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Highlights From AES2020, a Virtual American Epilepsy Society Experience

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Abstract

Due to COVID-19 a live, in-person meeting was not possible for the American Epilepsy Society in 2020. An alternative, virtual event, the AES2020, was held instead. AES2020 was a great success with 4679 attendees from 70 countries. The educational content was outstanding and spanned the causes, treatments, and outcomes from epileptic encephalopathy to the iatrogenicity of epilepsy interventions to neurocognitive disabilities to the approach to neocortical epilepsies. New gene therapy approaches such as antisense oligonucleotide treatment for Dravet syndrome were introduced and neuromodulation devices were discussed. There were many other topics discussed in special interest groups and investigators' workshops. A highlight was having a Nobel prize winner speak about memory processing. Human intracranial electrophysiology contributes insights into memory processing and complements animal work. In a special COVID symposium, the impact of COVID on patients with epilepsy was reviewed. Telehealth has been expanded rapidly and may be well suited for some parts of epilepsy care. In summary, the epilepsy community was alive and engaged despite being limited to a virtual platform.

Keywords

annual meeting, epilepsy, treatment, education, cognition

Introduction

COVID-19 has impacted all our lives. When the Annual Meeting Committee met in February 2020 to plan the American Epilepsy Society (AES) meeting 2020, we were unaware of what lay ahead of us. After initial stay-at-home orders and lock downs in spring of 2020, we were hoping we could get together for our meeting in December in Seattle. It quickly became apparent that this was likely not possible. We were forced to cancel the in-person meeting and move to a virtual alternative. The Annual Meeting Committee, with the very committed staff of the AES, worked hard to plan a completely new and unknown event, a virtual AES meeting, which would certainly fall short of opportunities for networking, talking to our friends and just being together. Despite all adversity, the AES2020 as a virtual event was outstanding and engaging. It enriched the knowledge and practice of all who attended. We had 4679 attendees from 70 countries participate; 25% of attendees were

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first timers, which speaks also to the vibrancy and accessibility of the meeting. Despite the limitations of the virtual platform there was social interaction, scientific exchange, interactive poster sessions, and many informative presentations.

We really want to thank the very dedicated staff of the AES, without whom the event would certainly not have been possible. This article is a brief overview of a selection of symposia presented at the meeting.

2020 Presidential Symposium

Epileptic Encephalopathy: Causes, Treatment, and Outcomes

Gaillard WD, Weckhuysen S, Amy McTague A, Theodore WH, Braun K, Smith ML

Epileptic encephalopathy has many causes including the underlying etiology of the epilepsy and the ongoing seizures or interictal activity. There is increasing recognition that early targeted intervention may ameliorate if not reverse aspects of encephalopathy. This is predominantly the realm of pediatrics but extends into adulthood. Childhood epilepsy has unique features, as there are age-specific epilepsy syndromes, ongoing brain development that interacts with the epilepsy, and the prospect of plasticity and recovery of function. This symposium reviewed the increasing appreciation of genetic mechanisms of disease and evolving concepts and approaches to disease modifying treatment and also addressed the focal epilepsies. There is also growing awareness of the potential benefits of early epilepsy surgery, increasingly a treatment choice in children, and effects on cognition and behavior.

Childhood epilepsy occurs in the setting of the maturation of distributed cognitive networks. The several cognitive networks have different trajectories for maturation: vision, followed by motor control, language by 6 to 10 years old, visual spatial by 10 years old, and episodic memory material specificity in the teens.[1] These networks are bilaterally represented, but some such as language, memory material specificity, and some aspects of visual spatial systems become lateralized in this time frame.[2] Paradoxically, lateralized cognitive systems can sustain (early) focal injury better than bilateral systems (vision, motor executive function).[3] This likely occurs because persistence of the immature bilateral patterns in the unaffected hemisphere can sustain function.[4] Global injury is associated with more severe cognitive outcomes than acute focal injury. These observations are important for considering the timing and aggressiveness of epilepsy surgery.

