2014 Annual Meeting & Courses

FEBRUARY 4-9, 2014

ATLANTA, GA

Westin Peachtree Plaza

www.acns.org
### Annual Courses Overview

**Tuesday, February 4, 2014**
- 9:00 AM – 5:00 PM: Intraoperative Monitoring Part I  
  International DE, 6th floor

**Wednesday, February 5, 2014**
- 7:00 – 8:30 AM: EP Reading Session  
  International DE, 6th floor
- 9:00 AM – 5:00 PM: Intraoperative EEG  
  International C, 6th floor
- 9:00 AM – 5:00 PM: Intraoperative Monitoring Part II  
  International DE, 6th floor

**Thursday, February 6, 2014**
- 7:00 – 8:30 AM: EMG and EEG Technology  
  International B, 6th floor
- 9:00 AM – 12:00 PM: ICU EEG  
  International DE, 6th floor
- 9:00 AM – 5:00 PM: Intraoperative EEG  
  International C, 6th floor
- 1:00 - 2:30 PM: Automatic Neurophysiology: Spells of Dysautonomia: From Epilepsy to West Nile  
  International B, 6th floor
- 2:30 – 5:00 PM: Case Studies in Peripheral Neurophysiology  
  International B, 6th floor

### Annual Meeting Overview

**Friday, February 7, 2014**
- 7:00 – 8:00 AM: Poster Viewing & Continental Breakfast  
  200 Peachtree, 7th floor
- 7:55 – 10:00 AM: Opening Ceremony and Plenary Session  
  Atlanta Ballroom, 7th floor
- 10:00 – 10:30 AM: Coffee Break - Visit Exhibits and Posters  
  200 Peachtree, 7th floor
- 10:30 AM – 12:30 PM: Wide Bandwidth Electrophysiology and Epilepsy Biomarkers  
  Atlanta Ballroom, 7th floor
- 12:30 – 1:30 PM: Lunch - Visit Exhibits and Posters - Poster Tour  
  200 Peachtree, 7th floor
- 1:30 – 3:30 PM: Ischemia Monitoring in Critical Care: EEG Trend Analysis to Detect Development of and Recovery From Cerebral Ischemia  
  Atlanta Ballroom, 7th floor
- 3:30 – 4:00 PM: Botulinum Toxic: Mechanism of Action and Ultrasound versus EMG Guidance  
  Augusto Balloon, 7th floor
- 4:00 – 5:00 PM: The Creation of Evidence Based Medicine in Intraoperative Monitoring  
  International DE, 6th floor
- 5:30 – 6:45 PM: Neurophys Bowl  
  Atlanta Ballroom, 7th floor
- 6:45 – 8:00 PM: Welcome Reception  
  200 Peachtree, 7th floor

**Saturday, February 8, 2014**
- 7:00 – 8:00 AM: Poster Viewing & Continental Breakfast  
  200 Peachtree, 7th floor
- 8:00 – 10:00 AM: Plenary Session  
  Atlanta Ballroom, 7th floor
- 10:00 – 10:30 AM: Coffee Break - Visit Exhibits and Posters  
  200 Peachtree, 7th floor
- 10:30 AM – 12:30 PM: EEG as a Basic Neuroscience and Psychology Research Tool  
  Atlanta Ballroom, 7th floor
- 12:30 – 2:00 PM: Lunch - Visit Exhibits and Posters and Poster Tour  
  200 Peachtree, 7th floor
- 12:50 – 1:50 PM: Professional Development Mentoring Program  
  International B, 6th floor
- 1:00 – 4:00 PM: Extending Critical Care EEG Monitoring to Community-Based Practice  
  Atlanta Ballroom, 7th floor
- 2:00 – 4:00 PM: Electrophysiological Approach to Neuromuscular Disorders  
  Atlanta Ballroom, 7th floor
- 4:00 – 4:30 PM: Coffee Break - Visit Posters  
  Atlanta Ballroom Foyer
- 4:30 – 6:00 PM: EMG  
  Augusto Ballroom, 7th floor
- 6:00 – 6:30 PM: Annual Business Meeting  
  Atlanta Ballroom, 7th floor

**Sunday, February 9, 2014**
- 7:15 – 8:00 AM: Continental Breakfast  
  Atlanta Ballroom Foyer
- 8:00 – 10:00 AM: Stereo Electroencephalography  
  Atlanta Ballroom, 7th floor
- 10:00 – 10:15 AM: Coffee Break  
  Atlanta Ballroom Foyer
- 10:15 AM – 12:15 PM: Neonatal and Pediatric EEG: Patterns of Epileptic Encephalopathies across the Age Range  
  International D, 6th floor
- 10:00 – 10:15 AM: Critical Care EEG Monitoring and Outcomes: Do We Have Enough Data?  
  International D, 6th floor
- 6:00 – 6:30 PM: Annual Business Meeting  
  Atlanta Ballroom, 7th floor
# Table of Contents

President’s Message ................................................................. 2  
Message from Program and Course Committee Co-Chairs .................. 2  

## ACNS Information

- Officers & Council 2013–14 ................................................. 3  
- Past Presidents ........................................................................ 4  
- Annual Meeting & Courses Committee Members ...................... 3  

## General Meeting Information

- Registration Desk ...................................................................... 5  
- Internet .................................................................................... 5  
- Certificate of Attendance & CME Certificates ......................... 5  
- Business Meeting ..................................................................... 5  
- Poster Sessions ........................................................................ 5  
- Publication of Abstracts ............................................................ 5  
- Venue Information .................................................................... 6  
- Westin Peachtree Plaza Floorplan ........................................... 6  
- Nearby Restaurants .................................................................. 7  

## CME Information

- Conflict of Interest Disclosures .............................................. 10  
- Award Recipients & Lectures .................................................. 15  
- Networking & Social Events .................................................... 15  
- Annual Courses Program ...................................................... 16  
- Annual Meeting Program ....................................................... 21  
- Speaker Abstracts .................................................................... 30  
- Poster Abstracts ....................................................................... 49  

## Exhibit Hall & Evening Programs

- Exhibit Hours .......................................................................... 69  
- Exhibit Hall Floorplan ............................................................. 69  
- Exhibitors ................................................................................. 69  
- Evening Programs .................................................................... 75  

---

**Executive Office**  
555 East Wells Street  
Suite 1100  
Milwaukee, WI 53202  
Phone: (414) 918-9803  
Fax: (414) 276-3349  
info@acns.org  
www.acns.org  

**Executive Director**  
Megan M. Kelley, CMP  
mkelley@acns.org
MESSAGE FROM THE PRESIDENT

Welcome to Atlanta and to the 68th Annual Meeting of the American Clinical Neurophysiology Society (ACNS).

The Annual Courses, expertly fashioned by Drs. Bill Tatum and Tobi Loddenkemper, will have commenced on Tuesday, February 4 and, as always, should be the best courses one can find on the latest in clinical methods in both ICU and Intraoperative Monitoring; peripheral nerve studies; EEG, whether neonatal or invasive (or both); evoked potentials; Autonomic Neurophysiology; and in Technology and other practical, business updates.

The Annual Meeting itself begins early Friday, February 7, and continues through Sunday, February 9 at noon. The Program Committee, led by Drs. Suzette La Roche and Gregory Worrell, has assembled an impressive array of lectures and symposia on the latest innovations and developments in all forms of Clinical Neurophysiology, as explicated by many of the world’s leading investigators and teachers. The roster of remarkably diverse topics underscores how rapidly our field is expanding. There should be something (indeed, many things) for everyone with a strong interest in Clinical Neurophysiology.

The ACNS Council and I want to extend a particularly hearty welcome to our international attendees and also to Neurophysiology fellows and others new to the meeting and the field. We believe strongly that you will have the opportunity to learn a great deal and to meet some leading clinical neurophysiologists in a small-group setting (sometimes, two people) to discuss very interesting insights into the function of the human nervous system. On a personal note, I’ve met some of my favorite colleagues at the ACNS meeting. We hope you enjoy the gathering.

Frank W. Drislane, MD
President

MESSAGE FROM COURSE AND PROGRAM COMMITTEE CO-CHAIRS

Dear Colleagues,

On behalf of the American Clinical Neurophysiology Society (ACNS), we are thrilled to welcome you to the 2014 Annual Meeting & Courses.

The Course Committee has again placed a priority on increased audience interaction and course directors have developed creative ways to engage with attendees. We hope you enjoy the Courses and provide feedback.

We are also pleased to announce an addition of non-CME evening programs to the Course schedule. During the evening hours, delegates will have the opportunity to explore in depth the technologies and products available to assist them in developing state of the art neurophysiology programs. Companies will have the opportunity to extend these conversations beyond the exhibit hall and to interact with delegates in a more hands-on environment. We urge you to be a part of it. See page XX for more information.

The Program Committee received a large number of impressive session proposals and it was truly very difficult to choose among them. We’re confident that those that were chosen will make up an outstanding Scientific Program so we encourage you to review the schedule of symposia, workshops, and special interest groups.

To kick-off each day, we’ve planned a number of plenary sessions, featuring our very own President, Frank W. Drislane, MD; David Burke, MD, DSc; and Rodolfo Llinas, MD, PhD. In addition to the plenary talks, we look forward to presenting the 2014 Pierre Gloor Award to Ronald Emerson, MD and the Herbert H. Jasper Award to Ernst Rodin, MD.

Of course, no ACNS Annual Meeting would be complete without the excitement of the “Neurophys Bowl,” the educational interactive-game. The Annual Neurophys Bowl is a mainstay of the Annual Meeting program, allowing members and delegates a chance to challenge their knowledge of clinical neurophysiology in a competitive format. Don’t forget to sign up your team and have some fun!

The ACNS Annual Meeting & Courses provides an ideal opportunity to learn, network and socialize with your colleagues, while also having the chance to see the latest equipment on display in the Exhibit Hall. We are certain that these opportunities, diverse course offerings, symposia, hands-on workshops, and special interest groups will ensure that whether your focus is in central or peripheral neurophysiology you will find much of interest and utility to your practice at this year’s ACNS Annual Meeting & Courses.

Welcome,

Tobias Loddenkemper, MD
Course Committee Co-Chair

William O. Tatum, DO

Suzette M. LaRoche, MD
Scientific Program Committee Co-Chair

Greg Worrell, MD
ACNS INFORMATION

ACNS Council
President
Frank W. Drislane, MD
Beth Israel Deaconess Medical Center

First Vice President
Aatif M. Husain, MD
Duke University Medical Center

Second Vice President
William O. Tatum, DO
Mayo College of Medicine

Secretary
Jonathan C. Edwards, MD
Medical University of South Carolina

Treasurer
Stephan U. Schuele, MD, MPH
Northwestern University

Immediate Past President
Susan T. Herman, MD
Beth Israel Deaconess Medical Center

Past President
Douglas R. Nordli, Jr., MD
Children’s Epilepsy Center

Councilors-at-Large
Jeffrey Britton, MD
Mayo Clinic

Cecil D. Hahn, MD, MPH
The Hospital for Sick Children

Tobias Loddenkemper, MD
Children’s Hospital Boston

Jaime Lopez, MD
Stanford University

Suraj Muley, MD
St. Joseph’s Hospital & Medical Center

Raj D. Sheth, MD
Mayo Clinic

Francis O. Walker, MD
Wake Forest University

AMA Officer
Marc R. Nuwer, MD, PhD
UCLA

Journal Editor
John Ebersole, MD
University of Chicago Medical Center

ACNS COURSE & PROGRAM COMMITTEES

2013-14 Course Committee
Co-Chairs:
Tobias Loddenkemper, MD
Children’s Hospital Boston

William O. Tatum, DO
Mayo College of Medicine

Members:
Nicholas S. Abend, MD
Children’s Hospital of Philadelphia

Lawrence J. Hirsch, MD
Yale University

Daniela Minecan, MD
University of Michigan

Juan Ochoa, MD
South Alabama Medical Science Foundation

Ex-Officio:
Suzette M. LaRoche, MD
Emory University School of Medicine

Greg Worrell, MD
Mayo Clinic Neurology

2013-14 Program Committee
Co-Chairs:
Suzette M. LaRoche, MD,
Emory University School of Medicine

Members:
Gloria Golloway, MD
Ohio State University

Cecil D. Hahn, MD, MPH
The Hospital for Sick Children

Mark Hallett, MD
National Institutes of Health

Lawrence J. Hirsch, MD
Yale University

Aatif M. Husain, MD
Duke University Medical Center

Ekrem Kurluay, MD
MUSC

Jong Woo Lee, MD, PhD
Brigham & Women’s Hospital

Alan D. Legott, MD, PhD
Montefiore Medical Center

Tobias Loddenkemper, MD
Children’s Hospital Boston

Jaime Lopez, MD
Stanford University

Ex-Officio:
Susan T. Herman, MD
Beth Israel Deaconess Medical Center

Stephan U. Schuele, MD, MPH
Northwestern University

William O. Tatum, DO
Mayo College of Medicine

About the American Clinical Neurophysiology Society (ACNS)

ACNS is a professional association dedicated to fostering excellence in clinical neurophysiology and furthering the understanding of central and peripheral nervous system function in health and disease through education, research, and the provision of a forum for discussion and interaction.

