ACNS Annual Meeting and Courses February 5-10, 2013 Miami Marriott Biscayne Bay Miami, Fla.

Speaker Abstracts

PRESIDENTIAL LECTURE

Continuous EEG Monitoring in the ICU: Defining a New Standard of Care

Susan T. Herman, MD

Continuous EEG monitoring (CEEG) in the intensive care unit (ICU) is an emerging technology for detection of secondary insults in patients with acute neurologic injuries. EEG can detect subclinical seizures after convulsive status epilepticus, traumatic brain injury, subarachnoid hemorrhage (SAH), and intracerebral hemorrhage, and identify delayed ischemic injury from vasospasm in patients with SAH. CEEG is expensive, labor-intensive, and requires continuously-available interpretation for optimal use. Despite the potential promise of CEEG in reducing neurologic morbidity, no prospective studies have yet demonstrated improvement in patient outcomes related solely to CEEG. In some cases, over-aggressive treatment of CEEG findings may result in iatrogenic complications. Artifactual or nonspecific abnormalities detected by CEEG may lead to more invasive testing, such as cerebral angiogram, which may increase patient risk. This lecture will give an overview of the development of this new technology, highlighting improvements in techniques which improve feasibility and decrease costs. The development of new guidelines for performance, standardized nomenclature, interpretation, and monitoring of ICU CEEG will be reviewed. Several ongoing studies utilizing CEEG to detect subclinical seizures and guide treatment with AEDs will be discussed. Finally, a model for optimal clinical use of ICU CEEG will be proposed, with discussion of how this can be validated by clinical effectiveness studies.

SYMPOSIUM: MEMORY & LANGUAGE INSIGHTS FROM INVASIVE AND NON-INVASIVE STUDIES Insights from Invasive Recording on the Neuronal Mechanisms of Declarative Memory Formation in the Human Medical Temporal Lobe Ueli Rutishauser. PhD

Neuroscience strives to understand how neuronal circuits enable behavior, such as learning from novel experiences. I will discuss insights on the basic mechanisms of memory obtained by observing the activity of single neurons in the temporal lobe. These discoveries were enabled by taking advantage of the rare opportunity for invasive neurophysiology in patients with drug resistant epilepsy that were implanted with depth electrodes in the amygdala and hippocampus.

We found that successful memory formation in humans is predicted by a tight coordination of spike timing with the local theta oscillation. More stereotyped spiking predicts better memory, as indicated by higher retrieval confidence reported by subjects. Further, we discovered a class of neurons that selectively respond only when a stimulus, such as a photograph or a face, is seen the very first time. These neurons signal stimulus novelty and exhibit rapid plasticity, a prerequisite for learning. The response of these neurons is predictive of whether subjects remember or forget a stimulus. Listening to a few such neurons allows a brain-machine interface to outperform the memory retrieval performance of subjects, suggesting that these neurons represent memory as such rather than decisions. These findings provide a link between memory and mechanisms of plasticity.

SYMPOSIUM: INTRAOPERATIVE NEUROPHSIOLOGIC MONITORING DURING FUNCTIONAL NEUROSURGERY The Decision Interface: Surgeon, Neurophysiologist, and making the best decisions during surgery Jeff E. Arle, MD, PhD

Intraoperative Neurophysiology or Monitoring (IONM) has become standard care in many surgical procedures, but the interaction between surgeon and neurophysiologist goes beyond simply performing the monitoring of neural function during a surgical case. There are important aspects of the interaction that allow the surgeon to make the best decisions even when information is sparse. Moreover, the relationship and the mutual understanding of what can be accomplished in the operative setting allows for the potential to develop new techniques, methods and therapies. Finally, a strong interaction between the IONM staff and the surgeon can result in the ability to troubleshoot devices that have already been implanted and need evaluation and potential repair, but beyond the means of the company representative or the simple replacement of the device. This talk gives examples and explores each of these important areas

in the IONM repertoire, including decisionmaking in the use of MER for DBS surgery, the development of a new method for safer and more accurate placement of paddle electrodes in spinal cord stimulation, and the formal assessments needed to examine lead or wire breakage in many different systems.