The developmental and epileptic encephalopathies (DEEs) are characterized by severe early-onset epilepsy and neurodevelopmental problems, and often have a genetic basis.[5] Lately, several success stories of "precision therapies" have been described for genetic DEEs. Until we manage to safely correct the genetic variant directly on a DNA level though, we have to understand the variants in DEE genes and their consequences on potential therapies. Indeed, variants in several DEE genes can act in either a loss of function or gain of function way, sometimes leading to distinct clinical entities that warrant a different therapeutic approach.[6]

Medical modification of disease expression and encephalopathy is becoming a reality. As the genomic revolution has revealed the genetic basis of the DEEs, several pathway-based, RNA and gene therapies that directly address the underlying etiology have emerged.[7] These include several already at clinical trial stage, such as antisense oligonucleotide treatment in Dravet syndrome, along with promising preclinical data for *SCN8A*-related epilepsy and other channelopathies.[8,9] Further advances including gene replacement therapies and CRISPR-Cas9 genome engineering with the potential to restore normal function are on the horizon.[10] However, the window of effective intervention remains unknown and natural history studies will be vital to assess impact on developmental trajectories and comorbidities.

Children with generalized or focal epilepsy syndromes may have cognitive deficits at seizure onset in concert with structural and functional brain abnormalities.[11] Cognitive development parallels healthy controls. Adults with focal epilepsy can show ongoing cognitive impairment and structural and functional loss associated with early seizure onset, high seizure frequency, long epilepsy duration, low baseline function, and lower levels of education. As many as 25% to 40% of adults with focal epilepsy appear to be at risk for ongoing cognitive impairment. Some patients may have a lower baseline, but not "decline" compared with healthy controls. Older patients may be at increased risk for dementia. For both children and adults, optimal social and educational opportunities as well as treatment of comorbidities such as diabetes, hypertension, depression, and social isolation can ameliorate risks of progression.[12]

Early surgery improves seizure and cognitive outcome in children.[13] Five years after epilepsy surgery on average 67% of children are seizure-free, with differences in subgroups largely driven by differences in etiologies. The mean interval from seizure onset to surgery in children is 5.3 years. Shorter duration of epilepsy is an important and independent predictor of postoperative seizure-freedom. Early surgery also prevents further cognitive decline in infants with epileptic encephalopathy, improves eventual cognitive functioning of children, and allows earlier withdrawal of anti-seizure drugs. Epilepsy surgery may even be considered in children with well-controlled epilepsy; it is an early treatment option rather than a last resort.

Surgical candidates, even those without epileptic encephalopathies, have high rates of cognitive impairment. Cognitive outcomes of surgery depend on the overlap of the resected area with functional tissue, potential neuroplasticity, and individual differences in development.[14] Cognition does not change in approximately 70% of children within the first year after epilepsy surgery, with equal numbers showing losses or gains. Studies find similar changes in children with intractable epilepsy who do not undergo surgery. Improvements in intelligence and aspects of memory are seen in longer-term follow-up. Change relates to seizure status rather than whether the child had surgery. Epilepsy

duration predicts both pre- and post-operative cognition, but typically not change in cognition.[15]

Epilepsy Therapies Symposium

Iatrogenicity in Epilepsy Care

Lawrence J. Hirsch, MD, and Dean Naritoku, MD

This year's Epilepsy Therapy Symposium was entitled "Iatrogenicity in Epilepsy Care." To start off, Dr Kimford Meador discussed "Cognitive Iatrogenicity."[16] Anti-seizure medications (ASMs) can produce adverse cognitive effects, especially in high doses or polytherapy. Fetal exposure to some ASMs can cause irreversible impairments (including cognitive) although risks are uncertain for most ASMs. Women of childbearing potential should be informed of risks and encouraged to take folate. Risks to cognition from epilepsy surgery depend on the function of the area resected. Stimulation therapy for epilepsy does not appear to have cognitive risks.