Founded in 1946 and originally named the American Electroencephalographic Society (AEEGS), ACNS is the major professional organization in the United States devoted to the establishment and maintenance of standards of professional excellence in clinical neurophysiology in the practice of neurology, neurosurgery and psychiatry. ACNS members utilize neurophysiology techniques in the diagnosis and management of patients with disorders of the nervous system and in research examining the function of the nervous system in health and disease.
PAST PRESIDENTS

1947 *Herbert H. Jasper, MD, PhD
1948 *Herbert H. Jasper, MD, PhD
1949 *Frederic A. Gibbs, MD
1950 *Hallowell Davis, MD
1951 *Robert Schwab, MD
1952 *James O’Leary, MD
1953 *Robert B. Aird, MD
1954 *Mary A.B. Brazier, DSc
1955 *A. Earl Walker, MD
1956 *Reginald G. Bickford, MD
1957 *John R. Knott, PhD
1958 *Robert S. Dow, MD
1959 *W. Theodore Liberson, MD
1960 *Arthur A. Ward, Jr., MD
1961 *Jerome K. Merlis, MD
1962 *Charles E. Henry, PhD
1963 *Cosimo Ajmone-Marsan, MD
1964 *Peter Kellaway, PhD
1965 *Donald B. Lindsley, PhD
1966 *David D. Daly, MD
1967 Kenneth A. Kooi, MD
1968 Gian-Emilio Chatrian, MD
1969 Robert J. Ellingson, PhD, MD
1970 Donald W. Klass, MD
1971 *Daniel Silverman, MD
1972 Eli S. Goldensohn, MD
1973 *Richard D. Walter, MD
1974 Janice R. Stevens, MD
1975 Ernst A. Rodin, MD
1976 *John S. Barlow, MD
1977 *Fernando Torres, MD
1978 *Frank Morrell, MD
1979 *Pierre Gloor, MD, PhD
1980 Richard N. Harner, MD
1981 Jack D. Grabow, MD
1982 Roger Q. Crocco, MD
1983 *Cesare T. Lombroso, MD
1984 Robert J. Gumnit, MD
1985 Andrew J. Gabor, MD, PhD
1986 John A. Wada, MD
1987 Frank W. Sharbrough, MD
1988 Joan B. Crocco, MD
1989 Barry R. Tharp, MD
1990 Timothy A. Pedley, MD
1991 *Ernst Niedermeyer, MD
1992 Barbara F. Westmoreland, MD
1993 Jerome Engel, MD, PhD
1994 Marc R. Nuwer, MD, PhD
1995 Michael J. Aminoff, MD
1996 John S. Ebersole, MD
1997 Solomon L. Moshe, MD
1998 Warren T. Blume, MD
1999 C. William Erwin, MD
2000 Michael R. Sperling, MD
2001 Eli M. Mizrahi, MD
2002 Bruce J. Fisch, MD
2003 Charles M. Epstein, MD
2004 Donald L. Schomer, MD
2005 Ronald G. Emerson, MD
2006 Richard P. Brenner, MD
2007 Mark A. Ross, MD
2008 Alan D. Legatt, MD, PhD
2009 Gareth J. Parry, MD
2010 Peter W. Kaplan, MB, FRCP
2011 Douglas R. Nordli, Jr., MD
2012 Susan T. Herman, MD
* Deceased

30th International Congress of Clinical Neurophysiology (ICCN) of the IFCN

58th Annual Meeting of the German Society for Clinical Neurophysiology and Functional Imaging (DGKN)

March 19-23, 2014

Late abstract submission for ICCN and DGKN closes February 15, 2014!

Will you be in Berlin?

Please join ACNS and the Canadian Society of Clinical Neurophysiologists (CSCN) as they present their bid to host the 31st ICCN in 2018! Voting occurs immediately following bid presentations to the General Assembly on Saturday, March 22, at approximately 3:00 PM.
GENERAL MEETING INFORMATION

Registration Desk
Location: International Foyer, 6th floor
Tuesday, February 4: 8:00 AM – 5:00 PM
Wednesday, February 5: 6:30 AM – 5:00 PM
Thursday, February 6: 6:30 AM – 5:00 PM
Location: Atlanta Ballroom Foyer, 7th floor
Friday, February 7: 7:00 AM – 5:00 PM
Saturday, February 8: 7:00 AM – 5:00 PM
Sunday, February 9: 7:00 AM – 12:00 PM

Internet
Wireless internet access is available to Annual Meeting & Courses delegates throughout the meeting space. To access the internet, use the following network credentials:
Network: WEstin Meetings
Password: neurophys

Certificate of Attendance & CME Certificates
CME certificates will be available to pre-registered delegates immediately upon the close of the meeting at www.acns.org. Delegates who registered on-site will receive an email with further information within 3 weeks of the end of the meeting.

Delegates are REQUIRED to complete session evaluations to obtain a CME Certificate or Certificate of Attendance. Delegates should log on to the website listed above and enter their last name and the ID# listed at the top of their Annual Meeting & Courses confirmation form. The system will then ask delegates to indicate which sessions they attended, to complete evaluation forms for each of those sessions, and then will generate a PDF certificate which may be printed or saved to the delegate’s computer. Session attendance and evaluation information are saved in the database, and certificates may be accessed again, in the event the certificate is lost or another copy is required.

Please note that certificates will not be mailed or emailed after the meeting. The online certificate program is the only source for this documentation. Please contact ACNS at info@acns.org for any questions. ACNS asks that all CME certificates be claimed no later than April 1, 2014.

Business Meeting
The ACNS Annual Business Meeting will be held in Atlanta Ballroom, from 6:00 – 6:30 PM on Saturday February 8, 2014. This meeting is open to all attendees, but only ACNS Fellows, Members and Honorary Members may vote.

Poster Sessions
Authors will be present during poster tours between 12:30 – 1:30 PM on Friday, February 7 and 12:30 – 2:00 PM on Saturday, February 8 for discussion. Poster abstracts and presentation dates can be found on page 49.

Friday, February 7, 2014
7:00 AM – 4:00 PM
Exhibit & Poster Hall, Conference Center 1-5
Saturday, February 8, 2014
7:00 AM – 2:00 PM
Exhibit & Poster Hall, Conference Center 1-5

ACNS is not responsible for posters remaining on boards after presentation hours.

Publication of Abstracts
Speaker abstracts and poster abstracts will be published in the Journal of Clinical Neurophysiology.
GENERAL MEETING INFORMATION — CONTINUED

Venue Information
The Westin Peachtree Plaza is the location for the 2014 Annual Meeting and Courses. Calls should be directed to the American Clinical Neurophysiology Society Registration Desk.

210 Peachtree St NE
Atlanta, GA 30303
(404) 659-1400

Westin Peachtree Plaza Floorplan
NEARBY RESTAURANTS

Sun Dial Restaurant, Bar & View
Level 72 -Atop the Hotel
Extension 77200
American Cuisine & breathtaking view

AlmaCucina
Across from hotel (Peachtree St)
(404) 968-9662
Modern Mexican

Azio
229 Peachtree Street
(404) 968-9662
Modern Mexican

Corner Bakery
270 Peachtree Street
(404) 215-9000
Deli

Durango Steakhouse
230 Peachtree Street
(404) 222-0103
Steakhouse

Fire of Brazil
218 Peachtree Street
(404) 525-5255
Brazilian Steakhouse

Hard Rock Cafe
215 Peachtree Street
(404) 420-584
American Casual

Hsu’s
192 Peachtree Center Avenue
(404) 659-2788
Gourmet Chinese

Jalapeno Charlie’s
218 Peachtree Street
(404) 525-5825
Mexican

Mama Ninfa’s
231 Peachtree Street
(404) 521-3500
Mexican

Meehan’s Pub
Connected to hotel on 6th level
(404) 214-9821
Irish Fare & Burgers

Peachtree Center Food Court
Under Peachtree Center diagonal from 6th level entrance of hotel
Includes:
- Chick-Fil-A
- Williy’s Mexican
- Moe’s
- Subway
- Farmer’s Basket
- CafeMoMo
- My Friend’s Place
- Dairy Queen
- Benihana

Ray’s in the City
240 Peachtree Street
(404) 524-9224
Seafood and Sushi

Sweet Georgia’s Juke Joint
200 Peachtree Street
(404) 230-5853
Southern Fare
Educational Mission Statement

Purpose
The American Clinical Neurophysiology Society (ACNS) is a professional association dedicated to fostering excellence in clinical neurophysiology and furthering the understanding of central and peripheral nervous system function in health and disease through education, research, and the provision of a forum for discussion and interaction.

Content
ACNS is committed to providing continuing medical education to its members and others interested in clinical neurophysiology. Educational objectives include 1) Reviewing current knowledge of clinical neurophysiology including: electroencephalography, evoked potentials, electromyography, nerve conduction studies, intraoperative monitoring, polysomnography and other sleep technology, quantitative neurophysiological methods, magnetoencephalography, sleep disorders, epilepsy, neuromuscular disorders, brain stimulation, brain-computer interfacing, and related areas; and 2) Informing course and meeting attendees of recent technological developments and their implications for clinical practice.

Target Audience
The Society’s educational activities are directed to clinical neurophysiologists, neurologists, psychiatrists, physical therapists, neurosurgeons, trainees in these disciplines and other physicians and researchers who utilize clinical neurophysiological techniques and knowledge in the diagnosis and management of patients with disorders of the nervous system.

Expected Result
Attendees will improve competence in clinical neurophysiology procedures and incorporate new technological advancements into their practice.

Gaps And Needs
In compliance with the Updated Accreditation Criteria of the Accreditation Council for Continuing Medical Education (ACCME), the Continuing Medical Education Committee of the ACNS has identified “professional practice gaps.” Definition: A “professional practice gap” is the difference between what a health professional is doing or accomplishing compared to what is achievable on the basis of current professional knowledge.

The following professional practice gaps and educational needs were identified by a combined effort of the Program, Course and CME Committees.

Identified Gaps And Needs

Gap 1. Emerging Areas of Practice
Neurological intraoperative monitoring (NIOM) and intensive care unit EEG monitoring (ICU EEG) are new and rapidly evolving areas of clinical neurophysiology. Few practicing neurologists have adequate training in these techniques, and physicians with competence in these areas are in great demand. Educational activities should cover both basic methodologies for those practitioners new to ICU EEG and NIOM, and innovative techniques.

Gap 2. General Practice of Clinical Neurophysiology
Clinical neurophysiology procedures are performed by a large proportion of practicing US neurologists, many of whom have little or no formal training in clinical neurophysiology. Many clinical neurophysiology procedures (e.g. evoked potentials, invasive EEG) are performed at low volume at most centers, and a forum for review and hands-on interpretation are essential to maintain competence in these areas.

Several specific topics with significant gaps between current practice and ideal practice have been identified via review of the literature, review of clinical neurophysiology fellowship curricula, and surveys of ACNS members and Annual Meeting attendees. These include:

- Peripheral neurophysiology, Pediatric EMG, critical illness related neurophysiology, and muscle ultrasound
- Basic EEG: Identification of normal variants, identification of artifacts, clinical correlation
- Pediatric EEG, especially neonatal EEG
- Digital EEG processing, e.g. quantitative EEG and trends for use in the intensive care unit, source localization, coregistration with neuroimaging, etc.
- Full band EEG, Ultrafast and ultraslow EEG
- NIOM: Motor evoked potentials, guidelines and standards of care for NIOM (e.g. indications, cost effectiveness)
- Evoked potentials: Current role of short-and long-latency EPs
- Video-EEG monitoring
- Sleep, Use of new scoring system, implications for patient care

Objectives
It is intended that, as a result of attending the meeting and/or courses, physician attendees will be able to identify changes in competence or performance that are desirable. Definitions: “Competence” is knowing how to do something. “Performance” is what the physician would do in practice, if given the opportunity.

Evaluation
The updated ACCME accreditation criteria are designed to integrate with the new requirements for maintenance of certification (for more information see www.ABPN.org). Physicians are expected to perform self-assessments of their practice, but the ACNS, as an organization accredited by the ACCME, is expected to measure how its educational activities assist physicians in this activity. Thus, there are new questions in the evaluation form. These questions address your intended changes in competence or performance. In a few months, we will contact all physician meeting attendees to ask you if you actually HAVE experienced changes in competence or performance. Your responses, now and in the future, will assist us and ultimately you in determining educational activities that are most useful to you.
CME INFORMATION — CONTINUED

Meeting Description
This year’s scientific program will feature the latest scientific advances in clinical neurophysiology presented by leading national and international experts in the field. Increased audience interactivity will be a theme throughout all the programs, and session chairs are developing creative ways to engage with the audience. This dynamic program has more choices than ever. The parallel sessions will usually provide simultaneous sessions for interests in EEG, electrodiagnosis and monitoring. There will also be workshops and Special Interest Groups.

Annual Courses Learning Objectives
At the conclusion of the Annual Courses, the learner should be able to:
1. Describe the indications for use of clinical neurophysiology techniques in diagnosis of disorders of the nervous system;
2. Incorporate new neurophysiology procedures and technological advances into his/her own clinical practice; and
3. Perform and interpret a broad range of clinical neurophysiology procedures, and integrate the results of these tests into comprehensive patient management plans.

Specific objectives related to each session are provided on pages 16-20.

Annual Meeting Learning Objectives
At the conclusion of the Annual Meeting, the learner should be able to:
1. Discuss recent advances in electroencephalography, evoked potentials, ALS, magnetoencephalography, practice technologies, nerve conduction studies and other clinical neurophysiology techniques; and
2. Apply advances in clinical neurophysiology techniques to improve the diagnosis of neurologic disorders.

Specific objectives related to each session are provided on pages 21-29.

Target Audience
The Society’s educational activities are directed to clinical neurophysiologists, neurologists, psychiatrists, physiatrists, neurosurgeons, trainees in these disciplines, other physicians and researchers, and neurophysiology technologists who specialize in the utilization of clinical neurophysiological techniques that advance the knowledge in the diagnosis and management of patients with disorders of the peripheral and central nervous system.

Accreditation Statement
This activity has been planned and implemented in accordance with the Essential Areas and Policies of the Accreditation Council for Continuing Medical Education (ACCME) through the sponsorship of ACNS. ACNS is accredited by ACCME to provide continuing medical education for physicians.

Credit Designation
ACNS designates the Annual Meeting for a maximum of 20 AMA PRA Category 1 Credit(s)TM. Physicians should claim only credit commensurate with the extent of their participation in the activity.

Important Dates

ACNS designates the Annual Courses for the maximum number of AMA PRA Category 1 Credit(s)TM indicated below:

- Intraoperative Monitoring Part I
  6.5 AMA PRA Category 1 Credit(s)TM

- EP Reading Session
  1.5 AMA PRA Category 1 Credit(s)TM

- Neonatal EEG
  1.5 AMA PRA Category 1 Credit(s)TM

- Intracranial EEG
  6.5 AMA PRA Category 1 Credit(s)TM

- Intraoperative Monitoring Part II
  6.5 AMA PRA Category 1 Credit(s)TM

- EMG and EEG Technology
  1.5 AMA PRA Category 1 Credit(s)TM

- Updates in the Business of Neurophysiology: From Washington to Your Office
  1.5 AMA PRA Category 1 Credit(s)TM

- ICU EEG
  6.5 AMA PRA Category 1 Credit(s)TM

- EMG
  3 AMA PRA Category 1 Credit(s)TM

- Video-EEG
  3 AMA PRA Category 1 Credit(s)TM

- Applied Autonomic Neurophysiology
  1.5 AMA PRA Category 1 Credit(s)TM

- Case Studies in Peripheral Neurophysiology
  2.5 AMA PRA Category 1 Credit(s)TM

Physicians should claim only credit commensurate with the extent of their participation in the activity.