Intra-operative Methodology and Communication During Functional Neurosurgical Procedures: Placing Permanent Electrodes in the Brain

Jay L. Shils, PhD, D.ABNM, FASNM

Surgical treatment for movement disorders in the basal ganglia dates back to the 1930's when Meyers first described Campotomy. In the 1940's Spiegel and Wycis described the first human use of stereotactic surgery for treatment of psychiatric illnesses. From the 1930's through the 1960's neurophysiology played a small role in these procedures, but it was the work of Dr. Albe-Fessard in the 1960's that opened the door for intra-operative micro-electrode recording which has become a critical tool for functional localization during Deep Brain Stimulation (DBS) placement surgery at a large number of centers. Specifically, this type of monitoring falls into a category termed Interventional Neurophysiology by Dr. Marc Sindou. As in brain mapping the neurophysiologists is no longer the reporter of previous events, their information helps in guiding the surgeon's actions in real time. In other words, the information gained, interpreted, and analyzed by the neurophysiologist is used by the surgeon to plan the course of the procedure.

In order for the neurophysiologist to perform these actions they need to not only have a detailed knowledge of the anatomy, basic physiology, and equipment, they also need to be familiar with the affects of the disease on the physiology, and the affects of the particular operative technique on the physiology. For example the Internal Globus Pallidum (GPi) has different single unit firing characteristics when recording from a PD patient as compared to a dystonia patient as compared to a Tourette's patient; the activity level of the zona incerta varies with age in PD patients; minor vascular artifacts can affect the interpreted rate of firing in all areas.

As in all areas of intraoperative neurophysiology the neurophysiologist in part of the surgical team and how the neurophysiologist passes information is critical to the success of the procedure. What information and how that information is presented to the functional neurosurgeon is critical in how the functional neurosurgeon uses that information. The neurophysiologist's interpretive skills and how they impart that information affects how the surgeon uses that data in their decision process. IONM data interpretation may cause the surgeon; (1) to stop the surgeon (The physiology is not consistent with the particular target area); (2) modify their plan (need to perform more tracts then the team is used to); (3) revise something they have done (the optimal tract is the first tract). Any of these changes are potentially beneficial or detrimental to the patient. Thus, communication and trust are critical factors in the surgeon neurophysiologists relation in the operating room and understanding these are key element to beneficial patient outcome.

At present there are three primary targets for movement disorders surgery that the neurophysiologists needs to be familiar with and they are the Ventral Intermediate nucleus of the thalamus (VIM), the Internal Globus Pallidum (GPi) and the Sub-thalamic Nucleus (STN). Yet, there are research protocols involving many more structures for other disease states including: (1) depression; (2) OCD; (3) Epilepsy; (4) Weight; (5) Pain; and (6) functional restoration. This lecture will describe the role of the neurophysiologist in the operating room, the methodology that is used by the neurophysiologist to locate the appropriate targets, and physiological characteristics of the targets.

SYMPOSIUM: THE CHALLENGES OF NON-LESIONAL FOCAL EPILEPSY: WHAT CAN NEWER NON-INVASIVE NEUROPHYSIOLOGY TOOLS TELL US TODAY? High Frequency Oscillations and Infraslow Activity: Two Useful Tools in Seizure Onset Localization

Pradeep Modur, MD, MS

Seizure onset zone (SOZ) is usually defined by the earliest rhythmic conventional frequency activity (CFA: 1–70 Hz). In neocortical epilepsy using intracranial recordings, SOZ was defined by ictal HFOs (\geq 70 Hz) that evolved subsequently into slower frequency activity (HFO+) while those that didn't evolve (HFO-) were not considered part of the SOZ; HFO+ had smaller spatial distribution than CFA, and resection of HFO+ channels resulted in favorable seizure outcome (Modur et al., Epilepsia 2011). A subsequent study in the same patients extended the analysis to ictal baseline ("DC") shifts (IBS) and peri-ictal infraslow activity (ISA 0.02– 0.2 Hz) (Modur et al., J Clin Neurophysiol 2012). It was shown that the seizure onset defined by HFO+ preceded or followed IBS closely (<300 ms). IBS were negative or positive, ~1 mV, and 2–3 seconds long. Compared with CFA, HFO+ and IBS were spatially restricted and likely to be concordant. Peri-ictal ISA consisted of periodic or rhythmic (0.12– 0.16 Hz) patterns. Better seizure outcome tended to correlate with smaller SOZs and more complete resection of the HFO+ and IBS contacts. In conclusion, ictal HFOs and baseline shifts define smaller

and probably more accurate SOZs in neocortical epilepsy. Future studies should address automated analysis and noninvasive recordings.