Dr Heidi Munger-Clary reviewed "Psychiatric Iatrogenicity."[17] This includes direct impact from ASM changes or epilepsy surgery techniques, and indirect effects such as epilepsy monitoring-triggered postictal psychiatric phenomena or ASM interactions with psychotropic drugs. Psychiatric iatrogenicity is mitigated by carefully selecting epilepsy therapies for high-risk patients (those with personal or family history of psychiatric condition or active symptoms) and managing psychiatric conditions prior to epilepsy surgery.

Dr Piero Perucca covered "Iatrogenicity from Anti-Seizure Medications," concentrating on idiosyncratic reactions.[18] He reviewed definitions, pathophysiological mechanisms, and important examples of idiosyncratic reactions of ASMs. He highlighted risk factors for such reactions, focusing on recent advances in the field. He discussed management approaches, emphasizing the importance of consideration of risk factors in drug selection, prompt recognition of potentially serious idiosyncratic reactions and discontinuation of the offending agent, which should be substituted with a medication with the lowest risk for cross-reactivity reactions.

Dr Jacqueline French continued with "Iatrogenicity from Anti-Seizure Medications," covering all other adverse effects and general principles.[19] She concentrated on dose-related side effects and outlined that these could in some cases be prevented by slowing titration, eliminating background drugs that were more likely to cause pharmacodynamic interactions, or reducing the amount given in each dose by increasing dosing frequency (eg, from twice a day to thrice a day). She then discussed individualizing therapy by considering comorbidity, age, and gender when selecting therapies.

Dr Barbara Jobst discussed "Iatrogenicity from Neuromodulation." Deep brain stimulation (DBS) of anterior nucleus of the thalamus and responsive neurostimulation are intracranial stimulation approaches, vagal nerve stimulation is applied extracranially. Usually, only hemorrhages and infections are seen as direct consequences of neuromodulation.[20] With intracranial stimulation, the hemorrhage rate per implantation ranges between 2.7% and 4.5%, and the infection rate over the lifetime of those devices is around 12%. Hardware related iatrogenicity such as lead breakage or device malfunction occurs in 1% to 2%. Stimulation-related iatrogenicity such as paresthesias with DBS occur in 4.5%. It is difficult to assess the effects on depression, worsening of seizures, and cognition with any stimulation device as the prevalence is high in epilepsy independent from neurostimulation. Neuromodulation devices reduce the incidence of sudden unexpected death in epilepsy (SUDEP). Vagal nerve stimulation has less risk of hemorrhage but can frequently cause hoarseness and rarely vocal cord paralysis. Sleep apnea can worsen with vagal nerve stimulation and some studies have shown that DBS can trigger arousals during sleep.

Finally, Dr Gonzalez-Martinez reviewed "Iatrogenicity from Epilepsy Surgery." He started the presentation by describing the most common complications related to epilepsy surgery. More specifically, he described the complications related to resective procedures followed by nonresective interventions. He also described the iatrogenic events related to invasive monitoring with a brief comparison between the subdural method and the stereo-EEG method.[21] In the last part of his presentation, he discussed the mechanisms and rates of worsening seizures after surgery. He concluded by stating that epilepsy surgery is safe, but efforts must be taken to further reduce the morbidity and adverse events related to surgery.

The audience was reminded that although iatrogenicity should be kept in mind, prevented or minimized, and recognized as soon as possible, it should also be weighed against the risks of *not* providing the treatment—that is, the significant risks of living with uncontrolled seizures.

Best Practices in Clinical Epilepsy Symposium

Neurocognitive Disabilities, a Best Practice

Shanna Guilfoyle, PhD, Madona Plueger, MSN, APRN, ACNS-BC, CNRN

Challenges can arise in providing comprehensive care for persons with intractable epilepsy, particularly among patients with neurocognitive disabilities. The symposium presented the latest research and novel approaches that address quality of life and integrate examples of an interprofessional approach for care. The supportive thread throughout was the rich inclusion of our patient, Adam.

Dr Mackenzie C. Cervenka started the symposium by addressing hospital admissions of patients with intellectual developmental disabilities (IDD) to the Epilepsy Monitoring Unit (EMU).[22] The unique challenges of individuals admitted to the unit, as well as their caregivers and treating team were discussed. The patient Adam offered key takeaways that brought the message home from the lens of a patient. He offered insights into the importance of communication with his providers and the need for explanation in the unfamiliar EMU environment.