Important dates:
- CME Certificate Program Opens (pre-registered delegates)
  February 9, 2014
- CME Certificate Program Opens (delegates registering onsite)
  March 7, 2014
- CME Certificate Claim Deadline
  April 30, 2014
CONFLICT OF INTEREST DISCLOSURES

It is the policy of ACNS to ensure balance, independence, objectivity and scientific rigor in all its individually sponsored or jointly sponsored educational programs. In order to comply with the ACCME's Updated Standards for Commercial Support, ACNS requires that anyone who is in a position to control the content of an educational activity discloses all relevant financial relationships with any commercial interest pertaining to the content of the presentation. Should it be determined that a conflict of interest exists as a result of a financial relationship of a planner of the CME activity, the planner must recuse himself or herself from the planning for that activity or relevant portion of that activity. All presentations for which the presenter disclosed a potential conflict of interest were peer reviewed by two members of the CME Committee with no relationships. If bias was found, the presenter was asked to make changes to the presentation and it was re-reviewed for bias before final approval. Refusal to disclose a conflict or the inability to resolve an identified conflict precludes participation in the CME activity. Complete conflict of interest disclosure information pertaining to the Annual Meeting and Courses may be found below.

<table>
<thead>
<tr>
<th>Council</th>
</tr>
</thead>
<tbody>
<tr>
<td>Jeffrey Britton, MD</td>
</tr>
<tr>
<td>Frank Drislane, MD</td>
</tr>
<tr>
<td>John Ebersole, MD</td>
</tr>
<tr>
<td>Jonathan Edwards, MD</td>
</tr>
<tr>
<td>Cecil Hahn, MD, MPH</td>
</tr>
<tr>
<td>Susan Herman, MD</td>
</tr>
<tr>
<td>Aatif Husain, MD</td>
</tr>
<tr>
<td>Tobias Loddenkemper, MD</td>
</tr>
<tr>
<td>Jaime Lopez, MD</td>
</tr>
<tr>
<td>Suraj Muley, MD</td>
</tr>
<tr>
<td>Douglas Nordli, MD</td>
</tr>
<tr>
<td>Marc Nuwer, MD, PhD</td>
</tr>
<tr>
<td>Stephan Schuele, MD, MPH</td>
</tr>
<tr>
<td>Raj Sheth, MD</td>
</tr>
<tr>
<td>William Tatum, DO</td>
</tr>
<tr>
<td>Francis Walker, MD</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Course Committee (if not included above)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nicholas Abend, MD</td>
</tr>
<tr>
<td>Lawrence Hirsch, MD</td>
</tr>
<tr>
<td>Suzette LaRoche, MD</td>
</tr>
<tr>
<td>Alan Legatt, MD, PhD</td>
</tr>
<tr>
<td>Daniela N. Minecan, MD</td>
</tr>
<tr>
<td>Juan Ochoa, MD</td>
</tr>
<tr>
<td>Greg Worrell, MD</td>
</tr>
</tbody>
</table>

a. Grants/Research Support; b. Consultant; c. Stock/Shareholder (self-managed); d. Speaker’s Bureau; e. Advisory Board or Panel; f. Salary, Contractual Services; g. Other Financial or Material Support (royalties, patents, etc.)
### Program Committee (if not included above)

<table>
<thead>
<tr>
<th>Name</th>
<th>Institution</th>
<th>Relationships</th>
</tr>
</thead>
<tbody>
<tr>
<td>Judy Ahn-Ewing</td>
<td>ASET - The Neurodiagnostic Society</td>
<td>No Relationships</td>
</tr>
<tr>
<td>Anto Bagic, MD, PhD</td>
<td>University of Pittsburgh</td>
<td>No Relationships</td>
</tr>
<tr>
<td>Jane Boggs, MD</td>
<td>Wake Forest University</td>
<td>Lundbeck (a)</td>
</tr>
<tr>
<td>Jocelyn Cheng, MD</td>
<td>Drexel University</td>
<td>No Relationships</td>
</tr>
<tr>
<td>Ronald Emerson, MD</td>
<td>Hospital for Special Surgery</td>
<td>Amgen Inc (c); Dow Chemical (c); Eli Lilly (c); Express Scripts (c); Forest Labs (c); General Electric (c); Johnson &amp; Johnson (c); Thermo Fisher Scientific (c); Allergan (c); Bristol Myers Squibb (c); Teva Pharmaceuticals (c); Reach Bionics (b)</td>
</tr>
<tr>
<td>Morris Fisher, MD</td>
<td>Hines VAH</td>
<td>No Relationships</td>
</tr>
<tr>
<td>Gloria Galloway, MD</td>
<td>Ohio State University</td>
<td>No Relationships</td>
</tr>
<tr>
<td>Mark Hallett, MD</td>
<td>NIH</td>
<td>No Relationships</td>
</tr>
<tr>
<td>Ekrem Kutluay, MD</td>
<td>MUSC</td>
<td>No Relationships</td>
</tr>
<tr>
<td>Jong Woo Lee, MD, PhD</td>
<td>Brigham &amp; Women's Hospital</td>
<td>UCB Inc (a); Cephalon/TEVA (a); SleepMed/DigiTrace (f)</td>
</tr>
<tr>
<td>Faye McNall, MEd, REEGT</td>
<td>ASET - The Neurodiagnostic Society</td>
<td>No Relationships</td>
</tr>
<tr>
<td>Mark Ross, MD</td>
<td>Mayo Clinic Arizona</td>
<td>Baxter (e)</td>
</tr>
<tr>
<td>Mirela Simon, MD, MSc</td>
<td>Massachusetts General Hospital</td>
<td>No Relationships</td>
</tr>
<tr>
<td>Saurabh Sinha, MD, PhD</td>
<td>Duke University Medical Center</td>
<td>Wyeth/Pfizer Pharmaceuticals (e); Lundbeck Pharmaceuticals (d,e,g); UCB Pharmaceuticals (d,g)</td>
</tr>
<tr>
<td>Cynthia Stack, MD</td>
<td>Lurie Children’s Hospital</td>
<td>KINEX (c)</td>
</tr>
<tr>
<td>Adriana Tanner, MD</td>
<td>Saint Mary’s Health Care</td>
<td>No Relationships</td>
</tr>
<tr>
<td>Courtney Wusthoff, MD</td>
<td>Stanford University</td>
<td>No Relationships</td>
</tr>
</tbody>
</table>

### CME Committee (if not included above)

<table>
<thead>
<tr>
<th>Name</th>
<th>Institution</th>
<th>Relationships</th>
</tr>
</thead>
<tbody>
<tr>
<td>Jayant Acharya, MD</td>
<td>Penn State Hershey Medical Center</td>
<td>Cyberonics, Inc (d)</td>
</tr>
<tr>
<td>Meriem Bensalem-Owen, MD</td>
<td>University of Kentucky</td>
<td>UCB (a); Eisai (a); Sunovion (a); Lundbeck (a)</td>
</tr>
<tr>
<td>Rohit Das, MD</td>
<td>Indiana University</td>
<td>Satellite Healthcare (a); Satellite Healthcare (g)</td>
</tr>
<tr>
<td>Kitt Kaiboriboon, MD</td>
<td>UH Case Medical Center</td>
<td>No Relationships</td>
</tr>
</tbody>
</table>

### Course Directors and Faculty (if not included above)

<table>
<thead>
<tr>
<th>Name</th>
<th>Institution</th>
<th>Relationships</th>
</tr>
</thead>
<tbody>
<tr>
<td>Deborah Briggs, MD</td>
<td>SBSI/UTSW - Austin</td>
<td>No Relationships</td>
</tr>
<tr>
<td>Sydney Cash, MD PhD</td>
<td>Mass General Hospital</td>
<td>No Relationships</td>
</tr>
<tr>
<td>Elliot Dimberg, MD</td>
<td>Mayo Clinic</td>
<td>No Relationships</td>
</tr>
<tr>
<td>Dennis Dlugos, MD</td>
<td>CHOP</td>
<td>No Relationships</td>
</tr>
<tr>
<td>Charles Epstein, MD</td>
<td>Emory University School of Medicine</td>
<td>Neuronetics, Inc (g)</td>
</tr>
<tr>
<td>William Gellentine, DO</td>
<td>Duke University Medical Center</td>
<td>No Relationships</td>
</tr>
<tr>
<td>Paul Garcia, MD, PhD</td>
<td>Emory Health</td>
<td>No Relationships</td>
</tr>
<tr>
<td>Nicolas Gaspard, MD, PhD</td>
<td>Yale</td>
<td>No Relationships</td>
</tr>
<tr>
<td>Emily Gilmore, MD</td>
<td>Yale University School of Medicine</td>
<td>No Relationships</td>
</tr>
<tr>
<td>Andres Gonzalez, MD, MMM</td>
<td>University of Southern California</td>
<td>No Relationships</td>
</tr>
<tr>
<td>Stephen Hantus, MD</td>
<td>Cleveland Clinic</td>
<td>No Relationships</td>
</tr>
<tr>
<td>Leo Happel, PhD</td>
<td>LSU Health Science Center</td>
<td>No Relationships</td>
</tr>
<tr>
<td>Monica Islam, MD</td>
<td>Nationwide Children’s Hospital</td>
<td>No Relationships</td>
</tr>
<tr>
<td>Randa Jarrar, MD</td>
<td>Phoenix Children’s Hospital</td>
<td>No Relationships</td>
</tr>
</tbody>
</table>
## Course Directors and Faculty (if not included above) — Continued

<table>
<thead>
<tr>
<th>Name</th>
<th>Institution</th>
<th>Relationships</th>
</tr>
</thead>
<tbody>
<tr>
<td>Andrew Kim, MD</td>
<td>Ann and Robert H. Lurie Children’s Hospital of Chicago</td>
<td>No Relationships</td>
</tr>
<tr>
<td>A. Arturo Leis, MD</td>
<td>Methodist Rehabilitation Center</td>
<td>No Relationships</td>
</tr>
<tr>
<td>Michael McGarvey, MD</td>
<td>Hospital of the University of Pennsylvania</td>
<td>No Relationships</td>
</tr>
<tr>
<td>Daniel Menkes, MD</td>
<td>University of Connecticut</td>
<td>No Relationships</td>
</tr>
<tr>
<td>Yafa Minazad, DO</td>
<td>Southern California Neurology Consultants</td>
<td>No Relationships</td>
</tr>
<tr>
<td>L. Elizabeth Mullikin, MPA, FACHE, FASET</td>
<td>Sutter Health East Bay Regional Executive Neuroscience Service Line</td>
<td>No Relationships</td>
</tr>
<tr>
<td>Chris Nance, MD</td>
<td>West Virginia University</td>
<td>No Relationships</td>
</tr>
<tr>
<td>Brett Netherton, MS, FASNM, CNIM</td>
<td>Rocworks, LLC</td>
<td>Signal Gear, LLC (g); Rocworks, LLC (g)</td>
</tr>
<tr>
<td>Viet Nguyen, MD</td>
<td>Stanford</td>
<td>No Relationships</td>
</tr>
<tr>
<td>Eva Ritzl, MD</td>
<td>Johns Hopkins University</td>
<td>No Relationships</td>
</tr>
<tr>
<td>Elayna Rubens, MD</td>
<td>New York Presbyterian Hospital-Weill Cornell Medical Center</td>
<td>No Relationships</td>
</tr>
<tr>
<td>Devon Rubin, MD</td>
<td>Mayo Clinic</td>
<td>AANEM (g); AAN (g)</td>
</tr>
<tr>
<td>Steven Schachter, MD</td>
<td>CIMIT</td>
<td>No Relationships</td>
</tr>
<tr>
<td>Mark Scher, MD</td>
<td>Rainbow Babies and Children’s Hospital</td>
<td>No Relationships</td>
</tr>
<tr>
<td>Sarah Schmitt, MD</td>
<td>University of Pennsylvania</td>
<td>No Relationships</td>
</tr>
<tr>
<td>Donald Schomer, MD</td>
<td>Harvard</td>
<td>No Relationships</td>
</tr>
<tr>
<td>Stanley Skinner, MD</td>
<td>Abbott Northwestern Hospital</td>
<td>Medtronic (g)</td>
</tr>
<tr>
<td>Michael Sperling, MD</td>
<td>Thomas Jefferson University</td>
<td>UCB (a); Eisai (a); SK Life Sciences (a); Vertex (a); Medtronics (a); Marinus (a); Sunovion (a); Visualase (a); Neuronex (a); Accorda Therapeutics (b); electroCore (b)</td>
</tr>
<tr>
<td>William Stacey, MD PhD</td>
<td>University of Michigan</td>
<td>No Relationships</td>
</tr>
<tr>
<td>M. Brandon Westover, MD PhD</td>
<td>Massachusetts General Hospital</td>
<td>No Relationships</td>
</tr>
</tbody>
</table>