SYMPOSIUM: CONTROVERSIAL TOPICS IN EMG

Anticoagulated Patients Need INR Before EMG and the Results Influence the Muscles Studied

Jasvinder Chawla, MD; Devon Rubin, MD

Needle EMG is a slightly invasive procedure that poses the potential risk of intramuscular bleeding and hematoma formation. Several reports in the literature describe hematomas that have developed from needle EMG. The risk of hematoma development following needle EMG is theoretically increased in patient more susceptible to bleeding, such as those on anticoagulants or anti-platelet medications. Several studies have attempted to assess the risk of hematoma development following needle EMG, in all patients and those on anticoagulants. In assessing paraspinal muscles with MRI techniques following needle EMG of the paraspinals, 5/17 patients were found to have subclinical hematomas. More recently, a review of 431 patients who had undergone needle EMG of paraspinals, 10 of whom were on warfarin and 138 on aspirin, did not detect hematoma in the paraspinals of any patient. Using ultrasound to examine the anterior tibialis and other high risk muscles 30 minutes following needle EMG in 101 patients on warfarin, only 2% of patients were found to have small, subclinical hematomas. No clinically significant hematomas were found. Therefore, given the current evidence, in patient on stable doses of anticoagulation who are monitored regularly by their physician, an INR is not needed immediately prior to needle EMG.

All Patients Undergoing Nerve Conduction Studies Must Have a Needle Exam

Arturo Leis, MD; Mark A. Ross, MD

EMG and nerve conduction studies (NCS) are used to test the integrity of the peripheral nervous system (PNS). From a neuroanatomical perspective, injury or damage to five different levels gives rise to the common neuromuscular disorders: 1) **muscle** (myopathies), 2) **neuromuscular junction** (myasthenia gravis, MG; Lambert-Eaton myasthenic syndrome, LEMS; other defects in neuromuscular transmission), 3) **peripheral nerves** (mononeuropathy, plexopathy, polyneuropathy), 4) **roots** (radiculopathy), and 5) **anterior horn cell** (motor neuron disease, poliomyelitis).

In **muscle** disorders, needle EMG is essential to identify the characteristic "myopathic" recruitment (excessive number of small MUPs are recruited relative to the force of contraction), which is the hallmark of myopathic disorders. In **neuromuscular junction** disorders, single fiber EMG (increased jitter) remains the most sensitive test for MG (sensitivity > 95% positive in generalized and ocular MG when facial muscles examined). In **root lesions**, EMG is crucial to delineate the distribution of affected muscles, to localize the root(s) involved, and to provide information about the severity and chronicity of the radiculopathy. In **anterior horn cell** disorders, EMG is indispensable to document progression (early stages show localized denervation, "neurogenic" recruitment, and fasciculation potentials, while later stages show widespread denervation and chronic neuropathic changes involving multiple limbs or tongue). In most **peripheral nerve** disorders, EMG is also a essential diagnostic tool. In diffuse polyneuropathy, denervation or chronic neuropathic changes are often limited to distal muscles (in conjunction with distal weakness and stocking-glove distribution sensory loss). In plexopathy, EMG abnormalities demarcate the distribution of affected muscles and confirm the presence of axonal loss.

In the above conditions, EMG is generally acknowledged to be a necessary component of the electrodiagnostic evaluation. In contrast, there has been a longstanding controversy regarding the role of EMG in entrapment neuropathies, particularly carpal tunnel syndrome (CTS). The justification for this debate arises from the fact that CMAP amplitude is a predictor of axonal degeneration. Hence, if the degree of axonal loss is mild, then CMAPs are only minimally affected; with more severe axonal loss, amplitudes are reduced or absent and prognosis for recovery of function is less favorable. However, the argument that EMG can be restricted to certain CTS patients whose CMAP amplitude lies within a certain range or that EMG should not be performed when CMAP is absent is tenuous. An absent CMAP is *not* synonymous with axonotmesis, and evidence of preserved innervation can often be found only by performing the needle exam. Similarly, a relatively preserved CMAP does *not* exclude axonal degeneration, and evidence of axonal loss (which alters prognosis) can sometimes only be found by performing EMG. EMG in cases of low CMAPs in CTS would also be important in evaluating patients who fail to improve after carpal tunnel release. Without a pre-op EMG, there is no way to determine if there was preserved innervation prior to surgery or to compare pre-and-post-operative denervation and recruitment pattern. From my own experience, I have often regretted not performing a pre-op needle EMG when CMAP was absent or markedly reduced and patients failed to improve. Moreover, if the decision tree is whether surgery is indicated, any discomfort from a needle EMG is minor.