Meghann Soby, LCSW, addressed barriers to transitions of adolescent and young adults with IDD and the importance of involving patients in decision-making at age 18, stressing independence as part of the clinical care team. This session addressed the understanding of legal process at transition to adulthood and health care proxy-psychosocial support.

Jay Salpekar, MD, FANPA, presented an overview of the treatment of patients with psychiatric comorbidities.[23] His presentation highlighted the importance of balancing the improvement of quality of life, as well as the behavioral outcomes in relation to seizure outcomes. Emphasis on knowledge of the physiological overlay of pharmacologic and nonpharmacologic interventions were provided by interactive video recordings between our patient representative and his provider.

Saskia Hendriks, MD, PhD, spoke on the ethical and legal concerns that exist with this vulnerable population.[24] The ethical underpinnings of the informed consent process and the importance of assessing a person's ability to make decisions provided attendees with the opportunity to recognize some of the challenges that are now occurring as cutting-edge research involving neural devices and other treatments for epilepsy emerge. Dr Hendriks briefly addressed the importance of recognition of our own moral distress

Our patient representative Adam closed our speaker sessions out by providing a message of hope for patients with epilepsy. A live panel discussion cumulated the symposium. Adam, along with the speakers, answered questions from attendees.

Epilepsy Specialist Symposium

Controversies in Neocortical Epilepsies

Ignacio Valencia, MD, Jennifer L. Hopp, MD

The symposium addressed a broad range of topics related to the basic underpinnings of neocortical epilepsy and cortical networks and the evaluation of neocortical epilepsy including functional assessment, EEG evaluation with high density and stereo-EEG (SEEG), and surgical approaches and outcomes.

After a case presentation by Ignacio Valencia, MD, Ivan Soltesz, PhD, kicked off the symposium by discussing corticocortical networks in human and animal models of epilepsy. Key disease mechanisms at the cellular and microcircuit levels are not captured by routine EEG. He explained the development of new tools that now enable multiscale investigations to begin resolving controversies in the understanding of seizure networks. He described microelectrode arrays that capture the activity of hundreds of neurons, optical fiber arrays, cellular-scale imaging, macro scale imaging, and multisite fiber photometry.

In the second presentation on "Clinical Expression of Neocortical Epilepsies as a function of cortical networks: What have we learned from SEEG," Dr Aileen McGonigal described that the introduction of SEEG has allowed the study of 3-D brain networks. Early studies suggested that distant structures could simultaneously participate in the seizure generation. Several frontal, temporal, and insular studies with SEEG have shown the utility of SEEG. She presented some of her studies correlating semiology of seizures and epileptic networks as captured by SEEG[25] showing either increase or decrease network synchrony.

In the third lecture about assessing language and memory dominance, Dr Eugen Trinka compared different testing modalities including functional MRI (fMRI), MEG, and Wada. Memory decline can worsen quality of life after epilepsy surgery. Intracarotid sodium amytal test (Wada test) has been the gold standard to predict memory decline after surgery. This procedure carries an overall complication rate of 1% and permanent morbidity of around 0.4%. He reviewed a meta-analysis of different modalities to assess memory and language.[26] This showed that in the best case, the Wada test has a sensitivity estimate of 0.79 and a specificity of 0.65. Evidence for noninvasive tests to predict memory decline is limited.