## Annual Meeting Directors and Faculty (if not included above)

<table>
<thead>
<tr>
<th>Name</th>
<th>Institution</th>
<th>Relationships</th>
</tr>
</thead>
<tbody>
<tr>
<td>Katharine Alter, MD</td>
<td>Mt. Washington Pediatric Hospital</td>
<td>Allergan (b,d,e); Ipsen (e); Terason (g)</td>
</tr>
<tr>
<td>Juan Bulacio, MD</td>
<td>Cleveland Clinic Foundation</td>
<td>No Relationships</td>
</tr>
<tr>
<td>Richard Burgess, MD, PhD</td>
<td>Cleveland Clinic Epilepsy Center</td>
<td>No Relationships</td>
</tr>
<tr>
<td>David Burke, MD, DSc</td>
<td>The University of Sydney</td>
<td>No Relationships</td>
</tr>
<tr>
<td>Michael Cartwright, MD, MS</td>
<td>Wake Forest School of Medicine</td>
<td>Elsevier Publishing (g)</td>
</tr>
<tr>
<td>Kevin Chapman, MD</td>
<td>Children’s Hospital Colorado</td>
<td>Lundbeck (a)</td>
</tr>
<tr>
<td>Jan Cloassen, MD, PhD</td>
<td>Columbia</td>
<td>No Relationships</td>
</tr>
<tr>
<td>Peter Dempsey, MD</td>
<td>The Lahey Hospital and Health System</td>
<td>No Relationships</td>
</tr>
<tr>
<td>Matthew Eccher, MD, MSPH</td>
<td>University Hospitals Case Medical Center</td>
<td>No Relationships</td>
</tr>
<tr>
<td>Joshua Ehrenberg, BS, R EEG T, CNIM</td>
<td>Emory University</td>
<td>No Relationships</td>
</tr>
<tr>
<td>Joshua Ewen, MD</td>
<td>Kennedy Krieger Institute</td>
<td>No Relationships</td>
</tr>
<tr>
<td>Name</td>
<td>Institution</td>
<td>Relationships</td>
</tr>
<tr>
<td>-------------------------------------------</td>
<td>-------------------------------------------------------</td>
<td>--------------------------------</td>
</tr>
<tr>
<td>Evan Fertig, MD</td>
<td>Northeast Regional Epilepsy Group</td>
<td>UCB (a)</td>
</tr>
<tr>
<td>Brandon Foreman, MD</td>
<td>Columbia</td>
<td>No Relationships</td>
</tr>
<tr>
<td>Michael Gandal, MD, PhD</td>
<td>UCLA</td>
<td>No Relationships</td>
</tr>
<tr>
<td>Jay Gavvala, MD</td>
<td>Northwestern Memorial Hospital</td>
<td>No Relationships</td>
</tr>
<tr>
<td>David Gloss, MD, MPH&amp;TM</td>
<td>Barrow Neurologic Institute</td>
<td>No Relationships</td>
</tr>
<tr>
<td>Jorge Alvaro Gonzalez-Martinez, MD, PhD</td>
<td>Cleveland Clinic Foundation</td>
<td>No Relationships</td>
</tr>
<tr>
<td>Brent Goodman, MD</td>
<td>Mayo Clinic</td>
<td>No Relationships</td>
</tr>
<tr>
<td>Greg Hajcak, PhD</td>
<td>Stony Brook University</td>
<td>NIH (a,b)</td>
</tr>
<tr>
<td>Tyson Hale, AuD</td>
<td>Geisinger Medical Center</td>
<td>No Relationships</td>
</tr>
<tr>
<td>Stuart Hoffman, DO</td>
<td>Geisinger Medical Center</td>
<td>No Relationships</td>
</tr>
<tr>
<td>Safwan Jaradeh, MD</td>
<td>Stanford University</td>
<td>No Relationships</td>
</tr>
<tr>
<td>Jennifer Jones, DO</td>
<td>Mission Neurology Associates</td>
<td>No Relationships</td>
</tr>
<tr>
<td>Peter Kang, MD</td>
<td>University of Florida College of Medicine</td>
<td>ISIS Pharmaceuticals (a); 3rd Rock Ventures (b)</td>
</tr>
<tr>
<td>Peter Kaplan, MD, FRCP</td>
<td>Johns Hopkins Bayview Medical Center</td>
<td>Johns Hopkins (g)</td>
</tr>
<tr>
<td>Ioannis Karakis, MD, MSc</td>
<td>Emory University</td>
<td>No Relationships</td>
</tr>
<tr>
<td>Jeffrey Kennedy, MD</td>
<td>Northwestern Medical Faculty Foundation</td>
<td>No Relationships</td>
</tr>
<tr>
<td>Vita Kesner, MD</td>
<td>Emory University Hospital</td>
<td>No Relationships</td>
</tr>
<tr>
<td>Cliff Klein, PhD</td>
<td>Rehabilitation Institute of Chicago</td>
<td>No Relationships</td>
</tr>
<tr>
<td>Ekrem Kufluay, MD</td>
<td>MUSC</td>
<td>No Relationships</td>
</tr>
<tr>
<td>Shafeeq Ladha, MD</td>
<td>Barrow Neurological Institute</td>
<td>No Relationships</td>
</tr>
<tr>
<td>Samden Lhatoo, MD</td>
<td>Case Medical Center and Case Western Reserve University School of Medicine</td>
<td>Lundbeck (d)</td>
</tr>
<tr>
<td>Brian Litt, MD</td>
<td>University of Pennsylvania</td>
<td>NeuroPace (c,g); Glaxo Smith Kline (a,g); Medtronic (a,g)</td>
</tr>
<tr>
<td>Rodolfo Llanas, MD, PhD</td>
<td>NYU School of Medicine</td>
<td>No Relationships</td>
</tr>
<tr>
<td>David Loring, PhD</td>
<td>Emory</td>
<td>Pfizer (a); UCB (a); Oxford University Press (g); NeuroPace (b); Biogen (d); Questcor (d); Teva (d)</td>
</tr>
<tr>
<td>Hugh McMillan, MD, MSc, FRCP, FAAN</td>
<td>Children’s Hospital of Eastern Ontario</td>
<td>No Relationships</td>
</tr>
<tr>
<td>Jonathan Miller, MD</td>
<td>Case Medical Center and Case Western Reserve University School of Medicine</td>
<td>No Relationships</td>
</tr>
<tr>
<td>Sherry Nehamkin, R. EEG/EP T., CNIM, CLTM</td>
<td>Cleveland clinic</td>
<td>No Relationships</td>
</tr>
<tr>
<td>Heather Olson, MD</td>
<td>Boston Children’s Hospital</td>
<td>No Relationships</td>
</tr>
<tr>
<td>Erik Ortega, MD</td>
<td>St Joseph’s Hospital and Medical Center</td>
<td>No Relationships</td>
</tr>
<tr>
<td>Matthew Pitt, MD, FRCP</td>
<td>Great Ormond Street Hospital</td>
<td>No Relationships</td>
</tr>
</tbody>
</table>
### CONFLICT OF INTEREST DISCLOSURES — CONTINUED

<table>
<thead>
<tr>
<th>Annual Meeting Directors and Faculty (if not included above) — Continued</th>
</tr>
</thead>
<tbody>
<tr>
<td>James John Riviello, MD</td>
</tr>
<tr>
<td>Eric Rosenthal, MD</td>
</tr>
<tr>
<td>W. Zev Rymer, MD, PhD</td>
</tr>
<tr>
<td>Francesco Sala, MD</td>
</tr>
<tr>
<td>Iván Sánchez Fernández, MD</td>
</tr>
<tr>
<td>Kathleen Seidel, MD</td>
</tr>
<tr>
<td>Jay Shils, PhD</td>
</tr>
<tr>
<td>Mark Stecker, MD, PhD</td>
</tr>
<tr>
<td>Parthasarathy Thirumala, MD</td>
</tr>
<tr>
<td>Tammy Tsuchida, MD PhD</td>
</tr>
<tr>
<td>Michel Van Putten, MD, PhD</td>
</tr>
<tr>
<td>Jay Varma, MD</td>
</tr>
</tbody>
</table>

### Executive Office Staff

| Megan M. Kelley, CNP | ACNS | No Relationships |

---

a. Grants/Research Support; b. Consultant; c. Stock/Shareholder (self-managed); d. Speaker’s Bureau; e. Advisory Board or Panel; f. Salary, Contractual Services; g. Other Financial or Material Support (royalties, patents, etc.)
AWARD RECIPIENTS & LECTURES

Friday, February 7, 2014

2014 Robert S. Schwab Award Presentation & Lecture
“THE EXCITABLE AXON”
David Burke, MD, DSc
The Schwab Award is presented annually by to an individual who has made significant contributions in the area of clinical neurophysiology. Dr. Burke is Professor at the University of Sydney Medical School and current editor-in-chief of Clinical Neurophysiology, the official journal of the International Federation of Clinical Neurophysiology (IFCN).

2013 Pierre Gloor Award & Plenary Lecture
“THALAMOCORTICAL RHYTHMS AND DYSRHYTHMIAS”
Rodolfo Llinas, MD, PhD
The Gloor Award is presented annually for outstanding current contributions to clinical neurophysiology research. Dr. Rodolfo Llinas, MD, PhD was named the 2013 Gloor Award recipient but was unable to attend the 2013 Annual Meeting due to weather conditions. ACNS is pleased to welcome Dr. Llinas back to deliver his address and receive his award. Dr. Llinas is currently the Thomas and Suzanne Murphy Professor of Neuroscience and Chairman of the department of Physiology & Neuroscience at the NYU School of Medicine.

Saturday, February 8, 2014

2014 Pierre Gloor Award Presentation & Lecture
“All Waves That Glitter Are Not Gold - Lessons of the Penumbra and the Core”
Ronald Emerson, MD
The Gloor Award is presented annually for outstanding current contributions to clinical neurophysiology research. Dr. Emerson is an Attending Neurologist at New York City’s Hospital for Special Surgery (HSS) and a former president of ACNS.

2014 Herbert H. Jasper Award Presentation
Ernst Rodin, MD
The Jasper Award is presented annually to an individual who has made a lifetime of outstanding contributions to the field of clinical neurophysiology. Dr. Rodin is an Adjunct Professor in the Department of Neurology at the University of Utah and a former president and Honorary Member of ACNS.

NETWORKING & SOCIAL EVENTS

WELCOME RECEPTION
Friday, February 7, 2014
6:45 – 8:00 PM
Dr. Frank W. Drislane, MD formally invites all Annual Meeting delegates to attend the ACNS Welcome Reception on Friday, February 7, from 6:45 – 8:00 PM in ACNS Exhibit Hall, 200 Peachtree, 7th Floor. There will be complimentary hors d’oeuvre provided and you will get a chance to see all the new and familiar exhibitors.

PROFESSIONAL DEVELOPMENT MENTORING PROGRAM
ACNS is happy to continue the Professional Development Mentoring Program on Saturday, February 8, from 1:30 – 2:00 PM. If you signed up to be a Mentor or Mentee, please pick up a boxed lunch in the Exhibit Hall and join us in International B, 6th Floor!
### ANNUAL COURSES PROGRAM

**Tuesday, February 4, 2014**

**Intraoperative Monitoring Course, Part I**

9:00 AM — 5:00 PM  
International DE, 6th floor  
*Course Co-Directors: Jaime R. Lopez, MD and Michael L. McGarvey, MD*

**Objectives:**
At the conclusion of this activity, participants will be able to:

1. Employ a thorough understanding of neuroanatomy and neurophysiology to identify risks for injury to the brain, spine, and cranial and peripheral nerves during surgical and other invasive procedures, and to select appropriate monitoring techniques to minimize these risks.

2. Design a comprehensive monitoring plan for individual patients, including multimodality intraoperative monitoring techniques (e.g. recordings of sensory and motor evoked potentials, EEG, EMG, and spinal reflex activity) to monitor segments of the nervous system at risk during surgery.

3. Recognize changes in intraoperative neurophysiologic tests which indicate damage to neural structures, and distinguish these from common technical artifacts.

4. Communicate normal and abnormal results to the surgical team, and incorporate results into clinical recommendations that may alter the surgical technique to avoid, limit or reverse injury to neural structures.

5. Identify the effects of anesthetic drugs on neurophysiology and employ methods to limit the adverse impact of anesthetics on intraoperative monitoring techniques.

**Agenda:**

<table>
<thead>
<tr>
<th>Time</th>
<th>Activity</th>
</tr>
</thead>
<tbody>
<tr>
<td>9:00</td>
<td>Welcome and Introduction</td>
</tr>
<tr>
<td>9:05</td>
<td>BAEP Monitoring</td>
</tr>
<tr>
<td></td>
<td>Alan Legatt, MD, PhD</td>
</tr>
<tr>
<td>9:45</td>
<td>SEP Monitoring</td>
</tr>
<tr>
<td></td>
<td>Andras Gonzalez, MD, MMA</td>
</tr>
<tr>
<td>10:25</td>
<td>Break</td>
</tr>
<tr>
<td>10:40</td>
<td>MEP Monitoring</td>
</tr>
<tr>
<td></td>
<td>Ronald Emerson, MD</td>
</tr>
<tr>
<td>11:20</td>
<td>EEG and Doppler Ultrasound Monitoring</td>
</tr>
<tr>
<td></td>
<td>Michael McGarvey, MD</td>
</tr>
<tr>
<td>12:00</td>
<td>Panel Discussion</td>
</tr>
<tr>
<td>12:15</td>
<td>Lunch (on own, see p. 7 for nearby dining)</td>
</tr>
<tr>
<td>1:15</td>
<td>EMG Monitoring of Central Motor Pathways During Spine Surgery</td>
</tr>
<tr>
<td></td>
<td>Stanley Skinner, MD</td>
</tr>
<tr>
<td>1:55</td>
<td>Monitoring of Spinal Nerve Roots</td>
</tr>
<tr>
<td></td>
<td>Monica Islam, MD</td>
</tr>
<tr>
<td>2:35</td>
<td>Monitoring of Peripheral Nerve Surgery</td>
</tr>
<tr>
<td></td>
<td>Leo Happel, PhD</td>
</tr>
<tr>
<td>3:15</td>
<td>Break</td>
</tr>
<tr>
<td>3:30</td>
<td>Anesthetic Management and IOM</td>
</tr>
<tr>
<td></td>
<td>Paul Garcia, MD, PhD</td>
</tr>
<tr>
<td>4:10</td>
<td>Case Presentations and Discussion</td>
</tr>
<tr>
<td>4:50</td>
<td>Panel Discussion</td>
</tr>
</tbody>
</table>

**Wednesday, February 5, 2014**

**EP Reading Session**

7:00 — 8:30 AM  
International DE, 6th floor  
*Course Director: Alan D. Legatt, MD, PhD*

**Objectives:**
At the conclusion of this activity, participants will be able to:

1. Select appropriate evoked potential techniques (visual, brainstem auditory, and somatosensory) based on a thorough understanding of neuroanatomy and neurophysiology.

2. Accurately interpret visual, brainstem auditory, and somatosensory evoked potentials to localize dysfunction of the nervous system.