There has also been controversy regarding the role of needle EMG in acute traumatic nerve injury. This debate arises from the fact that there is a latent period of 2 to 3 weeks after nerve injury until Wallerian degeneration has fully manifested and spontaneous activity is evident. Hence, the absence of spontaneous activity does not rule out denervation. This delay in the development of spontaneous activity has given rise to a common myth that the EMG must be postponed 2 to 3 weeks following nerve injury before reliable information can be obtained. In fact, demonstrating preserved conduction in nerves of an injured limb may be of paramount importance immediately or early after injury, since it can help to identify preserved nerves from damaged nerves. If there is substantial axonal or neurapraxic injury, the pattern of recruitment of MUPs will immediately become abnormal, allowing an experienced electromyographer to approximate the degree of nerve injury and to determine future management. NCS across an injured segment of nerve will also be abnormal immediately after injury. However, the limitation of EMG and NCS performed early after injury is that one cannot differentiate severe neurapraxic injury with complete conduction block from axonotmesis associated with severe axonal loss or nerve transection

Those who would argue to forego the needle exam should also be aware that their recommendation may have an unintended consequence of encouraging the wave of poorly trained physicians or technicians who routinely perform NCS in the absence of EMG.

SYMPOSIUM: CHALLENGES IN INTRAOPERATIVE NEUROPHYSIOLOGIC MAPPING ALONG THE NEUROAXIS Supratentorial Mapping

Mirela V. Simon, MD; Eva K. Ritzl, MD

We will present a case of neurophysiologic mapping of cortical and subcortical motor structures, followed by continuous monitoring of these structures during supratentorial resection. Several troubleshooting tips for central sulcus localization using the SSEPs phase reversal technique will be presented. The technique of direct cortical stimulation via contacts of a subdural strip electrode will be detailed, as well as the instances in which its use can be most beneficial. During continuous motor monitoring, tips for interpretation of fluctuations in the amplitudes of motor evoked responses will be emphasized.

SYMPOSIUM: BRAIN STIMULATION

Brain Stimulation for Epilepsy

Robert S. Fisher, MD, PhD

Electrical stimulation for epilepsy has been advocated since the 1950's. In the US, only VNS has been approved, but DBS is now approved in Europe, several Asian and South American countries and Canada. The anti-seizure mechanism of DBS is unknown, but it likely disrupts synchronous networks during seizures. Numerous structures in brain have been stimulated, most effectively including: the anterior nucleus of thalamus (ANT) by time-cycling stimulation, the seizure focus by responsive neurostimulation, centromedian thalamus, and hippocampus. The first two of these now have Class I evidence from randomized placebo-controlled trials. ANT stimulation reduces seizures by 41% of baseline by the end of a 3-month blinded trial and by 66% after 4 years. Most severe seizures and injuries from seizures were reduced. Complications include a few cases of reversible triggered seizures or status, radiologically apparent blood around the electrode, peripheral infections, and possible increased depression and memory symptoms. Responsive neurostimulation reduced disabling seizures by 29% in the blinded phase, with continued improvement over time. Mood and memory did not seem adversely affected. How to select the best candidates, the best stimulation methods remain open questions.

Transcranial Magnetic Stimulation of the Motor System

Mark Hallett, MD, Human Motor Control Section, NINDS, NIH, Bethesda

Transcranial magnetic stimulation (TMS) has now been around for about two decades. It has been shown to be useful for limited purposes in the clinic, particularly in regard to central motor conduction velocity. In relation to therapeutics, while it is now an approved therapy for the treatment of depression, it has not had any major successes in movement disorders. TMS has been a particularly fruitful technique for physiological investigations of the motor system (Hallett, 2007). TMS can examine many facets of cortical excitability and transiently activate or inhibit the motor system. TMS methods can also be used to assess and manipulate cortical plasticity. These methods will be illustrated by examining studies of patients with focal hand dystonia. Studies of motor excitability show a decrease of surround inhibition, a failure of inhibition of muscles not intended to be moved. Studies of brain excitability show deranged motor cortex plasticity, both an exaggerated response and a failure of homeostatic plasticity.