Dr Mark D. Holmes reviewed high-density scalp EEG in the localization of neocortical epileptogenic foci. Surface high-density EEG can assist in reducing localization error rates as more spatial information is extracted. It can be combined with 3-D localization of the electrodes to improve accuracy. He explained that simultaneous high-density EEG and intracranial recording have shown good correlation suggesting high reliability. This technique may assist in planning placement of intracranial electrodes. Ictal source analysis and oscillations at seizure onset might be useful for localization of epileptogenic sites.[27]

Juan Bulacio, MD, discussed the use of SEEG in the localization of neocortical epileptogenic foci. He emphasized that the strategy for implantation should be based on a careful analysis of preimplantation noninvasive testing. He used several cases to demonstrate the concept that the focus should be on the sequence of semiology to identify seizure onset and spread. He noted that orthogonal placement allows simultaneous mesial and lateral sampling and suggested caution in using standard placement, favoring tailored SEEG implantation based on data analysis.[28]

The next lecture was a review of neocortical functional mapping of sites and networks by Nathan Crone, MD. He began with an overview of the terminology and then reviewed the process of electrocortical stimulation which is typically used to map sites and passive intracranial EEG which can be used to map networks.[29] He noted that although we don't have a comprehensive map of functional and epileptogenic networks for each patient, these current tools can guide surgical evaluation and expansion of their function may help to further understand the networks themselves.

Dr Bradley Lega reviewed surgical approaches for neocortical epilepsy with a focus on how to prevent damage of eloquent cortex. He noted that noninvasive methods can augment invasive mapping of motor areas and outlined examples of when this mapping might be done with the patient awake. He reviewed challenges in awake language mapping in the operating room (OR) including the assessment of receptive functions. Visual mapping may be relevant for patients with posterior temporal resections or heterotopias. He concluded

with a discussion of memory mapping and the need to improve episodic memory assessment in patients undergoing resective surgery.

The symposium concluded with a broad overview of outcomes after neocortical surgical resection by Jennifer Hopp, MD. She suggested caution when comparing definitions and outcome measures as they can vary regarding quality of life, seizure frequency, and length of postoperative seizure freedom. Dr Hopp reviewed key studies in patients undergoing neocortical epilepsy surgery, emphasizing that outcomes tend to be better for patients with temporal onset,[30] imaging positive or lesional epilepsy,[31] and those with resections not involving eloquent cortex.[32] She concluded with updates in outcomes in neuromodulation showing that it is also effective in this patient population.[33]

Hot Topics Symposium

Representation of Space and Time in the Medial Temporal Lobe: From Rodents to Humans

Douglas Coulter, PhD, Barbara Jobst, MD, and Itzhak Fried, MD, PhD

As a special event, we were honored to welcome Nobel laureate, Dr Edvard Moser to speak at AES 2020, highlighting the importance of research in epilepsy patients to complement memory research in animals. Drs Edvard Moser and May-Brit Moser received the Nobel Prize in 2014 for their work on grid cells. They shared the prize with John O'Keefe who had discovered place cells in the rodent hippocampus. Place cells encode specific locations in the environment by increased firing. Grid cells in the medial entorhinal cortex (MEC) have spatial firing fields with a periodic hexagonal structure and are a possible metric of the brain's map of space.[34] They coexist with head direction cells, speed cells, border cells, and object vector cells that fire specifically to their respective purposes. Topographical analysis of firing patterns using newer technologies that can record from thousands of neurons may provide further insight into the organization of neuronal firing patterns. While the joint activity of grid cells in MEC resides on a toroidal manifold and provides population code for the animal position across environments, the lateral entorhinal cortex appears to provide a time code.[35] The entorhinal network dynamics during coding of space, time, and memory may provide clues to cognitive dysfunction as noted in Alzheimer's disease (AD) and as a comorbidity of epilepsy.

Dr Fried spoke about these concepts in humans, describing studies with intracranial depth electrodes (stereo-EEG), which can allow for single neuron activity recordings in the human brain. Humans share with rodents hippocampal-entorhinal mechanisms of spatial memory including place cells, grid-like cells, theta rhythms, and phase precession.[36] Nonspatial cellular mechanisms of human memory encoding include concept cells that have a particular firing pattern to a defined concept.[37] Time may be encoded by cellular mechanisms in the entorhinal-hippocampal circuit. Studies analogous to animal studies using navigation as a memory surrogate have investigated intracranial oscillatory activity in ambulatory patients with a responsive neurostimulator which can record human oscillatory activity in a natural, real-world environment.[38] Theta burst stimulation of the human entorhinal cortex enhanced memory specificity and may open venues to improve memory with intracranial electrical stimulation.[39] Closed-loop electrical stimulation guided by hippocampal slow waves enhances consolidation and cortico-hippocampal dialogue during slow wave sleep.

