-Wylie, APRN
Elizabeth Iskander, NP
Jorge Martinez-Prieto, MD

What's in a name?

Omotala A. Hope, MD, MHS

Assistant Professor of Neurology, Epilepsy Fellowship Director

McGovern Medical School, University of Texas Health Science Center- Houston

THE NEW CLASSIFICATION FOR SEIZURES AND EPILEPSIES

In case you missed it, the last decade has been a hotbed for revisions of definitions and classifications in the field of epilepsy as the field rushes to catch up with the rapid expansion of knowledge and to update old definitions to reflect new information. While there are other classification schemes, the International League Against Epilepsy (ILAE)'s is the most widely used classification scheme reflecting its role as the most authoritative body in the field of epilepsy across the globe. The ILAE presents its new classification for seizures and the epilepsies in Epilepsia April 2017, e-published in March 2017 (Fisher, 2017; Scheffer, 2017; Fisher, 2017). This is the culmination of more than a decade of work by the ILAE. An initial proposed classification scheme was published in 2010 (Berg, 2010). After feedback from clinicians and researchers across the globe, the ILAE submitted a revised classification for public comment and feedback in 2013 (ref). This final classification has been influenced by extensive feedback from the international epilepsy community and has incorporated perspectives from basic science research, drug development and from clinicians all over the world including lower and middle income countries such that it represents the best agreement possible on core ideas in categorizing seizures and epilepsies. Furthermore the language has been simplified to minimize errors and promote understanding by patients and families. In this summary, I will first review updated definitions of seizure and epilepsy published 2005 (Fisher, 2005) and further clarified in 2014 (Fisher, 2014) which were necessary precursors to the updated classification then present the new classification for seizures followed by the new classification for epilepsies. I will briefly discuss one controversial category of genetic epilepsies, some new terminology, and the implications for ICD coding for epilepsy (Jette, 2015).

UPDATED DEFINITIONS OF SEIZURE AND EPILEPSY.

It is important to first agree on basic/conceptual definition of a seizure before classifying or organizing into categories. A seizure is defined as "a transient occurrence of signs/and or symptoms due to abnormal excessive or synchronous neuronal activity in the brain" (Fisher, 2005). Epilepsy is a disorder of the brain characterized by an enduring predisposition to generate epileptic seizures, and by the neurobiologic, cognitive, psychological, and social consequences of this condition. The definition of epilepsy requires the occurrence of at least one epileptic seizure.

This conceptual definition of epilepsy was operationalized as either: 1) after two unprovoked seizures more than 24 hours apart (the old definition) or 2) one unprovoked seizure when the risk for another is known to be high (>60%) or reflex seizures and seizures that are part of an epilepsy syndrome constitute epilepsy; furthermore, epilepsy may be considered resolved when an age-dependent syndrome is outgrown or when a person is seizure-free for at least 10 years, the last 5 off anti-seizure medicines. (Fisher, 2014).

THE CLASSIFICATION OF SEIZURES.

This new classification scheme still carries many of the basic principles proposed by Gastaut in 1964 but has been influenced by modern research which shows that seizures are not only manifestations of local brain abnormalities but is also a network disease thus seizures could arise from cortical, thalamocortical, limbic and brain tem networks. Despite this knowledge, the classification is still an operational classification i.e. not based entirely on scientific data but it's flexible enough that new scientific concepts and additional data could be incorporated with time. The Task Force (ILAE Seizure Type Classification Task Force) further defined a seizure type as a useful grouping of seizure characteristics for purposes of communications across several domains including clinical care, teaching and research. An explicit goal was to make the classification understandable by patients and families and applicable to all ages including neonates. When a patient presents to your office with an event where there is loss of awareness or other transient behaviors in which a seizure is one of the possible explanations, the clinician must first make the diagnosis of seizure (explicitly epilepsy seizure) before applying this classification scheme to the event(s) in question.

THE CLASSIFICATION OF EPILEPSIES.

Once the seizure has been appropriately classified every effort should be made to classify the epilepsy and specify an epilepsy syndrome whenever possible. Efforts should also be made to determine the etiology of the epilepsy. The diagram below describes how the classification can be performed at multiple levels. In many clinical practice settings seizure classification may be the best level of classification attainable as the epilepsy classification may require imaging, long term video-electroencephalography and perhaps even genetic studies. To begin classification of the epilepsy make sure that the patient has a diagnosis of epilepsy (per updated 2014 criteria). Then classify the seizures are described above. The seizure type (s) informs the next level classification into focal, generalized, combined generalized and focal epilepsy and unknown epilepsy types.

Focal Epilepsies are disorders where the patients have focal seizures of all types including focal to bilateral tonic clonic. The diagnosis is based on clinical history but iiEEG with focal findings supports the diagnosis.

Generalized epilepsies are disorders where patients have generalized seizures. An individual may have a range of seizures including motor onset seizures such as tonic clonic, myoclonic tonic clonic but also non-motor seizures such as typical absence. The iiEEG will show generalized spike-wave activity which

ILAE 2017 Classification of Seizure Types Basic Version

Focal Onset		
*Modifier 1	Aware	Unaware
*Modifier 1	Motor	Nonmotor
# Focal to bilateral tonic clonic		

*The modifiers used above are not hierarchical; do not need to use both modifiers

This is a special category as it reflects seizure propagation not a unique seizure type but it is important because of its common occurrence; please note that the that there is deliberate use of the word bilateral here to differentiate from generalized as in generalized onset below.