PLENARY LECTURE Small-world networks: The Clinical Neurophysiology of communication in the brain

C. J. Stam, MD, PhD

In Clinical Neurophysiology and neuroimaging there is an increasing interest to study the brain from a complex networks perspective.1.2 The central question is: how do the different and widely distributed components that make up our complex brain networks communicate and how does this communication break down in disease? This shift towards a network paradigm of brain studies has been stimulated by advances in neurophysiology, in particular the discovery of synchronous oscillations underlying information processing, memory and awareness, and by rapid progress in modern imaging techniques such as high density EEG, MEG, structural and functional MRI. Another important breakthrough has been brought about by the merging of the modern science of networks (based upon graph theory) and neuroscience. We now know that nervous systems in animals and humans are characterized by a combination of high clustering (local integration) and short path lengths (high integration). This combination of local clustering and global integration is characteristic of so called "small-world" networks. In addition, complex brain networks have other important organizational features such as scale-free degree distributions, the presence of a connectivity backbone with highly connected hubs, and a hierarchical, modular structure. This organization of brain networks emerges during development, is under strong genetic control, and is strongly correlated with cognitive performance. Importantly, brain network organization has been shown to be important for understanding the mechanisms of neuropsychiatric disease. Disorders such as Alzheimer's disease, epilepsy and schizophrenia may represent different scenario's characterized by loss of small-world features, hub failure or disorganization of modular structure. A heuristic model of the brain as a hierarchical modular complex network can help to integrate these findings in a single comprehensive framework.

References:

1 Stam CJ. Characterization of anatomical and functional connectivity in the brain: a complex networks perspective. Int J Psychophysiol. 2010 Sep;77(3):186-94.

2 Stam CJ, van Straaten EC. The organization of physiological brain networks. Clin Neurophysiol. 2012 Jun;123(6):1067-87.

SYMPOSIUM: NEW DIRECTIONS FOR QUANTITATIVE EEG IN NEUROCRITIAL CARE

EEG Monitoring and Closed-Loop Control of Burst Suppression

M. Brandon Westover, MD, PhD; ShiNung Ching, MD

Medical coma is an anesthetic-induced state of profound brain inactivation used to treat status epilepticus and to facilitate recovery following traumatic and hypoxic brain injuries. Under current practice burst suppression is maintained by intensive care unit staff continually monitoring the level of burst suppression on the electroencephalogram (EEG) and manually titrating the anesthetic infusion rate to target a specific level. However, such open-loop control is labor intensive and prone to over- and under-shooting. An automatic, closed-loop control is desirable to overcome these limitations.

In this session we will first review the physiology of normal and pathological burst suppression; the rationale for burst suppression as a medical therapeutic intervention; and how to depth of burst suppression can be quantified. Finally, we discuss the theory of closed-loop feedback control in relation to burst suppression, and describe the necessary components (e.g. signal processing and pharmacokinetics and pharmacodynamics models) and practical challenges involved in building a working system.

We then present a closed loop anesthesia delivery (CLAD) system recently developed by our group to control medical coma, and describe results obtained so far in testing the system in rodents. The system uses a computer-controlled infusion of propofol to maintain a specified target depth of burst suppression. The system performs automatic binary segmentation of the EEG into discreet suppressed vs "burst" segments. At run time, our system quantifies the initial effects propofol on the individual's EEG, in order to estimation the subject's pharmacokinetic and pharmacodynamic parameters. These parameters are then used to determine the system's feedback control gains. We introduce the burst suppression probability (BSP)filter algorithm and the metabolic state probability filters (MSP) to compute in real time from the EEG the instantaneous depth of cerebral metabolic suppression.