3. Integrate the results of evoked potentials with clinical history and other diagnostic techniques to improve accuracy of neurologic diagnosis.

**Agenda:**

<table>
<thead>
<tr>
<th>Time</th>
<th>Activity</th>
</tr>
</thead>
<tbody>
<tr>
<td>7:00</td>
<td>Brainstem Auditory Evoked Potentials (BAEPs)</td>
</tr>
<tr>
<td></td>
<td>Alan D. Legatt, MD, PhD</td>
</tr>
<tr>
<td>7:30</td>
<td>Visual Evoked Potentials (VEPs)</td>
</tr>
<tr>
<td></td>
<td>Elayna Rubens, MD</td>
</tr>
<tr>
<td>8:00</td>
<td>Somatosensory Evoked Potentials (SEPs)</td>
</tr>
<tr>
<td></td>
<td>Ronald Emerson, MD</td>
</tr>
</tbody>
</table>

**Neonatal EEG**

7:00 — 8:30 AM  
International C, 6th floor  
*Course Director: Nicholas S. Abend, MD*

**Objectives:**
At the conclusion of this activity, participants will be able to:

1. Identify neonatal electrographic seizures using scalp EEG and differentiate seizures from non-ictal EEG patterns.

2. Incorporate neonatal EEG findings into prognostic models to predict outcome in high risk neonates.

3. Select appropriate evidence-based treatment for neonatal seizures.

**Agenda:**

<table>
<thead>
<tr>
<th>Time</th>
<th>Activity</th>
</tr>
</thead>
<tbody>
<tr>
<td>7:00</td>
<td>Neonatal EEG Background - Overview and ACNS Terminology</td>
</tr>
<tr>
<td></td>
<td>Dennis Dlugos, MD</td>
</tr>
<tr>
<td>7:30</td>
<td>EEG Background and Prognosis</td>
</tr>
<tr>
<td></td>
<td>Courtney J. Wusthoff, MD</td>
</tr>
<tr>
<td>8:00</td>
<td>Sleep Assessment and Implications</td>
</tr>
<tr>
<td></td>
<td>Mark Scher, MD</td>
</tr>
</tbody>
</table>
Intracranial EEG
9:00 AM – 5:00 PM
International C, 6th floor
Course Co-Directors: Greg Worrell, MD and Donald L. Schomer, MD

Objectives:
At the conclusion of this activity, participants will be able to:
1. Discuss the appropriate indications for and limitations of intracranial EEG in patients with drug-resistant epilepsy and recurrent focal seizures.
2. Design a comprehensive plan for invasive EEG monitoring, including electrode type, electrode placement, minimization of risks, and discussion of risks and potential benefits with patients.
3. Evaluate the results of intracranial EEG monitoring, including identification of the epileptogenic zone and of nearby eloquent cortex, to develop a surgical plan most likely to result in seizure freedom and minimize surgical risks.
4. Incorporate new EEG analysis techniques into presurgical evaluations, such as analysis of infra-slow and high frequency EEG activity, to improve identification of the epileptogenic zone and to gain new research insights into normal and pathological brain function.

Agenda:
9:00 AM Intro/Overview
Donald Schomer, MD
9:15 AM Phase 1 Evaluations that Lead to Phase 2 Testing
Donald Schomer, MD
9:45 AM Quick Presentation of the Phase I Evaluation in 3 Cases
Michael Sperling, MD
10:15 AM Choosing Phase 2 Electrodes
Stephan Schuele, MD, MPH
10:45 AM Break
11:00 AM Discussion and Demonstrations of Patterns of Seizure Onsets & Spread, Underlying Pathological Substrates, Surgical Outcomes in Adults and Children
Michael Sperling, MD
11:30 AM Discussion and Demonstrations of Patterns of Seizure Onsets & Spread, Underlying Pathological Substrates, Surgical Outcomes in Adults and Children
Tobias Loddenkemper, MD
12:00 PM Lunch (on own, see p. 7 for nearby dining)
1:00 PM The Use on Invasive Electrodes to Map “Epileptic Zones”
William Stacey, MD PhD
1:45 PM The Use on Invasive Electrodes to Map Function
Tobias Loddenkemper, MD
2:30 PM Wideband Intracranial EEG and Localization
Greg Worrell, MD
3:00 PM Break
3:15 PM Lessons Regarding Normal Physiology and Physiology of Epilepsy through IEEG
Sydney Cash, MD PhD
4:15 PM Presentation of Two “Difficult Cases” One Child, One Adult
4:45 PM Summary

Intraoperative Monitoring Part II
9:00 AM – 5:00 PM
International DE, 6th floor
Course Co-Directors: Jamie R. Lopez, MD and Michael L. McGarvey, MD

Objectives:
At the conclusion of this activity, participants will be able to:
1. Employ a thorough understanding of neuroanatomy and neurophysiology to identify risks for injury to the brain, spine, and cranial and peripheral nerves during surgical and other invasive procedures, and to select appropriate monitoring techniques to minimize these risks.
2. Design a comprehensive monitoring plan for individual patients, including multimodality intraoperative monitoring techniques (e.g., recordings of sensory and motor evoked potentials, EEG, EMG, and spinal reflex activity) to monitor segments of the nervous system at risk during surgery.
3. Recognize changes in intraoperative neurophysiologic tests which indicate damage to neural structures, and distinguish these from common technical artifacts.
4. Communicate normal and abnormal results to the surgical team, and incorporate results into clinical recommendations that may alter the surgical technique to avoid, limit or reverse injury to neural structures.
5. Identify the effects of anesthetic drugs on neurophysiology and employ methods to limit the adverse impact of anesthetics on intraoperative monitoring techniques.

Agenda:
9:00 AM Monitoring Cerebral and Spinal Endovascular Procedures
Viet Nguyen, MD
9:40 AM Electrocorticography During Pediatric Epilepsy Surgery
Andrew Kim, MD
10:20 AM Break
10:35 AM Mapping of Cortical and Subcortical Brain Structures
Mirela Simon, MD, MSc
11:15 AM Regulatory, Medical-Legal, and Coding/Billing Issues
Marc Nuwer, MD, PhD
11:55 AM Panel Discussion
12:10 PM Lunch (on own, see p. 7 for nearby dining)
1:10 PM Monitoring of Spinal D-Waves
Eva Ritzl, MD
1:50 PM Monitoring of Motor Cranial Nerves and Cranial Nerve Nuclei
Jaime Lopez, MD
2:30 PM Evidenced Based Studies in IOM
Jonathan Edwards, MD
3:10 PM Break
3:25 PM Troubleshooting During IOM
Brett Netherton, MS, FASNM, CNIM
4:05 PM Case Presentations and Discussion
4:45 PM Panel Discussion
EMG and EEG Technology
7:00 – 8:30 AM
International B, 6th floor
Course Co-Directors: Charles Epstein, MD and Susan T. Herman, MD

Objectives:
At the conclusion of this activity, participants will be able to:
1. Describe the fundamental operation of neurophysiologic recording equipment, including differential amplifiers, common-mode noise rejection, grounds, and filters.
2. Explain the concepts of analog-to-digital conversion, aliasing and general frequency analysis.
3. Evaluate and select neurophysiologic equipment based on knowledge of appropriate technical specifications for clinical or research use.

Faculty:
Charles Epstein, MD
Susan T. Herman, MD

Updates in the Business of Neurophysiology: From Washington to Your Office
7:00 – 8:30 AM
International C, 6th floor
Course Co-Directors: Yafa Minazad, DO and Deborah Briggs, MD

Objectives:
At the conclusion of this activity, participants will be able to:
1. Plan and develop an interdisciplinary team for neurophysiological practice in the neurophysiology laboratory, video-EEG monitoring unit, intensive care unit and operating room.
2. Apply business principles, quality assurance, and efficiency guidelines to neurophysiologic practice to improve clinical effectiveness and cost effectiveness of patient care.
3. Incorporate risk minimization strategies into neurophysiology practice, particularly in regards to invasive and remote monitoring.

Agenda:
7:00 AM Innovation and Integration: Neurophysiologist - Hospitalist
Yafa Minazad, DO

7:20 AM Working with Your Local Hospital in Becoming a Designated Epilepsy Center
Deborah Briggs, MD

7:40 AM Medicare Hospital Outpatient PPS Proposed Rule for 2014 Update
Marc R. Nowee, MD, PhD

8:00 AM ObamaCare and it’s Impact on Neurophysiology
Lynn Elizabeth Mullikin, MPA, FACHE, FASET

ICU EEG
9:00 AM – 5:00 PM
International DE, 6th floor
Course Co-Directors: Lawrence J. Hirsch, MD and Cecil D. Hahn, MD, MPH

Objectives:
At the conclusion of this activity, participants will be able to:
1. Discuss current guidelines and evaluate various practice models for ICU EEG monitoring to improve patient care.
2. Apply the revised ACNS nomenclature to ICU EEG recordings, to improve standardization of ICU EEG reports and communication between providers.
3. Recognize controversial EEG patterns in ICU patients with altered mental status, and formulate a rational plan for treatment based on these EEG patterns.
4. Develop a comprehensive ICU EEG monitoring program, including equipment selection, training of interdisciplinary staff, quality improvement, and risk management.

Agenda:
9:00 AM Overview of ICU EEG Monitoring in Neonates, Children and Adults
Nicholas Abend, MD

9:30 AM Q & A, Discussion

9:40 AM Guidelines and Nomenclature for ICU EEG Monitoring
Lawrence Hirsch, MD

10:10 AM Q & A, Discussion

10:20 AM Break

10:35 AM The Ictal-interictal Continuum: Case Studies
Suzette LaRoche, MD

11:05 AM Q & A, Discussion

11:10 AM EEG Monitoring in the Medical and Surgical ICUs
Emily Gilmore, MD

11:40 AM Case Presentation
Nicolas Gaspard, MD, PhD

12:00 PM Lunch (on own, see p. 7 for nearby dining)

1:00 PM Quantitative EEG for Seizure Identification
Cecil Hahn, MD, MPH

1:30 PM Q & A, Discussion

1:40 PM Treatment of Nonconvulsive Seizures and Status Epilepticus
Aatif Husain, MD

2:10 PM Case Presentation

2:25 PM Break

2:40 PM The Business of ICU EEG Monitoring
Stephen Hantus, MD

3:10 PM Q & A, Discussion

3:15 PM Ischemia Detection
M. Brandon Westover, MD PhD

3:45 PM Case Presentation
Sarah Schmitt, MD
ANNUAL COURSES PROGRAM

Thursday, February 6, 2014

<table>
<thead>
<tr>
<th>Time</th>
<th>Course</th>
<th>Location</th>
<th>Course Co-Directors</th>
</tr>
</thead>
<tbody>
<tr>
<td>4:00 PM</td>
<td>ICU EEG Reading Session: Neonatal, Pediatric and Adult Cases</td>
<td></td>
<td>Courtney Wusthoff, MD</td>
</tr>
<tr>
<td>4:20 PM</td>
<td>ICU EEG Reading Session: Neonatal, Pediatric and Adult Cases</td>
<td></td>
<td>William Gallentine, DO</td>
</tr>
<tr>
<td>4:40 PM</td>
<td>ICU EEG Reading Session: Neonatal, Pediatric and Adult Cases</td>
<td></td>
<td>Jong Woo Lee, MD, PhD</td>
</tr>
</tbody>
</table>

**EMG**

9:00 AM – 12:00 PM
International B, 6th floor
Course Co-Directors: Devon Rubin, MD and Francis O. Walker, MD

Objectives:
At the conclusion of this activity, participants will be able to:
1. Apply basic and advanced EMG techniques to diagnose common entrapment neuropathies.
2. Incorporate advances in electrodiagnostic techniques and avoid technical pitfalls in evaluation of radiculopathies and plexopathies.
3. Recognize characteristic EMG patterns of neuropathic and myopathic disorders and interpret the clinical significance to improve neurologic diagnosis.

Agenda:
9:00 AM Assessment of Common Entrapment Neuropathies with EMG and Ultrasound
Francis O. Walker, MD
10:00 AM Assessment of Radiculopathies and Plexopathies - EDX Approaches and Limitations
Daniel L. Menkes, MD
11:00 AM Improving EMG Waveform Recognition Skills — Identifying Unknown Waveforms
Devon Rubin, MD

**VIDEO-EEG**

9:00 AM – 12:00 PM
International C, 6th floor
Course Co-Directors: William O. Tatum, DO and Tobias Loddenkemper, MD

Objectives:
At the conclusion of this activity, participants will be able to:
1. Describe the technical requirements for optimal video-EEG monitoring in inpatient and outpatient settings.
2. Recognize the electroencephalographic and clinical features of seizures and nonepileptic events in adults and children commonly encountered in the video-EEG monitoring unit.
3. Translate EEG and video interpretations into clinical reports which accurately describe diagnosis, seizure localization, and implications for patient management, including candidacy for epilepsy surgery.
4. Determine the localization of seizure onsets based on combined video and intracranial EEG recordings.

Agenda:
9:00 AM The Essentials of Video-EEG
Michael R. Sperling, MD
9:30 AM Applying Video - EEG in Practice (Focus on Pediatric Patients)
Tobias Loddenkemper, MD
10:00 AM Video - EEG Pearls - A Case-Based Approach (Focus on Adults)
William O. Tatum, DO
10:30 AM Relevance of Patient Monitoring - Sudden Unexpected Death in Epilepsy
Stephan U. Schuele, MD, MPH
11:00 AM Video - EEG Monitoring in the Home and Beyond — New Approaches
Steven Schachter, MD
ANNUAL COURSES PROGRAM

Thursday, February 6, 2014

Autonomic Neurophysiology. Spells of Dysautonomia: From Epilepsy to West Nile Virus.
1:00 – 2:30 PM
International B, 6th floor
Course Director: A. Arturo Leis, MD

Objectives:
At the conclusion of this activity, participants will be able to:
1. Recognize and formulate a differential diagnosis of the clinical disorders that may present with acute spells of dysautonomia or autonomic emergencies, including traumatic brain injury, epilepsy, botulism, Lambert-Eaton myasthenic syndrome (LEMS), Guillain-Barre syndrome, Miller-Fisher syndrome, West Nile virus infection, and spinal cord injury.
2. Direct appropriate diagnostic evaluation and treatment of these clinical disorders.
3. Improve ability to communicate with and educate patients, families, and other healthcare colleagues about the clinical features of acute autonomic nervous system dysfunction.