Dr Kahana discussed the electrophysiology of episodic memory by reviewing the laws of memory: recency, primacy, contiguity, and similarity. He described univariate and multivariate biomarkers of variable mnemonic function. Multivariate decoders can predict memory success.[40] Electrical stimulation can either inhibit or enhance memory function depending on stimulation parameters and stimulus location and the functional state of the network.[41] Biomarker driven stimulation of cortical areas has been applied to enhance memory function.[42] Devices are being developed to enhance memory function not only in patients with epilepsy but also with traumatic brain injury.

The last presentation by Dr Lam explained the crossroads between AD and epilepsy. Epilepsy is a common feature of Alzheimer's and can arise early in the disease course. Silent hippocampal seizures may go undetected in Alzheimer's patients without visible signs on EEG.[43] Epileptiform activity in animal models has been shown to contribute to cognitive deficits in AD and there is PET data in humans that demonstrates a link between epileptiform activity and increased amyloid and tau deposition in AD.

Merritt Putnam Symposium

Neuromodulation for Seizure Control, Next Generation Strategies and Devices

Catherine Schevon, MD, PhD

The Merritt-Putnam symposium addressed next-generation neuromodulation for controlling intractable focal seizures. Esther Krook-Magnusen discussed her work on closed-loop, on-demand optogenetic stimulation to test the effects of cell-type specific neuronal populations in different subcortical and cortical brain locations. Her experiments revealed that excitation, but not inhibition, of the cerebellar fastigial nucleus, a site remote from the seizure focus, powerfully inhibits seizures in a rodent model of temporal lobe epilepsy.[44,45]

Mohamed Koubeissi discussed recent studies of low frequency electrical stimulation. In animal models of limbic or neocortical seizures, electrical stimulation of a white matter tract connected to a seizure focus has been effective in ameliorating seizures.[46] This has been translated in part to a proof-of-principle human trial.[47] More recent data about the efficacy of low frequency electrical stimulation of the piriform cortex in focal epilepsy have been published.[48]

Current research by several speakers aims to utilize a variety of measures of physiological or brain activity to personalize therapeutic stimulation and optimize its efficacy. Esther Krook-Magnuson discussed Bayesian optimization as a closed-loop approach to identify optimal stimulation parameters. David Mogul presented his work on changes in multisite network dynamics in the brain as a seizure progresses. Effects of exogenous deep brain stimulation (DBS) on long-range coherence were associated with changes in seizure progression, with coherence patterns varying between patients. Perturbing these coherence relationships may therefore provide a route to altering the effect of DBS on seizures.

Both Gregory Worrell and Timothy Denison addressed design considerations for future epilepsy neuromodulation systems. While the first generation of neurostimulators drew largely from cardiac devices, the next generation of systems are being refined for the unique characteristics of epilepsy.[49] Gregory Worrell discussed integrating wearable sensors and off-the-body computing resources, such as smart phone and cloud resources. These additions open new vistas for dense bidirectional behavioral and physiological tracking.[50,51] These are part of an envisioned next-generation system for treating epilepsy and mood, sleep, and cognitive comorbidities. Timothy Denison discussed factoring circadian and multidien rhythms into control methods, developing objective measures of seizure burden, and pivoting toward algorithms that aim to prevent seizure onset rather than merely responding to them.[52] These strategies are enabled by the development of new digital platforms that catalyze community collaboration.