We present results from tests our CLAD system in controlling burst suppression in a rodent model of medical coma. In each of 6 animals we demonstrate control at the individual level, by maintaining the BSP in steady state for 15 minutes at each of 3 different levels target levels, spanning the range targeted in ICU care of neurological patients. We show that burst suppression can be precisely and quantitatively monitored in real-time, and tightly controlled in individual animals.

Our findings establish the feasibility of using a CLAD system to control medically-induced coma in rodents and suggests that the paradigm of burst suppression control could be used to maintain medically-induced coma in patients.

WORKSHOP: EEG-VIDEO: EXPERT CONSENSUS

EEG-Video: Expert Consensus

William O. Tatum IV, DO; Johnathan J. Halford, MD; Selim R. Benbadis, MD; Jonathan C. Edwards, MD; Peter W. Kaplan, MB, FRCP

This 2-part Workshop will be composed of computer-based consensus and expert-based consensus of common EEG and video examples that are challenging to neurologists.

Part 1—Computer-based expert consensus in EEG

Web-based software viewing "suspicious" routine scalp EEG will be displayed.

The audience will be polled with the ARS.

Consensus by a group of expert readers will be compared with the results of the audience and input from the moderator will "finalize" the challenge posed by the sample presented.

Part 2—EEG & video individual expert v group analysis

Difficult to interpret paroxysmal EEG potentials will be displayed to highlight the interictal and ictal EEG features of patients with and without epilepsy.

Audience response will identify the events as epileptiform, normal physiologic, abnormal physiologic or artifact. The faculty will make their determination prior to the clinical "answer".

Videos will then be presented to the expert panel composed of 3 individuals. Each will sequentially discuss their opinion as to whether the event is epileptic, physiologic, or psychogenic. Subsequently the audience will be polled with ARS. Individual v group consensus will then be highlighted with respect to the final clinical diagnosis.

SPECIAL INTEREST GROUP: MODELS OF PROFESSIONAL CARE IN IOM: A COLLEGICAL DEBATE Models of Professional Care in IOM: A Collegial Debate

Stanlev Skinner. MD

Two major IONM models ("remote monitoring" and "nearby/available ... in house") currently account for the vast majority of professionally supervised IONM cases in the United States. These models do differ in important ways. For instance, in the "nearby/available" model, the IONM physician/professional may electively engage in personal pre-operative patient assessment, may personally go to the operating room if requested, and commonly enjoys a mentoring relationship with the supervised, in-room technologist (direct supervision) (2). Remote and nearby/available monitoring utilizes waveform telemetry over a secured web-based or intranet connection (3, 4). Both models usually rely on a phone-based connection between the IONM physician/professional and the other operating room practitioners. But neither model presumes the close collegial relationships with the surgeon/proceduralist or the anesthesiologist that naturally develop when the IONM physician/professional is routinely physically present in the operating room.

SYMPOSIUM: CONTINUOUS EEG MONITORING IN NEONATES: THE NEW ACNS GUIDELINES Indications for Long-Term EEG Monitoring in Neonates Robert R. Clancy, MD

The ACNS Guideline on Continuous EEG Monitoring in Neonates synthesizes evidence to provide recommendations on appropriate indications for long term EEG monitoring (LTM). The broad purposes of conventional long term EEG monitoring in neonates are (i) "seizure-centric" and (ii) prognosis oriented. Newborns have an intrinsically high susceptibility to seizures and are commonly exposed to adverse clinical scenarios (such as hypoxia-ischemia) that are conducive to provoke seizures. The clinical diagnosis of seizures in the newborn is now well known to be fraught with unacceptably high errors of under- and over-diagnosis. The first "seizure-centric" goal of neonatal LTM is to confirm or refute the epileptic basis of abnormal appearing, paroxysmal motor, behavioral or autonomic "spells". If the basis of these attacks is confirmed to be electrographic seizures, then LTM is continued to accurately measure seizure burden, to gauge their response to anti-seizure medications and even to check for seizure relapse if anti-seizure medications are weaned.

Some clinical scenarios inherently carry a high risk of EEG seizures or status epilepticus, even if the patient has not demonstrated any outward clinical signs of seizures. Examples include acute neonatal encephalopathy (including hypoxic ischemic encephalopathy), stroke, sinovenous thrombosis, newborn heart surgery, ECMO, birth trauma and many others scenarios. Relevant literature illustrates the high yield for seizure detection by LTM in two specific circumstances: newborn heart surgery and ECMO.