Agenda:
1:00 PM Introduction
A. Arturo Leis, MD
1:05 PM Peripheral Nervous System Spells of Dysautonomia.
A. Arturo Leis, MD
1:45 PM Central Nervous System Spells of Dysautonomia.
TBD
2:25 PM Questions and Discussion

Case Studies in Peripheral Neurophysiology
2:30 – 5:00 PM
International DE, 6th floor
Course Director: Elliot Dimberg, MD

Objectives:
At the conclusion of this activity, participants will be able to:
1. Interpret patterns of clinical neurophysiological findings in peripheral nervous system disease; and
2. Appropriately localize neuromuscular abnormalities according to the neurophysiological findings.

Faculty:
Elliot Dimberg, MD
Christopher S. Nance, MD
Randa Jarra, MD

Learn more about the latest advancements and technologies.

Join us in the Evening Programs…

5:30 – 7:30 PM | International B and C, 6th floor

ACNS is pleased to introduce the inaugural Evening Programs. Beverages and snacks will be served. CME credits are NOT available for the Evening Programs.

See page 75 for more information.
ANNUAL MEETING PROGRAM

Friday, February 7, 2014

Poster Viewing & Continental Breakfast
7:00 – 8:00 AM
200 Peachtree, 7th floor

Opening Ceremony
7:55 – 10:00 AM
Atlanta Ballroom, 7th floor
Chairs: Suzette M. LaRoche, MD and Greg Worrell, MD

7:55 – 8:00 AM Welcome
8:00 – 8:45 AM Presidential Lecture
Introduction:
Suzette M. LaRoche, MD
History of the American Clinical Neurophysiology Society's Furthering of the Understanding of Status Epilepticus
Frank W. Drislane, MD
8:45 – 9:20 AM 2014 Robert S. Schwab Award & Lecture
David Burke, MD, DSc
9:20 – 10:00 AM 2013 Pierre Gloor Award & Lecture
Rodolfo Llinas, MD, PhD

Coffee Break - Visit Exhibits and Posters
10:00 – 10:30 AM
200 Peachtree, 7th floor

10:30 AM – 12:30 PM CONCURRENT SESSIONS

Pediatric EMG in the Molecular Era
Augusta Ballroom, 7th floor
Chair: Ioannis Karakis, MD, MSc

Objectives:
At the conclusion of this session, participants will be able to:
1. Identify the most common indications for ordering and performing pediatric EMG; and
2. Illustrate how to use EMG findings in the diagnosis of disorders of the muscles, nerves and neuromuscular junction in childhood.

Agenda:
10:30 AM Current Practice and Temporal Trends of Pediatric EMG in the New Millennium
Ioannis Karakis, MD, MSc
11:00 AM Chronic Inflammatory Demyelinating Polyneuropathies in Childhood
Hugh J. McMillan, MD, MSc
11:30 AM Myotonia in Childhood
Peter Kang, MD
12:00 PM Pediatric Neuromuscular Junction Disorders and the Use of Stimulated SFEMG
Matthew Pitt, MD

Intraoperative Neurophysiological Monitoring During Skull Base Surgeries
International DE, 6th floor
Chair: Parthasarathy Thirumala, MD

Objectives:
At the conclusion of this session, participants will be able to:
1. Describe the use of Intraoperative Neurophysiological Monitoring during skull base surgeries;
2. Explain the various modalities utilized during skull base surgeries; and
3. Identify the limitations of the use of SSEP, EMG, and BAEPs during skull base surgeries.

Agenda:
10:30 AM Brainstem Auditory Evoked Potentials during Microvascular Decompression
Parthasarathy Thirumala, MD
11:00 AM Facial Nerve and Auditory Nerve Monitoring during CP Angle Tumor Removal
Aatif M. Husain, MD
11:30 AM Intraoperative Monitoring During Endoscopic Endonasal Procedures
Parthasarathy Thirumala, MD
12:00 PM Somatosensory Evoked Potentials and Arm Positioning Related Changes during Skull Base Surgeries
Ronald Emerson, MD

Wide Bandwidth Electrophysiology and Epilepsy Biomarkers
Atlanta Ballroom, 7th floor
Chair: Greg Worrell, MD

Objectives:
At the conclusion of this session, participants will be able to:
1. List recent advances in wide bandwidth intracranial EEG recordings;
2. Describe the concept of electrophysiological biomarkers and role in epilepsy diagnosis and treatment;
3. Restate the cellular origins of neuronal oscillations (physiological and pathological);
4. Summarize recent advances in quantitative electrophysiology applied to intracranial EEG; and
5. Discuss the role of interictal biomarkers in epilepsy surgery.

Agenda:
10:30 AM Introduction & Electrophysiological Biomarkers
Greg Worrell, MD
11:00 AM International Electrophysiology Database and Collaborative Research
Brian Litt, MD
12:00 PM Origin of Pathological and Physiological Oscillations
William Stacey, MD, PhD
5. Identify characteristic EEG patterns in patients with postanoxic encephalopathy which signal potential for recovery from versus poor long-term neurological prognosis.

Agenda:

1:30 PM  cEEG Monitoring in Subarachnoid Hemorrhage: Current Practices and Limitations
          Nicolas Gaspard, MD, PhD

1:54 PM  The EEG of Cerebral Ischemia
          Jan Claassen, MD, PhD

2:18 PM  Evolution of EEG Patterns after Global Cerebral Ischemia
          Michel Van Putten, MD

2:42 PM  Emerging Methods for cEEG Ischemia Detection in Subarachnoid Hemorrhage
          Eric Rosenthal, MD

3:06 PM  The EEG of Cerebral Ischemia
          Brandon P. Foreman, MD and Jan Claassen, MD, PhD

---

Botulinum Toxin: Mechanism of Action and Ultrasound Versus EMG Guidance
Augusta Ballroom, 7th floor
Chair: Francis O. Walker, MD

Objectives:
At the conclusion of this session, participants will be able to:
1. Describe how botulinum toxin improves dystonia and spasticity;
2. List limitations of injection guidance by palpation; and
3. Compare and contrast EMG, electrical stimulation, and ultrasound guidance for administering therapeutic injections of botulinum toxin.

Agenda:

1:30 PM  The Clinical Neuropharmacology and Neurophysiology of Botulinum Toxin
          Francis O. Walker, MD

3:30 PM  Ultrasound Guidance of Botulinum Toxin Therapy
          Katharine Alter, MD

5:30 PM  EMG and Electrical Stimulation Guidance for Botulinum Toxin
          Jaime R. Lopez, MD

---

Ischemia Monitoring in Critical Care: EEG Trend Analysis to Detect Development of and Recovery from Cerebral Ischemia
Augusta Ballroom, 7th floor
Chair: M. Brandon Westover, MD, PhD

Objectives:
At the conclusion of this session, participants will be able to:
1. Describe the systematic progression of EEG changes that occur in response to increasing levels of cerebral ischemia, and relate these changes to the probability of irreversible ischemic injury;
2. Discuss the possible pathophysiological mechanisms thought to underlie delayed cerebral ischemia after acute brain injury, including subarachnoid hemorrhage and traumatic brain injury;
3. Summarize current methods and limitations of ischemia monitoring, including methods of serial visual analysis and QEEG trend monitoring;
4. List the special considerations which apply to ischemia monitoring outside the setting of subarachnoid hemorrhage; and

Coffee Break - Visit Exhibits and Posters
3:30 – 4:00 PM
200 Peachtree, 7th floor
CONCURRENT SESSIONS

Spasticity - What is it and What is the Electrophysiology?
Augusta Ballroom, 7th floor
Chair: Morris Fisher, MD

Objectives:
At the conclusion of this session, participants will be able to:
1. Recognize the complexities associated with using spasticity as a clinical descriptor;
2. Express the range of research methodologies used to investigate these motor system abnormalities; and
3. Identify research that is necessary to enhance the management of patients with spasticity.

Agenda:
4:00 PM Spasticity - A Historical and Clinical Conundrum
    Morris Fisher, MD
4:30 PM Investigative Approaches to Characterizing Spasticity
    W. Zev Rymer, MD
5:00 PM Ion Channel and F-Wave Findings in Patients with Strokes
    Cliff Klein, MD PhD

Spikes and Cognition: To Treat or Not to Treat?
Atlanta Ballroom, 7th floor
Chair: Tobias Loddenkemper, MD

Objectives:
At the conclusion of this session, participants will be able to:
1. Review clinical presentation and systematic diagnostic approaches to clinical, neurophysiological and neuropsychological outcome assessment in patients with frequent spiking;
2. Understand the electrophysiological assessment of frequent spiking and non-convulsive SE syndromes;
3. Discuss potential mechanisms and pathophysiology of sleep potentiated spiking in relationship to memory; and

Agenda:
4:00 PM Electrophysiological Assessment of Frequent Spiking and Non-Convulsive SE Syndromes
    Iván Sánchez Fernández, MD
4:25 PM Relationship between Spiking and Cognition
    David Loring, MD, PhD
4:45 PM Pathophysiology and Mechanisms of Frequent Spiking, Sleep, Memory, and Cognition
    Tobias Loddenkemper, MD
5:05 PM Should We Treat Spikes?
    Kevin Chapman, MD

Advanced Practice Technologists in the New World of Continuous Neurophysiological Monitoring
International DE, 6th floor
Co-Chairs: Joshua Ehrenberg, BS, R. EEG T., CNIM and Suzette M. LaRoche, MD

Objectives:
At the conclusion of this session, participants will be able to:
1. Be familiar with the education, experience, and credentials required for advanced practice technologists;
2. Describe the role of advanced practice technologists in the operating room; and
3. Describe the role of advanced practice technologists in continuous EEG monitoring.

Agenda:
4:00 PM Education, Knowledge, and Credentials for Advanced Practice Technologists
    Joshua Ehrenberg, BS, R. EEG T., CNIM
4:22 PM Advanced Practice in the OR
    Brett Netherton, MS, CNIM
4:45 PM Utilization of Advanced Practice in EEG Monitoring
    Sherry Nehamkin, R. EEG/EP T., CNIM, CLTM
5:07 PM Round Table Discussion
    Stephen Hantus, MD

Neurophys Bowl
5:30 — 6:45 PM
Atlanta Ballroom, 7th floor

Welcome Reception
6:45 – 8:00 PM
200 Peachtree, 7th floor

*SIG = Special Interest Group
ANNUAL MEETING PROGRAM

Saturday, February 8, 2014

Poster Viewing & Continental Breakfast
7:00 – 8:00 AM
200 Peachtree, 7th floor

Plenary Session
8:00 – 10:10 AM
Atlanta Ballroom, 7th floor
Chair: Frank W. Drislane, MD

8:00 – 8:30 AM 2014 Pierre Gloor Award & Lecture
Introduction
Aatif M. Husain, MD
All Waves That Glitter are Not Gold — Lessons of the Penumbra and the Core
Ronald Emerson, MD

8:30 – 8:40 AM 2014 Herbert H. Jasper Award Presentation to Ernst Rodin, MD
Introduction
Greg Worrell, MD

8:40 – 8:50 AM Travel Fellows’ Recognition Ceremony
Suzette M. LaRoche, MD and Greg Worrell, MD

8:50 – 10:10 AM Seizures, SUDEP and Autonomic Nervous System
Chair: Ekrem Kutluay, MD

Objectives:
At the conclusion of this session, participants will be able to:
1. Differentiate between ictal and interictal changes during and after epileptic seizures; and
2. Summarize the existing theories leading to SUDEP and the possible role of autonomic nervous system dysfunction; and
3. Recognize the various neurophysiological tests that measure the integrity and function of different parts of the autonomic nervous system.

Agenda:
8:50 AM Epilepsy and Autonomous Nervous System
Ekrem Kutluay, MD
9:20 AM Role of Autonomic Dysfunction in SUDEP
Lawrence J. Hirsch, MD
9:50 AM Neurophysiological Testing of Autonomous Nervous System
Safwan Jaradeh, MD

Coffee Break - Visit Exhibits and Posters
10:10 – 10:30 AM
200 Peachtree, 7th floor

10:30 AM – 12:30 PM CONCURRENT SESSION

EEG as a Basic Neuroscience and Psychology Research Tool
Atlanta Ballroom, 7th floor
Chair: Joshua Ewen, MD

Objectives:
At the conclusion of this session, participants will be able to:
1. Understand how EEG can be used in basic and clinical neuroscience and cognitive psychology to evaluate models of normal and pathological brain function; and
2. Recognize the steps required for novel observations of EEG phenomena to be developed and validated as clinical tests in neurological disorders.

Agenda:
10:30 AM ERPs from Basic to Clinical Research: Toward Biomarkers
Greg Hajcak Proudfit, PhD
11:10 AM Use of EEG and MEG in Studying Oscillatory in Normal and Pathological Brain States
Michael Gandal, MD
11:50 AM Development of Biomarkers and Clinical Tests from Basic EEG Research
Joshua Ewen, MD

Intraoperative Neurophysiologic Monitoring During Functional Neurosurgery
Augusta Ballroom, 7th floor
Chair: Jay L. Shils, MD, PhD

Objectives:
At the conclusion of this session, participants will be able to:
1. Describe the various neurophysiologic methods used during neuromodulation procedures;
2. Recognize the critical decision points in various neuromodulation surgeries;
3. Name the basic principles behind neuromodulation therapies; and
4. Give examples of the relation between intra-operative neurophysiologic data and post-operative management of the patients.

Agenda:
10:30 AM Post-Operative Management of the Neuromodulation Patient
Mark Stecker, MD, PhD
11:30 AM Neurosurgical Decisions during Placement of Neuromodulation Devices
Peter Dempsey, MD
### Amplitude-Integrated EEG in Neonates: When is it Used and When is it Useful?

**International DE, 7th floor**  
**Chair:** Courtney J. Wusthoff, MD

**Objectives:**  
At the conclusion of this session, participants will be able to:  
1. Describe the principles of amplitude-integrated EEG, including similarities and differences between aEEG and conventional EEG acquisition and display;  
2. Differentiate between normal and abnormal patterns on neonatal aEEG; and  
3. Evaluate the benefits and limitations of aEEG monitoring in critically ill newborns.