COVID Symposium

Epilepsy in the Times of COVID

Barbara C. Jobst, MD, R. Edward Hogan, MD

As the COVID-19 pandemic was the reason for the virtual AES2020 meeting, the influence of COVID on epilepsy care was included as a special symposium. Generally, acute neurological complications of COVID-19 disease are uncommon and are usually in the neurovascular domain with multiple cerebral infarcts. Seizures as a result of COVID can occur in more severe COVID, usually in critically ill patients admitted to the intensive care unit. In patients with preexisting epilepsy, an increased susceptibility to seizures is probable, related to systemic effects of COVID infection.[53] The largest barriers to epilepsy care during the pandemic relate to loss of employment and access to health care services.[54]

SARS-COV-2 directly invades the nervous system through peripheral nerves but most pathophysiologic abnormalities are related to indirect mechanisms such as hyperinflammation, hypercoagulation, disturbance in the brain-lung crosstalk, and imbalance of ACE2: angiotensin converting enzyme (ACE) ratio.[55] In the cerebrospinal fluid (CSF), the virus can only be detected in 1% to 2% of patients with active COVID.

Other neurologic complications such as meningitis or encephalitis can only be considered clearly COVID related if there is proof of intrathecal antibody production. COVID related acute demyelinating encephalomyelitis as well as acute inflammatory demyelinating neuropathy occurs about 6 weeks after infection.[56] Treatments are evolving but at the time of AES2020 there was no evidence for the benefits of hydroxychloroquine or HIV protease inhibitors.

There is no distinct EEG signature of COVID and the most frequent finding on EEG is diffuse slowing, reflecting encephalopathy.[57] The risk of electrographic seizures is low and mainly are seen in patients with a history of clinical seizures. Epileptiform changes on EEG are also rare.

The pandemic quickly made us specialists in telemedicine. Telehealth can provide access to health care during the pandemic and is well suited for epilepsy care.[58] Associated billing codes are evolving and dependent on current legislation.

Advanced Practice Provider Symposium

Epilepsy Diagnostics and Results Interpretation

Gigi Smith, PhD, RN, PNP, Kelly R. Conner, PhD, PA-C

This was the first annual symposium for advanced practice providers (APPs) organized by the APP Committee. Evaluating a person with epilepsy using appropriate diagnostics is an important first and ongoing step in the patient's treatment journey. This symposium shared evidence-based information regarding the thorough evaluation of persons with epilepsy based upon a comprehensive examination and history. Interpreting test results and how these results inform treatment was also addressed, including the use of and referral to specialists. Case studies were woven throughout the session to further illustrate important points.

Jonathan Edwards, MD, MBA, reviewed the physiology of EEG and general principles of how EEG works. He described the vital role that EEG and video-EEG monitoring play in the care of epilepsy patients as well as the limitations and pitfalls of EEG and video-EEG monitoring.[59] Critical points included that video-EEG is a powerful tool that assists in decision-making for treatment of epilepsy as well as for surgery evaluation. Limitations of VEEG evaluation include failure to capture the typical events, failure to record all typical events, and challenges assessing events with retained awareness.

Noriko Salamon, MD, reviewed when to utilize various imaging modalities in epilepsy diagnosis, and how to recognize the advantages of MEG, PET, and SPECT studies.[60] MRI protocols for structural evaluation that are best for certain diagnoses associated with epilepsy were presented. The use and limitations of magnetencephalgraphy (MEG) imaging and testing to localize epilepsy was discussed. Evaluating brain function using positron emission tomography (PET) and SPECT scans during or between seizures, including the sensitivity of the tests in both the ictal and inter-ictal assessment of temporal lobe epilepsy and extra temporal lobe epilepsy was reviewed. The new classification of Types I, II, and III focal cortical

dysplasia were shared. Dr Salamon emphasized her take-home point that using multimodality in imaging is important, as MRI provides the structural analysis, and the functional analysis is provided by PET, MEG, and SPECT.

Tiffany Cummings, PhD, reviewed the importance of preoperative neurocognitive testing as a standard of epilepsy care.[61] Preoperative neuropsychological evaluation can assist in lateralizing and localizing the seizure focus, predict the risk for postoperative cognitive impairment, establish a baseline from which to measure change, help predict seizure relief outcome, diagnose psychiatric disorders, and consider the potential impact on the patient's ability to cooperate with the epilepsy surgery process and post-operative adjustment.[62] She shared the advantages and considerations for WADA testing versus fMRI in evaluating language dominance in advance of surgery.[63]

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