The other broad goal of neonatal LTM is to help formulate a neurologic prognosis in serious acute conditions such as acute neonatal encephalopathy and in chronic conditions such as extreme prematurity. These "spectrum" disorders carry an inherently high risk for death or permanent neurologic disability and thoughtful judgment of the EEG background is a major component in the accurate prediction of outcome.

Methods and Reporting in Neonatal EEG Monitoring

Renee A. Shellhaas, MD

The new ACNS guidelines on neonatal EEG monitoring and neonatal EEG terminology provide suggested methods and reporting standards. Ideal methods for neonatal EEG monitoring include specific use of neonatal EEG montages, with several extracerebral channels. The appropriate duration of recording varies according to the indication for monitoring. Notably the ACNS guideline emphasizes that a routine length neonatal EEG is insufficient to screen for seizures. For high-risk neonates, 24-hour video EEG monitoring is considered the gold standard for seizure detection. Many neonatal intensive care units incorporate amplitude-integrated EEG monitoring, especially for characterization of evolving background patterns. In this presentation, we will discuss ideal methods for neonatal EEG monitoring, including montages and duration of recording. Suggested approaches for incorporation of digital trending techniques, especially amplitude-integrated EEG, will be reviewed. Finally, standards for frequency of record review and reporting will be discussed.

Standardizing Neonatal EEG Background Terminology

Tammy Tsuchida, MD, PhD

Neonatal EEG has a long history of utility for predicting clinical outcomes in neonates with hypoxia-ischemia or seizures. It is particularly important in the current era of hypothermia as it is one of the early markers of long term outcome after hypoxia-ischemia. The prognostic accuracy varies depending upon how a particular pattern is defined and when the EEG is obtained relative to the period of hypoxia-ischemia. Standardizing background terminology not only allows for multicenter collaboration but also may improve the ability to prognosticate based on different background patterns. This talk will present commonly used background patterns from the ACNS Standardized EEG Terminology for Continuous Monitoring in Neonates. Background patterns that can be difficult to categorize will also be discussed.

Seizure or Not? Categorizing Rhythmic Patterns

Courtney Wusthoff, MD

Clinical neurophysiologists face myriad abnormal patterns as they review continuous EEG recordings from ill neonates. While some patterns are easily distinguished as pathologic or seizures, there exists a daunting spectrum of rhythmic patterns that are not so straightforward. The new ACNS guideline tackles this grey area. An approach to neonatal EEG interpretation, including standardized terminology, is presented to facilitate systematic analysis of difficult rhythmic patterns. Clear criteria are offered to distinguish seizures from other rhythmic discharges. Consistent descriptors are suggested to allow better description of seizures and rhythmic patterns. Finally, a uniform definition of status epilepticus is proposed. Application of this framework to difficult tracings will illustrate its

usefulness for the clinical neurophysiologist. Overall, the ACNS guideline on neonatal EEG offers benefits in clear communication between physicians, a structured system for teaching and training, and is of high utility for research applications.

SYMPOSIUM: ENCEPHALOPATHIES: ELECTOPHYSIOLOGIC, CLINICAL AND IMAGING CORRELATIONS Ictal and Epileptiform Elements in Encephalopathy

Frank W. Drislane, MD

Encephalopathies involve widespread brain dysfunction, especially cortical. The EEG usually shows widespread slowing and often, lower voltage activity. Some encephalopathies also exhibit the abnormal cortical activity of epileptiform discharges and even seizures. Seizures involve excitatory rhythmic electrical activity and also cause cortical dysfunction and clinical deficits; they may also lead to a postictal encephalopathy. Clinically, seizures and encephalopathies can each cause impaired responsiveness or cognitive and behavioral dysfunction, but their physiologic mechanisms of causing neurologic dysfunction appear to be different. Despite their usually-contrasting natures, however, encephalopathies and seizures sometimes occur in the same patients, particularly in developmental illnesses, e.g. in the "epileptic encephalopathies." Anoxic encephalopathies damage cortical neurons severely, often leaving them capable of forming only brief bursts of sharp waves on the EEG, but occasionally epileptiform activity is so pronounced and prolonged as to constitute seizures. Even in metabolic encephalopathies, sharp features appear on the EEG, and their significance is often difficult to determine -- and controversial. Periodic epileptiform discharges are found in many encephalopathies. They do not always indicate the presence of seizures, but sometimes they do, and distinguishing the difference can be challenging. Generalized periodic epileptiform discharges (GPEDs), for example, can be particularly vexing [one case will be shown for illustration]. EEG findings are tremendously informative, but they are not always sufficient for a final diagnosis, or for making plans for treatment and management. Frequently, the clinical situation must be incorporated into the decision-making; the same epileptiform activity on the EEG may have a different significance in different clinical settings.