<table>
<thead>
<tr>
<th>Time</th>
<th>Agenda</th>
</tr>
</thead>
<tbody>
<tr>
<td>10:30 AM</td>
<td>Principles of aEEG</td>
</tr>
<tr>
<td></td>
<td>James John Riviello, MD</td>
</tr>
<tr>
<td>11:00 AM</td>
<td>Interpretation of aEEG</td>
</tr>
<tr>
<td></td>
<td>Courtney J. Wusthoff, MD</td>
</tr>
<tr>
<td>11:30 AM</td>
<td>Current Applications of aEEG in the NICU</td>
</tr>
<tr>
<td></td>
<td>Tammy Tsuchida, MD, PhD</td>
</tr>
<tr>
<td>12:00 PM</td>
<td>Impact of aEEG Use in the NICU</td>
</tr>
<tr>
<td></td>
<td>Cecil D. Hahn, MD, MPH</td>
</tr>
</tbody>
</table>

**Boxed Lunch**  
Visit Exhibits & Posters  
Poster Tour  
12:30 — 2:00 PM  
200 Peachtree, 7th floor

**Professional Development Mentoring Program**  
12:30 — 2:00 PM  
Please pick up a lunch in the Exhibit Hall and proceed to International B, 7th floor

### Extending Critical Care EEG Monitoring to Community-Based Practice (SIG)

**Atlanta Ballroom, 7th floor**  
**Chair:** Evan Fertig, MD

**Objectives:**  
At the conclusion of this session, participants will be able to:  
1. Create a proposal for a new community-based ICU monitoring program to present to hospital administration, including how to write a business plan;  
2. Explain how to set up and manage a program on a daily basis, including selection of equipment, hiring, scheduling and communication with colleagues;  
3. Recognize potential barriers, including resistance from colleagues to change clinical practice, time constraints, and medico-legal issues; and  
4. Describe how to create the framework to conduct clinical research.

<table>
<thead>
<tr>
<th>Time</th>
<th>Agenda</th>
</tr>
</thead>
<tbody>
<tr>
<td>12:50 PM</td>
<td>Mixing General Neurology and ICU Monitoring</td>
</tr>
<tr>
<td></td>
<td>Jennifer Jones, DO</td>
</tr>
<tr>
<td>1:10 PM</td>
<td>Starting an ICU Monitoring Program in a Community Hospital</td>
</tr>
<tr>
<td></td>
<td>Evan Fertig, MD</td>
</tr>
<tr>
<td>1:30 PM</td>
<td>Business Aspects of ICU Monitoring</td>
</tr>
<tr>
<td></td>
<td>Yafa Minazad, DO</td>
</tr>
</tbody>
</table>

### Semiology of Status Epilepticus in Adults

**Atlanta Ballroom, 7th floor**  
**Chair:** Jeffrey Kennedy, MD

**Objectives:**  
At the conclusion of this session, participants will be able to:  
1. Discuss and utilize clearly defined descriptive terminology for semiology of status epilepticus;  
2. Utilize descriptive semiological terminology to aid electro-clinical diagnosis of status epilepticus; and  
3. Apply semiological terminology in clinical practice to assist in defining specific syndromes underlying status epilepticus.

<table>
<thead>
<tr>
<th>Time</th>
<th>Agenda</th>
</tr>
</thead>
<tbody>
<tr>
<td>2:00 PM</td>
<td>Standardized Terminology for Status Epilepticus</td>
</tr>
<tr>
<td></td>
<td>Stephan U. Schuele, MD, MPH</td>
</tr>
<tr>
<td>2:30 PM</td>
<td>Semiology of Status Epilepticus in the Responsive Patient</td>
</tr>
<tr>
<td></td>
<td>Frank W. Drislane, MD</td>
</tr>
<tr>
<td>3:00 PM</td>
<td>Correlation of Semiology and EEG Pattern in Comatose Patients</td>
</tr>
<tr>
<td></td>
<td>Peter W. Kaplan, MD, FRCP</td>
</tr>
<tr>
<td>3:30 PM</td>
<td>Impact of Semiology on the Risk for Status Epilepticus and Long-Term Clinical Outcome</td>
</tr>
<tr>
<td></td>
<td>Stephen Hantus, MD</td>
</tr>
</tbody>
</table>

**2:00 PM — 4:00 PM**  
**Concurrent Session**

---

*SIG—Special Interest Group*
**Intraoperative Neuromonitoring Below the Belt**

*International DE, 6th floor*

**Chair: Stanley Skinner, MD**

At the conclusion of this session, participants will be able to:
1. Interpret anal sphincter and cremaster electromyography, the bulbocavernosus reflex, and pudendal SEPs;
2. Explain the NIOM problems presented by pelvic autonomic neuroanatomy and neurophysiology;
3. Describe current and possible future autonomic neuromonitoring approaches (corpus cavernosum electromyography, for example);
4. Practice multimodality neuromonitoring during complex spinal cord untethering; and
5. Recognize signal change, report neural topographic data, and discuss context-specific interventions in the event of signal change in tethered cord cases.

**Agenda:**
2:00 PM  
Bulbocavernous Reflex; Anal Sphincter and Cremaster Electromyography; Pelvic Autonomic Monitoring  
*Stanley Skinner, MD*

2:40 PM  
Pudendal Nerve and Sacral Root Evoked Potentials  
*Matthew Eccher, MD*

3:05 PM  
Neuophysiology of Complex Spinal Cord Untethering  
*Francesco Sala, MD, PhD*

3:45 PM  
Q&A: Discussion Panel  
*Francesco Sala, MD, PhD  
Stanley Skinner, MD  
Matthew Eccher, MD*

**Electrophysiological Approach to Neuromuscular Disorders**

*Augusta Ballroom, 7th floor*

**Chair: Suraj Muley, MD**

**Objectives:**
At the conclusion of this session, participants will be able to:
1. Recognize the clinical and diagnostic importance of changes in motor and sensory amplitudes during nerve conduction studies;
2. Explain the importance of various forms of abnormal spontaneous activity during electromyography;
3. Outline the clinical significance of size of motor units;
4. Describe the clinical implications of myotonia seen during electromyography; and
5. Discuss the diagnostic implications of conduction slowing during nerve conduction studies.

**Agenda:**
2:00 PM  
Segmental Amplitude Change  
*Mark Ross, MD*

2:20 PM  
Fibrillations, PSWs, Myokymia and CRDs  
*Brent Goodman, MD*

2:40 PM  
Small and Large Motor Units  
*Brent Goodman, MD*

3:00 PM  
Myotonia  
*Shaheeq Ladha, MD*

3:20 PM  
Conduction Slowing  
*Erik Ortega, MD*

**Coffee Break**
4:00 – 4:30 PM  
Atlanta Ballroom Foyer

**4:30 PM – 6:00 PM SPECIAL INTEREST GROUPS**

**Critical Care EEG Monitoring and Outcomes: Do We Have Enough Data? (SIG)**

*Atlanta Ballroom, 7th floor*

**Co-Chairs: Nicholas S. Abend, MD and Elizabeth Gerard, MD**

**Objectives:**
At the conclusion of this session, participants will be able to:
1. Describe the indications for continuous EEG monitoring in critically ill patients;
2. Evaluate the available evidence that continuous EEG monitoring impacts patient care or outcomes and define areas that require further research; and
3. Apply data regarding continuous EEG monitoring to their own practice including recognizing patients for whom EEG monitoring is indicated and determining the appropriate duration of monitoring for each patient.

**Agenda:**
4:30 PM  
cEEG in the Adult ICU (Seizure Indications)  
*Jeffrey Kennedy, MD*

4:48 PM  
Update of Current Practices  
*Jay Gavvala, MD*

5:06 PM  
cEEG in the Adult ICU (Non-Seizure Indications)  
*Jan Claassen, MD, PhD*

5:24 PM  
cEEG in the Neonatal ICU  
*Courtney J. Wusthoff, MD*

5:42 PM  
cEEG in the Pediatric ICU  
*Cecil Hahn, MD, MPH*

*SIG=Special Interest Group*
## Intraoperative Neurophysiologic Monitoring (SIG)

**International DE, 6th floor**  
*Chair: Jaime R. Lopez, MD*

**Objectives:**
At the conclusion of this session, participants will be able to:
1. Describe the proper method for coding of IOM time;
2. Distinguish the important IOM issues that overlap different medical and allied health societies; and
3. State the American Clinical Neurophysiology Society’s role in advocating and promoting IOM.

**Agenda:**
- **4:30 PM**  
  Cervical surgery and C5 palsy: IONM or not to IONM? And How? Evidence-Based?  
  *Viet Nguyen, MD*

- **5:05 PM**  
  Quantifying EMG and Critical Alert Criteria  
  *Vita Kesner, MD*

- **5:35 PM**  
  Panel Discussion

- **5:45 PM**  
  SIG Open Forum

---

## EMG (SIG)

**Augusta Ballroom, 7th floor**  
*Chair: Suraj Muley, MD*

**Objectives:**
At the conclusion of this session, participants will be able to:
1. Recognize the importance of motor conduction block in the diagnosis of multifocal motor neuropathy;
2. Distinguish whether electrophysiological studies can aid in determination of prognosis in Guillain Barre syndrome;
3. Discuss usefulness of skin biopsy in diagnosis of small fiber neuropathy; and
4. Describe the role of ultrasound in diagnosis of entrapment neuropathy.

**Agenda:**
- **4:30 PM**  
  Conduction Block is Essential for Diagnosis of MMN  
  *Michael Cartwright, MD*

- **4:50 PM**  
  Changes of Axonal Degeneration Imply a Poor Prognosis of Guillain Barre Syndrome  
  *Mark Ross, MD*

- **5:10 PM**  
  Skin Biopsy is the Best Diagnostic Test for Assessment of Small Fiber Neuropathy  
  *Morris Fisher, MD*

- **5:30 PM**  
  Ultrasound Improves Sensitivity in the Diagnosis of Entrapment Neuropathy  
  *Francis O. Walker, MD*

---

**Annual Business Meeting**

6:00 — 7:00 PM  
*Atlanta Ballroom, 7th floor*
ANNUAL MEETING PROGRAM

Sunday, February 9, 2014

Continental Breakfast
7:15 – 8:00 AM
Atlanta Ballroom Foyer

8:00 AM – 10:00 AM  CONCURRENT SESSIONS

Fast-Train Cortical and Sub-Cortical Stimulation for Motor Mapping and Monitoring
International D, 6th floor  
Chair: Matthew Eccher, MD

Objectives:
At the conclusion of this session, participants will be able to:
1. Describe the parameters of stimulation for direct brain delivery of MEP stimulation;
2. Recognize the application of MEP Stimulation for cortical motor mapping, subcortical motor tract proximity assessment, and motor trace monitoring;
3. Perform the technical setup for performance of these techniques;
4. Explain the application of the techniques necessary for cerebral hemispheric surgery of pericentral lesions; and
5. Identify advantages, disadvantages, and limitations of current techniques utilized for monitoring epilepsy surgery.

Agenda:
8:00 AM Intracranial MEP Stimulation and Recording - Practical Setup, Pearls and Pitfalls
   Tyson Hale, MD
8:30 AM Cortical and Sub-Cortical MEP Stimulation
   Stuart Hoffman, DO
9:00 AM DCS-MEP for Epilepsy Surgery: Current Evidence and Questions
   Matthew Eccher, MD
9:30 AM DCS-MEP and CST Motor Threshold: Utility for Cerebral Hemispheric Lesion Surgery
   Kathleen Seidel, MD

Diagnostic Advances in ALS
International E, 6th floor
Chair: Francis O. Walker, MD

Objectives:
At the conclusion of this session, participants will be able to:
1. Describe the differential diagnosis of ALS and its laboratory work up;
2. Interpret NCS and EMG findings in a typical patient with the disorder and discuss how to select which nerves and muscles to study;
3. Diagnose the appearance of fasciculations on real-time ultrasound; and
4. Recognize advanced findings of muscle atrophy and tissue change on static ultrasound images of affected muscles in ALS.

Agenda:
8:00 AM Ultrasound in ALS
   Michael Cartwright, MD
8:40 AM Overview of the Genetics and Clinical Features of ALS
   Shafeeq Ladha, MD
9:20 AM Electrodiagnosis in ALS
   Devon Rubin, MD

Stereo Electroencephalography
Chair: Stephan U. Schuele, MD, MPH

Objectives:
At the conclusion of this session, participants will be able to:
1. Identify the rationale and technology underlying sEEG;
2. Recognize indications for sEEG; and
3. Discuss advantages and limitations of sEEG in adults and children.

Agenda:
8:00 AM Why Stereo Electroencephalography?
   Samden Lhatoo, MD
8:30 AM Surgical Approach: Concept and Technique
   Jorge Alvaro Gonzalez-Martinez, MD
9:00 AM Outcome: Early Experience in Over 200 Patients
   Juan Bulacio, MD
9:30 AM Pediatric Stereo EEG: Challenges and Opportunities
   Jonathan Miller, MD

Coffee Break
10:00 – 10:15 AM
Atlanta Ballroom Foyer
## Concurrent Sessions

### Neonatal and Pediatric EEG: Patterns of Epileptic Encephalopathies Across the Age Range (SIG)

**International D, 6th floor**  
*Co-Chairs: Tobias Loddenkemper, MD and Heather Olson, MD*

**Objectives:**  
At the conclusion of this session, participants will be able to:
1. Illustrate EEG patterns of epileptic encephalopathy in neonates and discuss changes over time in relationship to etiology and outcome;  
2. Discuss variability in infantile EEG patterns based on underlying etiology of epileptic encephalopathy, including genetic and structural causes;  
3. Analyze how background and ictal EEG patterns provide clues to underlying etiology; and  
4. Describe new insights into age related pediatric encephalopathies with a focus on cortico-thalamic circuitry and ESES/CSWS patterns in children.