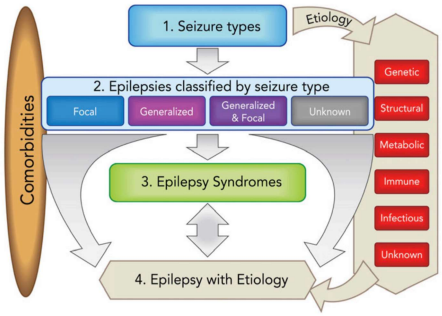
Generalized onset seizures
Motor • Tonic clonic • Other motor
Nonmotor (absence)

Unknown onset seizures
Motor • Tonic clonic • Other motor
Nonmotor (absence)
Unclassified

Figure 1. Adapted from Epilepsia April 2017. The basic classification of seizure types.

In 2010, the ILAE defined focal onset seizure" originating within networks limited to one hemisphere. They may be discretely localized or more widely distributed". Generalized onset seizure is defined as "originating at some point within and rapidly engaging bilateral distributed networks". The category of Unknown above is to be applied when the onset of the seizure was not witnessed. For example if a family member finds the patient already in the throes of a tonic clonic seizure it can be classified as unknown onset motor seizure. Thus, seizures are first separated by how they begin whether focally, generalized or unknown. For focal seizures, the first classifier is level of awareness. Focal aware replaces the term simple partial. Focal with impaired awareness or shorthand "focal unaware" replaces complex partial. The second classifier listed is motor vs nonmotor. These two classifiers are not hierarchical and one or both may be used depending on the situation or what is being emphasized.

It's also worth reviewing the expanded version noted below although it may not be necessary for many clinicians.



supportive of the diagnosis. In the case of tonic clonic seizure with normal EEG, clinician may need to elicit other evidence of a generalized epilepsy for ex history of myoclonic seizures also. The category of combined generalized and focal epilepsies exist because there are some patients with both generalized and focal seizures. For example patients with Dravet's syndrome may have both focal seizures and generalized seizures. The ii EEG is supportive of the diagnosis EEG is not essential for the diagnosis.

The category of unknown for the patient in whom you know he/she has epilepsy but there is insufficient information to inform what type. For example the patient several tonic clonic seizures as evidenced by lateral tongue bite but EEG was normal and no MRI obtained so seizure onset is unknown so epilepsy is unknown.

The next level of classification is the epilepsy syndromes. Epilepsy syndrome refers to the seizures plus a number of features such as age of onset, age of remission, seizure triggers, diurnal variation and often prognosis that have stood the test of time (see epilepsydiagnosis.org). The epilepsy syndrome also includes characteristic comorbidities such as intellectual dysfunction. There is an understanding of etiology in many syndrome for example Dravet syndrome is 2nd to mutations (often DE novo) the ubiquitous sodium channels, SCN1a.

ETIOLOGY

A big change in the new classification of epilepsy is the emphasis on etiology. The initial categories of structural/ metabolic vs genetic vs unknown have been expanded to include infectious and immune.

The categories are not hierarchical and the importance given to etiology depends on the circumstance. For example Tuberous Sclerosis is both a genetic and structural etiology for epilepsy but the structural etiology maybe emphasized for epilepsy surgery evaluation whereas the genetic may be emphasized with regards to genetic counselling for the family. The structural category is based on the idea that many structural abnormalities are associated with increased risk of epilepsy and in certain situations may define the seizure types and perhaps response to medical interventions. Some examples are gelastic seizures associated with hypothalamic hamartomas. Structural abnormalities may be genetic or acquired. Genetic etiology refers to epilepsy secondary to a known or presumed genetic mutation. For example twin studies and familial aggregation studies suggest that Juvenile Myoclonic Epilepsy (JME) is a genetic disorder but in most cases no specific genetic mutations has yet been found. On the other end of the spectrum for having the knowledge of a specific mutation, most patients with benign familial neonatal epilepsy have mutations in KCNQ2 or KCNQ3 genes. In autosomal dominant nocturnal frontal lobe epilepsy, the underlying mutation is only known in a small percentage of patients. Genetic does not mean inherited as an astonishing number of de novo mutations are being identified in mild and severe epilepsies. The infectious category does not refer to acute symptomatic seizures such as the acute seizures that occur with meningitis or encephalitis but refers instead the epilepsy that may follow for example the patient had meningitis at age 18 then two years later begins to experience focal unaware cognitive seizures. Other examples include epilepsy secondary to neurocysticercosis, cerebral malaria, and Zika. The metabolic category will overlap significantly with genetic as there is likely a genetic mutation that explains the metabolic disorder. However, rarely the metabolic abnormality will be acquired such as cerebral folate deficiency. The immune category describes epilepsy secondary to autoimmune mediated central nervous system inflammation. The hallmark disease anti- NMDA encephalitis. The unknown should be used when the etiology is unknown. This can easily be changed as more information becomes available with time and further investigations.

(continued on page 6)

ILAE 2017 Classification of Seizure Types Expanded Version

Focal Onset	
Aware	Unaware
Motor onset • automatisms • tonic • epileptic spasms • hyperkinetic • myoclonic • tonic	Nonmotor onset • autonomic • behavior arrest • cognitive • emotional • sensory
Focal to bilateral tonic clonic	

Generalized Onset	
Aware	Unaware
Motor onset • tonic clonic • clonic • tonic • myoclonic • myoclonic tonic clonic • myoclonic atonic • atonic • epileptic spasms	Nonmotor (absence) • typical absence • atypical absence • myoclonic absence • absence seizure with-eyelid myoclonia
Focal to bilateral tonic clonic	

Unknown Onset	
Aware	Unaware
Motor onset • tonic clonic • epileptic spasms	Nonmotor • behavior arrest
Unclassified	

Figure 2. Adapted from Epilepsia April 2017. The expanded classification of seizure types.

I have given a two examples to show how to classify seizures. Please read the excellent instructional manual published in Epilepsia (Fisher, 2017) for more details.