Imaging Correlations and EEG Patterns in Encephalopathy

Peter W. Kaplan, MD, FRCP

Encephalopathy, a diffuse dysfunction of higher cortical function, is frequently encountered in hospitalized patients particularly in intensive care units. It has been associated with adverse outcome. The EEG generally reveals a non-epileptiform slowing of background activity with or without presence of triphasic waves (TWs) or frontal intermittent rhythmic delta activity (FIRDA). The patterns are believed to reflect underlying toxic, infectious or metabolic problems, but with limited evidence linking specific EEG abnormalities imaging findings and purported risk factors.

In cats, Gloor noted specific EEG patterns with anatomical lesions restricted to the cortex, to undercutting the cortex in the white matter, and in both [1]. Slow EEG background activity without slow activity in the delta range is seen with cortical problems that spare subcortical structures. White matter abnormalities may associate with TWs, while cortical/subcortical problems may produce combined background slowing and slow wave activity.

We review the EEG patterns seen with different clinical characteristics and imaging abnormalities, and present data in 154 patients with altered mental status classified into five predefined patterns: isolated continuous slowing of background activity (theta, theta/delta, and delta activity) and patterns with slowing of the background activity with episodic TWs or FIRDA. In multivariable analyses, *theta* was associated with *brain atrophy* (OR 2.6, p = 0.020), *theta/delta* with *intracerebral hemorrhages* (OR 6.8, p = 0.005), *FIRDA* with *past cerebrovascular accidents* (OR 2.7, p = 0.004), *TWs* with *liver or multi-organ failure* (OR 6, p = 0.004; OR 4, p = 0.039), and *delta* activity with *alcohol/drug abuse with or without intoxication, and HIV infection* (OR 3.8, p = 0.003; OR 9, p = 0.004). TWs were associated with death (OR 4.5, p = 0.005); theta/delta with unfavorable outcomes (OR 2.5, p = 0.033), while patients with FIRDA had favorable outcomes (OR 4.8, p = 0.004)[2].

In encephalopathy, EEG patterns are associated with particular pathological conditions and outcomes, suggesting that mechanistic hypotheses underlie these specific EEG patterns.

- 1) Gloor P, Ball G, Schaul N (1977) Brain lesions that produce delta waves in the EEG. Neurology 27;326-333.
- 2) Sutter R, Stevens RD, Kaplan PW. Clinical and imaging correlates of EEG patterns in hospitalized patients with encephalopathy. J Neurol 2012 Epub Nov 30.

The Current State of Safety in the EMU

Joseph F. Drazkowski, MD; Katherine Noe, MD, PhD; Lisa Bateman, MD,

Significant and meaningful advances in EEG related technology have allowed for the proliferation of EMUs in the last decade. The EMU environment is associated with unique risks and challenges to providers, family members and ultimately patients. Along with the growth and utilization of such services, questions arise about the efficiency and safety associated with EMU admissions. Classification of spells and pre-surgical epilepsy evaluations typically require reduction or discontinuation of anti-seizure drugs to provoke events. Discontinuing medications likely carries a relative increased risk to the patient. Practices used to provoke seizures have been generally accepted as standard of care in the EMU community. Until recently, limited studies concerning safety in the EMU population have been available. Safety procedures utilized in the EMU has been largely determined by individual epilepsy centers. What defines best practices, acceptable risks and effective safety procedures is evolving. A recently convened expert opinion panel on EMU safety endeavored to provide guidance to EMU practitioners. The panel's conclusions and the evidence utilized in the process will be reviewed. Representative video-EEG cases will be used for teaching points and audience interaction. The formal certification process of individual EMUs is in development; safety standards will likely comprise a significant part of this process.