1) The patient typically has many sleep associated seizures with prominent thrashing and pedaling; iiEEG with right frontal spikes- focal motor hyperkinetic.

2) Patient describes déjà vu then loses awareness; witnesses report repetitive purposeless lip smacking, confusion, forced head to the right then right arm stiffening- focal unaware, motor with automatisms. You could also skip the unaware and just say focal motor with automatisms. If you want to emphasize why the patient should not drive you may use focal unaware.

CONTROVERSY IN THE USE OF GENETIC GENERALIZED EPILEPSIES VS IDIOPATHIC GENERALIZED EPILEPSIES

There is a group of generalized epilepsy syndromes that is particularly well known and generally pharmacoresponsive- Childhood Absence Epilepsy, Juvenile Absence Epilepsy, Juvenile Myoclonic Epilepsy and Generalized Tonic Clonic Seizures Alone (previously Generalized Tonic Clonic Seizures on Awakening), the classification of which stirred up significant debate. Scientifically, these syndromes are genetic, based on twin studies and population level studies but there was great reluctance to describe these as genetic generalized epilepsies due to the great stigma associated with the word genetic and a wish not to further stigmatize patients with these very treatable diseases. Based on clinician preference the task force will allow the old word Idiopathic generalized epilepsy only for these four syndromes despite the imprecision of the word idiopathic.

Generalized epilepsies
Idiopathic generalized epilepsies • childhood absence epilepsy • Juvenile Absence epilepsy • Juvenile Myoclonic Epilepsy • generalized tonic clonic seizures alone
Genetic generalized epilepsies

NOTABLE NEW TERMINOLOGY/ DESCRIPTORS.

Self-limited. This term can be applied to epilepsies that are expected to spontaneously resolve. Examples are CAE, epilepsy with centrotemporal spikes (benign rolandic epilepsy), and certain occipital epilepsies such as the early form described by Panayiotopoulos and the late form described by Gastaut.

Pharmacoresponsive. Used to imply that the epilepsy will likely respond to the appropriate antiepileptic drug.

Developmental and Epileptic Encephalopathies.
Epileptic encephalopathy now defined as where the epileptic activity itself contributes to severe cognitive and behavioral impairments above and beyond and beyond what might be expected from the underlying pathology alone. This term can be used to epilepsies at all ages beyond just the onsets in infancy and childhood. Many of these epileptic encephalopathies have a genetic etiology such as West Syndrome but some are acquired such as hypoxic ischemic encephalopathy. Many syndromes also have developmental consequences thus the term developmental and epileptic encephalopathy is recommended to be applied where appropriate. Malignant or catastrophic should no longer be used.

The World Health Organization (WHO) International Classification of Diseases (ICD) is a standard way to classify causes of morbidity and mortality, including epilepsy for half a century. In the US it serves the important function for billing for clinical care services. We in the US recently transitioned to the 10th version of this coding system with significant turmoil. Unfortunately this new classification of epilepsy is not reflected in the present ICD 10 system in wide use in the US. The ILAE is presently working with the WHO and ICD to make sure that this new classification scheme is reflected in the ICD version 11.

Overall, the new classification scheme does not fundamentally change the way we think about seizure physiology presently but it offers simpler terms and an organization scheme that will enable communication about epilepsy and seizures across different languages, among clinicians and between clinicians and researchers. It is simple enough to implement in day to day practice and also complex enough that it reflects the scientific advances over the past fifty years while flexible enough to incorporate new information as the science marches forward. Journal editors will request this new terminology in publications but there are no real implications for billing in clinical practice as yet. (Fisher, 2014)

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Neurology on the Hill Successes

Mike Amery, Esq., AAN Senior Legislative Counsel

The American Academy of Neurology's 15th annual Neurology on the Hill set records again this spring with 216 AAN members from 42 states meeting their members of Congress in Washington, DC. AAN advocates visited 272 congressional offices, created 2,072 tweets using #NOH17, generated 1.8 million social media impressions, and took lots of pictures of everyone with members of the House and Senate.

Once again, your colleagues were clad in AAN signature green bow ties and scarves that generated many conversations in congressional offices as well as in the halls and elevators about what our group was representing. The result is a significant increase in the profile of neurology to everyone on Capitol Hill.

The primary goal was to carry four messages to lawmakers:

- Congress should ensure that any changes to the Affordable Care Act are in alignment with the AAN's Principles for Health Care Delivery
- Members of Congress should cosponsor the Furthering Access to Stroke Telemedicine Act (S 431/HR 1148) that will allow Medicare to pay for stroke telemedicine consultations in urban and suburban areas.
- Congress should support full funding for the NIH including the BRAIN Initiative
- Congress should take steps to reduce drug prices that are impacting patients

Neurology on the Hill is a two-day event. Advocates go to Capitol Hill after receiving a full day of training on the issues and updates from key people in government. This year's program included an update on the amazing work of the BRAIN Initiative by AAN member Walter J. Koroshetz, MD, FAAN, who serves as the director of the National Institute for of Neurological Disease and Stroke (NINDS). This encouraged AAN advocates to let their congressional offices know of the benefits of this incredible program. Staff from the powerful House Energy and Commerce (E&C) Committee, which has jurisdiction over many health care issues, gave an update on the FAST Act and encouraged us to cross the 218 cosponsor threshold, which represents a majority of the House. The AAN generated 172 cosponsors for the same legislation in the last Congress. During NOH, the AAN's federal political action committee, BrainPAC, collected $56,000 from more than 100 attendees. BrainPAC hosted a dinner for major donors Monday evening. This event featured a key-note address by Energy and Commerce Committee Chair Greg Walden (R-OR), who discussed many of the major health care decisions being debated in Congress.

AAN members also were strongly encouraged to promote their efforts publicly on social media using the hashtag #NOH17. Doing so helps raise awareness of the event and the issues with policy makers, the public, and the AAN membership. By the end of the day, over 200 participants had generated nearly 500 tweets, reaching more than a million people, including patients, colleagues, friends, coworkers, family, and many of their associates, as well, as messages were forwarded and retweeted. Even seven legislators posted about their visits with the AAN members. They got to see a lot of enthusiasm and activism for neurology!

If you are interested in attending Neurology on the Hill 2018, visit the Public Policy page on the AAN's website for more information.

Texas Doctor Wins National Award For Memoir

Texas doctor Tom Hutton, M.D.'s memoir, *Carrying the Black Bag: A Neurologist's Bedside Tales* has been named among the winners in the Feathered Quill Literary Awards.

Sponsored by Feathered Quill, a leading web-based book review, the Feathered Quill Literary Awards is a national awards program that celebrates excellence in publishing. Recognizing books from both large and independent presses, the Feathered Quill Literary Awards honors the best books in numerous categories.

Carrying the Black Bag: A Neurologist's Bedside Tales, a memoir of Hutton's career in medicine, was awarded the Bronze medal in the "Best Debut Author" category. Published by Texas Tech University Press, *Carrying the Black Bag* is available in hardcover edition (6 x 9, 257 pages; photographs; ISBN: 978-0-89672-954-4)

According to Ellen Feld, Editor at Feathered Quill "We were overwhelmed by both the number and extraordinary quality of entries for this year's awards program. In particular, The Best Debut Author category was filled with worthy entries: consequently, it was difficult for our judges to pick among the many excellent contenders. Tom Hutton, M.D.'s memoir, *Carrying the Black Bag* was a real standout: compelling, well-written, and an incredibly beautiful and hopeful testament to the human spirit. It is our great honor to recognize Dr. Hutton among this year's Best Debut Authors. We can only hope he has more books in the works."

During his thirty-plus years of practicing in West Texas and Minnesota, physician and neurologist Tom Hutton discovered that a doctor's best teachers are often his patients. From these (extra) ordinary individuals, Hutton gained a wholehearted respect for the resourcefulness, courage, and resilience of the human spirit. Hutton's patients—and the valuable lessons they taught—served as the inspiration for Carrying the Black Bag. Part memoir and part tribute to the patients who faced major illness with grace, grit,

(continued on page 11


E. Golden, MD


J. Trivedi, MD

Evaluation and Treatment of Painful Peripheral Neuropathy

E. Golden, MD • J. Trivedi, MD

UT Southwestern Medical Center, Dallas

CASE

A 55 year old Caucasian woman presented for evaluation of foot tingling. It started about two years ago, initially as a "tingling or vibrating" sensation in both feet. Over time the sensation intensified and ascended. It was now at the level of the calves, and very painful with "cold, burning, shocking" sensations; the pain kept her awake at night. She felt that the bottoms of her feet were numb. She had no other medical problems, except that she had been told that her serum glucose was "borderline high" in the past. There was no family history of similar symptoms. She was a homemaker who worked out at the gym several times per week. She ate an unrestricted and well-balanced diet and did not take any supplemental vitamins. She drank about two glasses of wine per week; she denied any tobacco or illicit drug use.

Her neurological exam was unremarkable except for decreased temperature and pinprick sensation distally to the level of the mid-shin, and mildly decreased timed vibratory sensation at the toes (5 seconds).

CLINICAL QUESTIONS

- What workup is indicated in this case?
- What are the options for symptom management, and what is the evidence behind them?
- How to approach initial choice of therapy?

INTRODUCTION

Peripheral neuropathy is a common disorder, with prevalence estimated at 2.4%, rising to 8% when considering the elderly population.1 Patients present not only with the negative symptoms of numbness and reduced sensation, but also with a variety of positive symptoms which may be described as burning, stabbing, stinging, squeezing, aching, cramping, shooting, freezing, pins and needles, broken glass, and vicelike sensations.2 These symptoms prompt patients to seek medical attention, not only to evaluate the underlying etiology but also to seek relief from the pain.

WORKUP

A full discussion of the evaluation of peripheral neuropathy is beyond the scope of this article; we will focus on several high-yield points. Our patient presented with adult-onset, slowly progressive, sensory predominant, length-dependent neuropathy. A detailed medical history should be assessed; a variety of medical conditions can cause neuropathy, including diabetes, nutritional deficiency, human immunodeficiency virus (and treatment), cancer (and treatment), uremia, among others. A family history should be obtained, including family history of high arches, neuropathic pain, or otherwise unexplained need for assistive device with ambulation. Social history should include the amount and frequency of ethanol consumption, as well as any particular dietary constraints that might suggest a nutritional deficiency. A detailed medication review should be performed; many different medications can cause neuropathy. Furthermore, the patient should be asked specifically about other supplements (particularly B complex, as this is often touted for nerve health, but excess vitamin B6 may cause a painful neuropathy).

Regarding laboratory workup, the American Academy of Neurology released a practice parameter for evaluation of distal symmetric polyneuropathy.3 Laboratory screening tests with the highest yield include blood glucose (possibly followed by glucose tolerance test), serum B12 (with methylmalonic acid, with or without homocysteine), and serum protein immunofixation electrophoresis. Genetic testing may be considered if family history and electrophysiology is consistent with a hereditary neuropathy. Aside from these recommendations, the clinical scenario should guide any further investigation.

Electrophysiologic studies are performed to evaluate the extent and severity of the neuropathy and to also assess for certain patterns that may be seen in hereditary neuropathies. For instance, presence of uniform slowing of conduction velocities would point towards Charcot-Marie-Tooth, type 1A. This can subsequently guide genetic testing.

SYMPTOM MANAGEMENT

Although a variety of medications are commonly used in clinical practice, the evidence behind these is variable. Randomized controlled trials have been performed, but there are many challenges in design and interpretation.2 These include a significant placebo response, uncertainty regarding what constitutes a meaningful clinical response, short duration of studies, conflicting evidence for some agents, limited data for neuropathic pain other than that from diabetic neuropathy, and limited data regarding combination therapy.

ANTIDEPRESSANTS

The older tricyclic antidepressants (TCAs) have been extensively studied and utilized in the treatment of neuropathic pain. They presumably exert their analgesic effect by modulating voltage-gated sodium channels and inhibiting norepinephrine and serotonin reuptake, thus augmenting descending inhibitory pathways. A placebo-controlled trial of amitriptyline in diabetic neuropathy showed a benefit for neuropathic pain.4 Doses were started at 25 mg and up-titrated to 150 mg or maximum tolerable (mean 90 mg daily). Pain relief was seen as early as two weeks into treatment and was found to be independent of mood alteration. Side effects may be limiting when using TCAs: dry mouth, sedation, urinary retention, cardiac arrhythmia, orthostatic hypotension, dizziness, constipation, and weight gain. These may be less prominent with the secondary amines (nortriptyline, desipramine).

The newer serotonin and norepinephrine reuptake inhibitors (SNRIs) have a higher and more balanced affinity at transporter sites and are also used in the treatment

of neuropathic pain. Duloxetine was the first FDA-approved agent for the treatment of diabetic peripheral neuropathic pain; it also carries an indication for fibromyalgia. In placebo-controlled trials,5,6 doses of 60 and 120 mg per day showed benefit for pain control as early as one week, and dropout due to side effects was less than 20%. Doses of 120 mg per day were no more efficacious than 60 mg per day and caused more side effects. Venlafaxine also has demonstrated efficacy in diabetic neuropathic pain. A placebo-controlled trial7 demonstrated efficacy of doses from 150-225 mg per day; 50% pain reduction was seen at six weeks. Side effects of SNRIs commonly include nausea, somnolence, and dizziness.

ANTICONVULSANTS

Gabapentin and pregabalin are both commonly used in the treatment of neuropathic pain. Pregabalin carries an FDA indication for diabetic neuropathy and fibromyalgia, and both carry indications for post-herpetic neuralgia. The mechanism of action is thought to stem from high-affinity binding to the alpha-2-delta subunit of voltage-gated calcium channels, which inhibits neurotransmitter release and reduces neuronal hyperexcitability. In an 8-week trial for painful diabetic neuropathy,8 gabapentin monotherapy (900 mg per day up-titrated to 3600 mg per day or maximum tolerated dosage) significantly reduced daily pain severity compared to placebo; secondary outcomes of quality of life measurements also improved significantly. Pregabalin has also demonstrated efficacy in placebo-controlled trials.9,10 Doses of 300 and 600 mg per day were shown to improve pain as early as week 1; sleep interference scores and other global quality of life measures also improved. Side effects include dizziness, somnolence, and peripheral edema.

A variety of other anticonvulsants have also been studied for the treatment of neuropathic pain. Data on carbamazepine is too limited to draw firm conclusions,11 and data on oxcarbazepine are mixed.12 Two small placebo-controlled trials showed that valproic acid was beneficial for pain in diabetic neuropathy.13,14 Studies of topiramate and lamotrigine have yielded mixed results.2 Several trials have investigated the use of lacosamide for painful diabetic neuropathy.15-18 While each did suggest efficacy for pain relief, effect sizes were small and dose-response relationship was inconsistent, leading to a level B recommendation that lacosamide should probably not be considered for the treatment of painful diabetic neuropathy.12

ANALGESICS

Tramadol, a centrally-acting non-narcotic analgesic medication with monoaminergic and opiate effects, has been shown to be effective in the treatment of neuropathic pain. A placebo-controlled trial19 showed that in painful diabetic neuropathy, tramadol at an average dose of 210 mg per day was effective for pain as well as measures of physical and social functioning. Sleep measures were not improved. Side effects include nausea, constipation, headache, and somnolence. Opiate analgesics have also demonstrated efficacy. A 6-week placebo-controlled trial of controlled release oxycodone demonstrated effectiveness at an average dose of 37 mg per day.20 However, concerns regarding adverse effects and potential for dependence and addiction make opioids a less desirable option.

TOPICALS

Topical agents are an appealing alternative for treatment of painful neuropathy due to a lack of systemic side effects and drug-drug interactions. Unfortunately, clinical trial data are limited. Capsaicin is an alkaloid extracted from chili peppers that depletes substance P from sensory nerves; it causes an initial, sometimes intense, burning sensation upon topical application. Results from placebo-controlled trials have been mixed, though generally favorable when diabetic neuropathy is specifically studied.2 A major difficulty in these studies is blinding to the burning sensation, as well as high response rates to active and vehicle placebos. Topical lidocaine is another option. A 4-week, open-label randomized trial21 showed that 5% lidocaine medicated plaster was comparable to pregabalin in the treatment of painful diabetic neuropathy and superior for post-herpetic neuralgia, with fewer side effects than pregabalin. A variety of compounded topical agents are used with combinations of a wide variety of agents including gabapentin, ketamine, amitriptyline, baclofen, and others; data are limited.

OTHER

One small randomized trial showed benefit for intradermal injection of botulinum toxin for diabetic neuropathic pain; sleep quality also improved.22 Mexiletine, a class 1B antiarrhythmic agent and oral analogue of lidocaine, has been investigated for treatment of various etiologies of painful neuropathy, with mixed results.2

COMBINATION THERAPY

Combination therapy for treatment of neuropathic pain may be indicated in some cases, in an effort to increase efficacy while minimizing side effects that may develop with higher doses of the individual medications. Disadvantages may include increased cost, difficulty with compliance, drug-drug interactions, and possibly increased side effects. Multiple studies have been performed testing various combinations of drugs. A recent Cochrane review23 summarized these studies. The authors found multiple high-quality studies indicating increased efficacy with combination therapy, though with some increase in side effects such as sedation. However, given the variety of tested combinations, there was not enough evidence to specifically recommend a particular combination of agents.

NOTABLE STUDIES OF COMBINATION THERAPY INCLUDE:

- The combination of gabapentin and sustained-release morphine achieved better pain relief at lower doses of each agent, though with some increase in side effects of dry mouth and constipation.24
- Addition of oxycodone 10 mg per day to pregabalin did not improve pain relief in a study of diabetic neuropathy and post-herpetic neuralgia.25
- A randomized, controlled crossover trial showed that the combination of gabapentin and nortriptyline yielded better pain control than either agent alone.26
- In a study of post-herpetic neuralgia, a combination of pregabalin with 5% lidocaine medicated plaster yielded better pain control than either agent alone.27

CHOICE OF THERAPY

The American Diabetes Association recently released a position statement on diabetic neuropathy,28 and the Ameri-

(continued on page 10)

Table: Suggested medication sequence for treatment of neuropathic pain30

First Line	Second Line	Third Line
TCAs	Tramadol	Add: bupropion, citalopram, paroxetine
Duloxetine	Other opioids	Anticonvulsants: carbamazepine, lamotrigine, valproic acid, topiramate, oxcarbazepine
Pregabalin		Low concentration capsaicin
Gabapentin		Dextromethorphan
Topical lidocaine		Memantine
		Mexiletine

can Academy of Neurology issued an evidence-based guideline on the treatment of painful diabetic neuropathy.12 Both issued the highest level of recommendation regarding the use of pregabalin for the treatment of diabetic neuropathic pain; gabapentin, SNRIs, and TCAs also received at least level B recommendation.

As in other clinical scenarios, choice of initial therapy will need to take into consideration medical comorbidities, drug-drug interactions, and patient preferences regarding cost, route and frequency of administration, and potential side effects. Two common reasons for treatment failure are inadequate dose titration and immediate initiation of polypharmacy;29 therefore, a trial of four to six weeks is recommended before switching or adding medications.2

Suggested medications are summarized in the table.

CASE CONCLUSION

Laboratory evaluation included normal thyroid stimulating hormone, vitamin B12, serum protein electrophoresis. Glycosylated hemoglobin was at the high end of the reference range at 5.6%. Nerve conduction studies and electromyography were normal. Patient was diagnosed with cryptogenic sensory polyneuropathy; however, it may also have been related to impaired glucose tolerance given her borderline A1c. She was advised to follow up with her primary care physician regarding this.

Patient was started on gabapentin, which provided some symptom relief, but pain returned several months later. She was then prescribed duloxetine, gradually uptitrated to 60 mg daily, which provided sustained clinical benefit.

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TNS Advocacy Report 2017

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

It's been a slow year actually. I'm not good enough at the politics of it to be able to explain why exactly that is, but it is. That can be considered good or bad depending on how you look at it, they can't do much good, but not much damage either. In general a lot of health bills seem to have passed the Senate and are hung up in the House of Representatives, not sure why that is, but it is.

The House of Medicine has had some wins, and some additional ones are probably coming down the pike. Most are not particularly glamorous wins, just solid ones that help us practice and keep patients safe. The 'surprise billing' bill by Sen. Hancock and Rep. Frullo that preserves our right to mediation for out of network billing has been voted out of the Senate, and is waiting to be voted on the House floor. It should pass. The telemedicine bill that was negotiated by Sen. Schwertner and the TMA should also pass. It creates a framework for telemedicine care in Texas. The advanced practice nurses request independent practice authority every legislative session, just as they did this year. And they did not get it this year, just as they haven't in years past. We are happy with that.

A bill TNS has led on, SB 680 by Sen. Hancock/HB 1464 by Rep. Bonnen, MD pertaining to physician override of insurance mandatory drug 'step therapy' has cleared both chambers and is headed for the Governors desk. SB 2210 by Sen. Hancock and HB 2760 by Rep. Bonnen, MD was also voted out of the Senate and is on the House floor for a vote. It mandates that health plan directories be updated frequently, every 3-5 days instead of the ….never…that they seem to do now. It should be voted out and sent to the Governor's desk.

The House of Medicine supports raising the smoking age to 21; ensuring a parent's right to know about the vaccination exemption rates at each school; banning texting while driving; restricting the carrying of handguns at state hospitals; and restricting the use of tobacco products on state owned campuses and facilities. All of these bills have been moving along, but none are completely thru the process or ready to be signed as I write this article.

The Texas Medical Board is up for Sunset review this year and that has been tricky. There is some debate about whether their reauthorization will pass or there will be a temporary one. If it does not pass, then the Intrastate Compact will not pass, which would have the effect of continuing to make it difficult to get secondary licenses from other states.

A TMA/TNS priority has been to get a law passed that hospitals can't use maintenance of certification (MOC) as the sole criteria for determining hospital privileges. Sen. Dawn Buckingham's bill passed the Senate but Dr. Greg Bonnen's bill has had some trouble getting thru the House. It is still pending at this point but many thanks to those doctor legislators for working for us. Our own Dr. Kim Monday has carried much of the load on this one.

The budget has been tight this year – the legislature voted in some structural deficits last session, and there is some deficit on top of that this year. Get used to living in a state with a negative budget …it's here to stay I suspect.

Having said that, Medicaid is always at the top of the discussion list for physicians for obvious reasons including that it affects us along with so many Texans and so much of our budget. Nearly 30 percent of state plus federal aid in the 2016-17 Texas budget went to Medicaid ($63 billion/$211 billion), about 22% of state funds alone were appropriated for Medicaid. As the case load grows as our population grows there doesn't seem to be any will at all to make the fee schedule more equitable. In fact, unfortunately, they are still looking for ways to cut spending. It looks like dual-eligibles will carry the brunt again this year. There is general recognition that these cuts fall hardest on communities and physicians in south Texas.

GME is faring better. Legislators in both the House and the Senate are honoring their commitment to try to fund 1.1 GME spots for every 1 medical student graduate and there is additional money in each budget to do this. There is a bill now pending however that will require all new medical schools to have GME funding lined up ahead of time so that the state is not playing catch up.

As always, please attend fundraisers for your candidates when you can – it helps to put a name with a face and we rely on those connections during session. Out of ~7000 bills filed this session, almost 1500 had to do directly with the practice of medicine. Physicians cannot just ignore the Texas Legislature. If you have bills or issues that you would like us to work on, please contact us thru Ky. We would be happy to help if we can.

Memoir

CONTINUED FROM 9

and dignity, Carrying the Black Bag invites readers to experience what it is like to be a doctor's hands, eyes, and heart. Imagine the joy of witnessing a critically ill five-year-old who, against all odds, claws her way back from a coma and near certain death. Meet a lonely Texas widower with Parkinson's disease who hosts elaborate pinochle parties for a pack of imaginary canines. Step into the surgical booties of the author when he attempts to deliver his own child amid heart-stopping obstetrical complications—during a paralyzing Minnesota blizzard. Through real-life patient narratives, Hutton shines light on ordinary people facing extraordinary challenges. Moreover, this captivating tale captures the drama of medicine—its mystery, pathos, heroism, sacrifice, and humor. Tom Hutton, M. D., is an internationally-recognized clinical and research neurologist and educator. The past president of the Texas Neurological Society, Dr. Hutton served as professor and vice chairman of the Department of Medical and Surgical Neurology at the Texas Tech School of Medicine. He now lives on his cattle ranch near Fredericksburg, Texas. Visit Tom Hutton online at: https://jthomashutton.wordpress.com.

A Case of Reversible Hemichorea-Hemiballismus

Manzure Mawla, DO–TNS Resident Representative
The University of Texas at Austin Dell Medical School

CASE PRESENTATION

An 89 year old male with a past medical history of diabetes mellitus type II and hypertension presented with a 1 month history of abnormal movements on the left side of his body. It started off with repetitive writhing and twisting of the hand which occurred involuntarily and intermittently. This progressed over a course of a few weeks to whole arm "flailing". His family described it as if he "is conducting an orchestra". They also noted that his gait had changed with abnormal movements in his left leg and that he would often walk in circles. At baseline he is completely independent and lives an active lifestyle. At home they noted that his blood sugar had been consistently in the 300mg/dL to 400mg/dL range.

His physical exam revealed significant hemichorea-hemiballismus in the left upper and lower extremities, with no other deficits in neurologic testing. His serum laboratory testing showed initial elevation of blood glucose level at 426mg/dL and a hemoglobin A1c of 12.3%.

An initial CT scan of his head was done which showed subtle hypodensity of the basal ganglia bilaterally.

A follow up MRI of the brain showed increased T1 signal and decreased T2/Flair signal in the basal ganglia bilaterally.

The patient was treated with IV insulin during his admission and the symptoms began to abate within hours. The patient was seen again in follow up after 1 month, and the symptoms had then resolved completely with strict glucose control.

INTRODUCTION

Hemichorea (from the Greek word "to dance") is a movement disorder characterized by involuntary and irregular flinging of the upper and lower extremity that is unilateral. Hemiballismus (from the Greek word "to throw") refers to high amplitude, violent flinging and flailing of the extremities that is also unilateral.

The common causes of this disease include ischemic stroke, neoplasm, nonketotic hyperglycemia, Wilson's disease, and thyrotoxicosis. Literature has shown that this occurs mostly in the elderly population, people of Asian descent, and in females, which suggests that there may be a genetic predisposition.

This case illustrates how accurate identification of pathology and rapid treatment can quickly and completely resolve symptoms.

DISCUSSION

Hemichorea-hemiballismus is often characterized by unilateral, involuntary movements that develop over a period of hours to days. This syndrome occurs because of hyperglycemia-induced perfusion changes and vascular insufficiency, which causes transient dysfunction of the striatum. There is ischemic excitotoxicity of GABAergic neurons which causes increased inhibition of the subthalamic nuclei and excitatory cortical output.

The diagnosis is made based on clinical findings with the presence of choreiform and ballistic movements along with radiologic features. Characteristic findings on a CT scan show areas of hyperdensity in the basal ganglia. A T1-weighted MRI shows hyperintensity in the striatum and globus pallidus, and abnormal signal may extend to the medial part of the cerebral peduncle in the midbrain, along the striatonigral pathway. Positron emission tomography (PET) done in patients with this syndrome shows reduce glucose metabolism in the basal ganglia.

Hemichorea-hemiballismus syndrome can be medically managed, and the symptoms can be partially or completely controlled after immediate restoring of blood sugar to normal levels. Radiographic resolution is typically not seen until there has been 6 months of good blood sugar control.

CONCLUSION

Hemichorea-hemiballismus is a movement disorder with many causes such as ischemic stroke, neoplasm, and thyrotoxicosis, and is rarely caused by nonketotic hyperglycemia. It is beneficial to check a blood glucose level in patient's who present with these symptoms, as with early identification and treatment this process can be reversed. Neuroimaging is also beneficial with diagnosis as mineralization can be observed in the basal ganglia on CT images, and T1 and T2/Flair sequences on MRI. Multiple case reports in literature show resolution of symptoms, suggesting that this is a reversible process which has a good prognosis.

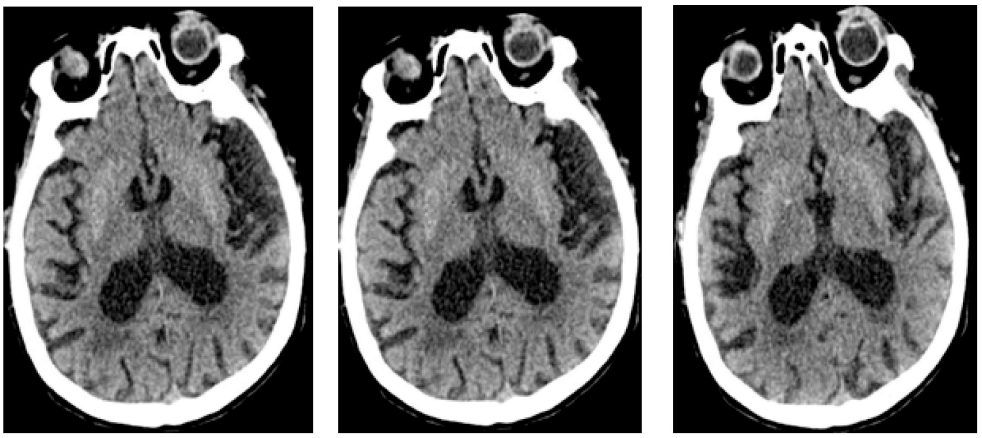

Figure 1. CT Head w/o contrast, axial view

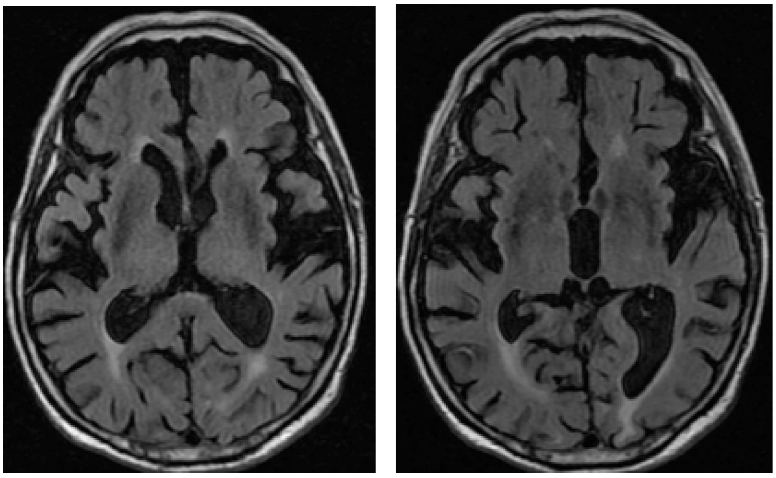

Figure 2. MR Brain T2/Flair Sequence, axial view

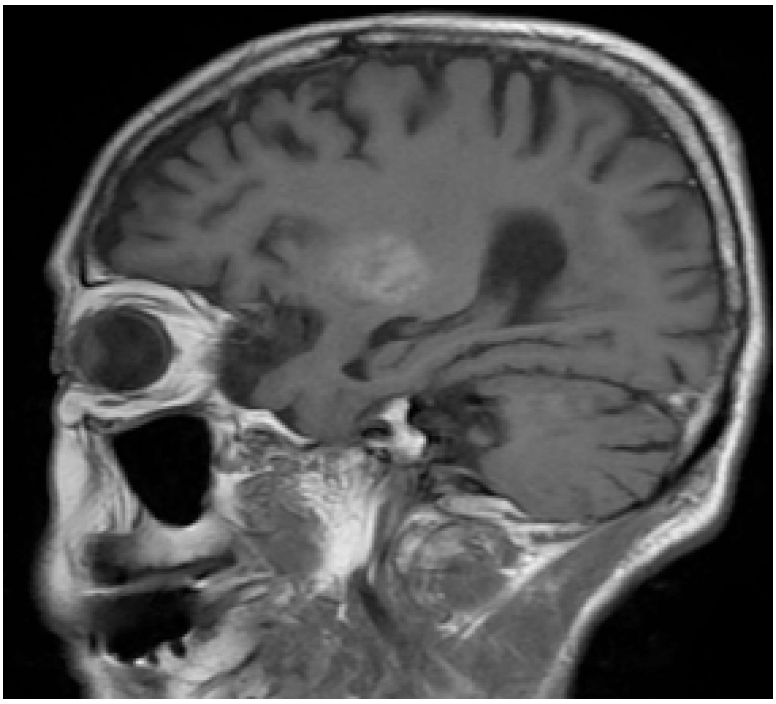

Figure 3. MR Brain T1 sequence, axial vie

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In accordance with the American with Disabilities Act, please let us know in writing, of any special accommodations you may need.

Registration Deadline: July 14, 2017 ★ Hotel Deadline: July 6, 2017

SAVE THE DATE!

TNS Winter Meeting
Feb. 2-4, 2018 • Austin, TX

Hi fellow Neurologists!

I am delighted to be the program director for the 2018 Winter TNS Conference in Austin from Friday, February 2nd through Sunday, February 6th, 2018. The education committee is hard at work preparing a relevant, comprehensive, and fun program for you to enjoy. We also aim to get our residents more involved in the organization and conference since they're our leads of tomorrow. Please do save the date, and I look forward to serving you this term.

Michael Soileau, MD

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