**Agenda:**  
- Evolution Over Time in Neonatal Epileptic Encephalopathies  
  - Courtney J. Wusthoff, MD  
- Infantile EEG Patterns in Relationship to Genetic or Structural Etiologies  
  - Tammy Tsuchida, MD, PhD  
- EEG in ESES and CSWS  
  - Iván Sánchez Fernández, MD

### Clinical Neurophysiology Trials in the Neurointensive Care Unit: Focus on Trends

**Atlanta Ballroom, 7th floor**  
*Chair: Jong Woo Lee, MD, PhD*

**Objectives:**  
At the conclusion of this session, participants will be able to:
1. Name the necessary steps in designing and implementing a clinical neurophysiological/pharmacological trial in the neurological ICU;  
2. Identify the processes necessary in determining whether one should participate in a clinical neurophysiology trial;  
3. Describe how to successfully execute a clinical neurophysiology trial; and  
4. Summarize the most important clinical questions yet to be answered through a clinical neurophysiology trial.

**Agenda:**  
- 10:15 AM Trends: From Conception to Implementation  
  - Aatif M. Husain, MD  
- 10:45 AM Participating in a Clinical Trial  
  - Jong Woo Lee, MD, PhD  
- 11:15 AM Steps to Successful Trial Execution  
  - M. Brandon Westover, MD, PhD  
- 11:45 AM Next Steps in Clinical Neurophysiology Trials  
  - Suzette M. LaRoche, MD

---

*SIG=Special Interest Group*
INTRAOPERATIVE MONITORING PART I (9:00 AM – 5:00 PM)

BAEP Monitoring
Alan Legatt, MD, PhD

Brainstem auditory evoked potentials (BAEPs) are useful for intraoperative monitoring of the ears, the auditory nerves, and the brainstem auditory pathways up through the level of the mesencephalon; they do not assess more rostral parts of the auditory pathways. BAEPs are relatively unaffected by anesthesia, though they are affected by hypothermia. Technical aspects of auditory stimulation for recording of BAEPs will be reviewed. During BAEP monitoring, each patient serves as his/her own control. Both amplitude and latency measurements should be followed. Wave I is generated in the distal eighth nerve. Subsequent components are composites of contributions from multiple generators, but wave III predominantly reflects activity in the caudal pons and wave V predominantly reflects activity in the mesencephalon. Adverse intraoperative changes in BAEPs can be caused by technical factors (including artifacts), hypothermia, acoustic masking, and localized dysfunction within the infratentorial auditory system. Possible causes of the latter include direct mechanical or thermal injury, compromise of the blood supply to a structure, and stretch of or traction on the eighth nerve.

MEP Monitoring
Ronald Emerson, MD

Use of motor evoked potential monitoring will be reviewed, including physiology, methodology, anesthetic considerations and example cases.

EEG and Doppler Ultrasound Monitoring
Michael McGarvey, MD

This course session serves as a review of Intraoperative EEG and Transcranial Doppler Ultrasound Monitoring during surgeries which place the nervous system at risk. The utility and importance of this monitoring will be reviewed. The specific techniques will be reviewed along with the evidence supporting their use and their limitations in identifying neurologic injury in specific surgical procedures.

EMG Monitoring of Central Motor Pathways During Spine Surgery
Stanley Skinner, MD

Peripheral motor axons will depolarize if struck with sufficient energy. For a given mass, end velocity of the injurious impact to a nerve fascicle determines 1) whether or not individual axons will fire and 2) how many axons will fire within the fascicle, i.e., the amplitude of the recorded compound nerve action potential. Acutely (or chronically) injured nerves may lose the ability to accommodate; that is, they keep firing repetitively well after the stimulus. Train neurotonics (“A trains” per Romstok) are likely explained by the “breakdown” of accommodation.

Our group has seen similar phenomena upon impact to the spinal cord. The first few cases were serendipitous recordings during intramedullary dissection; the meaning of associated lower limb EMG discharges, as the surgeon worked at cervical or thoracic level, seemed obscure at first. Apparently, inadvertent spinal cord impact of sufficient energy may generate trains of descending pulses which can depolarize pools of motor neurons caudally. Similar to intended electrical spinal cord stimulation, these pulses may traverse along the corticospinal tract . . . or they may descend non-specifically via non-corticospinal tracts, causing firing of motor neurons by indirect means. We have used this phenomenon to predict (and prevent) motor conduction block.

Monitoring of Spinal Nerve Roots
Monica Islam, MD

The goal of neurophysiology monitoring has been to provide feedback during surgery regarding the integrity of at-risk neural elements. Spinal nerve roots are at risks that may not be adequately assessed by evoked potentials alone. Typical surgeries potentially impacting spinal nerve roots include spinal fusion to correct scoliosis, spine tumor resection and spinal cord untethering. Continuous electromyography (EMG) of muscles helps to assess key nerve roots; it is highly sensitive though not highly specific. Triggered EMG furthermore helps to identify neural versus non-neural elements during spinal cord surgeries and to verify that placement of spine instrumentation has not breached the spinal column. These techniques provide real-time information. Findings are impacted importantly by paralytics.

Monitoring of Motor Cranial Nerves and Cranial Nerve Nuclei
Jaime Lopez, MD

Intraoperative neurophysiologic monitoring of cranial nerve motor function is used in those cranial nerves whose function has a motor component. The rationale, as well as the stimulation and recording techniques employed, is similar to that used in assessing the functional integrity of other motor peripheral nerves. The primary difference is in the placement of the recording electrodes and the neural structures that are at risk for injury. In addition, cranial nerve dysfunction is not solely confined to a cranial nerve but can also involve the nucleus of the specific nerve. Thus, a special application of cranial nerve monitoring is the functional assessment and monitoring of cranial nerve nuclei. The goal of this presentation is to review the neurophysiologic techniques, consisting primarily of EMG, used to monitor motor cranial nerves and cranial nerve nuclei.
Brainstem Auditory Evoked Potentials (BAEPs)
Alan Legatt, MD, PhD

Brainstem auditory evoked potentials (BAEPs) are the most useful auditory evoked potentials for clinical diagnosis and intraoperative monitoring. They can detect dysfunction of the ears, the auditory nerves, and the brainstem auditory pathways up through the level of the mesencephalon. BAEP recording techniques will be described. Interpretation of BAEPs requires knowledge about their anatomical generators. Wave I is generated in the distal eighth nerve. Subsequent components are composites of contributions from multiple generators, but wave III predominantly reflects activity in the caudal pons and wave V predominantly reflects activity in the mesencephalon. BAEPs cannot be used to assess the auditory pathways rostral to the mesencephalon. Interpretation of BAEPs is predominantly based on latency measurements of waves I, III, and V, on inter-peak interval measurements derived from the absolute latencies, and on right-left differences of these absolute latencies and of the inter-peak intervals. Examination of absolute component amplitudes is not useful because of marked amplitude variability across subjects, but examination of the IV-V:II amplitude ratio may detect some abnormalities that are not detected by examination of latencies alone.

Somatosensory Evoked Potentials (SEPs)
Ronald Emerson, MD

The methodology and physiology of routine laboratory somatosensory evoked potentials will be discussed, along with case examples.

NEONATAL EEG (7:00 – 8:30 AM)

Neonatal EEG Background - Overview and ACNS Terminology
Dennis Dlugos, MD

The objectives of this lecture are as follows: 1) To develop a systematic approach to neonatal EEG reading and apply it in clinical practice; 2) to know the upper limits of normal discontinuity in neonatal EEGs from 25-46 weeks; 3) to list criteria for normal voltage and abnormally low voltage neonatal EEGs; 4) to list 4 essential criteria for burst suppression on a neonatal EEG; and 5) to describe the predictive value of neonatal EEG background on outcome

EEG Background and Prognosis
Courtney Wusthoff, MD

This session will provide an update regarding the evidence basis for using EEG background assessment to guide prognostication for neonates. Topics discussed will include EEG findings in neonates with hypoxic-ischemic encephalopathy, the impact of therapeutic hypothermia on the EEG, and preterm neonates. Special emphasis will be placed on features of the EEG with highest prognostic sensitivity and specificity in the neonatal period. A suggested approach will be proposed for incorporating EEG findings into overall prognosis for individual patients.

Sleep Assessment and Implications
Mark Scher, MD

Neonatal EEG Sleep Assessment: Relevance to Diagnosis, Therapeutic Interventions and Prognosis

Serial neonatal EEG/Sleep studies document the ontogeny of cerebral and noncerebral physiologic behaviors based on visual inspection or computer analyses. EEG patterns and their relationships to other physiologic signals serve as templates for brain organization and maturation, subserving multiple interconnected neuronal networks during different arousal states. Integrated clinical/neurophysiologic databases of healthy preterm and full term subjects help elucidate the continuity of brain functions from in utero to extrauterine time periods in the context of multiple factors. Recognition of the ontogeny of behavioral and EEG patterns provides insight into the functional expression of adaptive and maladaptive neural plasticity for the sick or high risk neonate. Sleep ontogenesis documents expected postnatal patterns of brain from neonatal to infancy periods in healthy subjects to compare with altered expressions of EEG/Sleep under stressful or disease states. Automated analyses provide time- and frequency-dependent computational phenotypes of brain connectivities. Research pertaining to the developmental origins of health and disease can use these phenotypes to design longitudinal studies over the lifespan to assess cumulative gene-environment interactions. Computational strategies will ultimately improve diagnostic and prognostic acumen in the context of the assessment of neurotherapeutic interventions for fetal and neonatal patients at risk for developmental disorders.
Quick Presentation of the Phase I Evaluation in 3 Cases
Michael Sperling, MD

Intracranial video-EEG is performed when additional information is required to identify the epileptogenic zone, and at times, to map eloquent cortex. It is a supplement to the history, examination, and other tests, and data from intracranial monitoring should be taken in context. While the ictal EEG is most desired, best operative results are attained when the area of ictal onset corresponds to a structural lesion, defined either with MRI or with functional imaging. A variety of ictal onset patterns can be seen, and propagation patterns depend upon the site of origin. Examples of various ictal onset patterns and interpretation of the intracranial EEG will be shown.

Choosing Phase 2 Electrodes
Stephan Schuele, MD, MPH

Approximately 20-30% of surgical epilepsy patients require an invasive evaluation prior to resection. Chronic subdural grids offer systematic mapping and seizure recording optimal for patients with a suspected epileptogenic zone close to functional cortex. Depths electrodes seem to cause less morbidity and complications and can be placed more predictably including deeper areas inaccessible to grids. It is too early to say if three dimensional exploration with depths electrodes (Stereo EEG) can offer a better outcome for patient with nonlesional extratemporal lobe epilepsy than the disappointing results with grids have shown. Intraoperative electrocorticography can be of added benefit in lesional cases where the risk and resources necessary for chronic recordings might not be justified. In the near future, more and more epilepsy centers will need to be able to offer individualized invasive approaches to optimize outcome.

Discussion and Demonstrations of Patterns of Seizure Onsets & Spread, Underlying Pathological Substrates, Surgical Outcomes in Adults and Children
Michael Sperling, MD and Tobias Laddenkemper, MD

Intracranial video-EEG is performed when additional information is required to identify the epileptogenic zone, and at times, to map eloquent cortex. It is a supplement to the history, examination, and other tests, and data from intracranial monitoring should be taken in context. While the ictal EEG is most desired, best operative results are attained when the area of ictal onset corresponds to a structural lesion, defined either with MRI or with functional imaging. A variety of ictal onset patterns can be seen, and propagation patterns depend upon the site of origin. Examples of various ictal onset patterns and interpretation of the intracranial EEG will be shown.

The Use of Invasive Electrodes to Map “Epileptic Zones”
William Stacey, MD PhD

The use of invasive electrodes to map function and epileptic zones: Intracranial monitoring represents the state of the art for localizing epileptic networks, providing much better spatial and temporal resolution as well as better source localization. This technology has aided in the diagnosis and surgical treatment of a large number of patients, being especially effective in patients with lesional or temporal lobe epilepsy. However, nonlesional extratemporal patients consistently achieve low rates of seizure freedom after surgery, even when advanced imaging modalities such as SPECT and PET are added. The only clear biomarker predicting success in these patients is fast activity that is at the limits of standard EEG technology. This suggests that a new generation of monitoring tools may be able to improve treatment options in these patients. Emerging technologies such as electrodes with greater spatial and temporal resolution, optical imaging, and chemical sensing may hold the key to identifying and mapping epileptic zones in these patients. As these methods become available, we will also need to develop tools to analyze the unprecedented wealth of data they will acquire.

INTRAOPERATIVE MONITORING PART II (9:00 AM — 5:00 PM)

Monitoring Cerebral and Spinal Endovascular Procedures
Viet Nguyen, MD

Aneurysms, arteriovenous malformations (AVMs), stenoses, fistulas, and large vascular tumors in the brain and spinal cord can be treated via endovascular interventions, but not without risk of ischemic or hemorrhagic nervous system injury. Intraoperative neurophysiologic monitoring techniques, similar to those used in cerebrovascular and spinal surgeries, can be used to minimize morbidity and mortality. Challenges include learning new procedural events/sequences and new visualizations of anatomy, dealing with a harsh electrical environment, and, sometimes, an awake patient. Commonly monitored endovascular procedures will be reviewed, including coiling, embolization, balloon test occlusion, and angioplasty/stenting. Special periods of risk will be highlighted. Interactive case examples will be included.

Mapping of Cortical and Subcortical Brain Structures
Mirela Simon, MD, MSc

The presentation will include principles, methodologies, interpretation and troubleshooting of neurophysiologic techniques used for functional mapping and monitoring of eloquent structures in supratentorial surgery. Thus, the talk will extend beyond mapping the eloquent cortex. I considered this necessary due to the fact that in most instances, avoidance of postoperative neurologic deficit associated with supratentorial surgery also requires continuous feedback and guidance throughout the resection process. Finally, I will emphasize the importance of minimizing stimulation triggered seizures, as far as improving the patient’s safety and the accuracy of the mapping. The following techniques will be presented in detail: 1- Median somatosensory evoked potentials (SSEPs) phase reversal technique for central sulcus localization; 2- Cortical motor mapping via direct electrical stimulation; 3- Subcortical motor mapping via direct electrical stimulation; 4- Continuous motor monitoring for protection of the mapped eloquent cortical and subcortical structures; 5- Language mapping; 6- Monitoring for and management of stimulation triggered afterdischarges (ADs) and seizures; 7- Anesthesia Considerations. Important points of each technique will be exemplified by neurophysiologic recordings and pictures.

Regulatory, Medical-Legal, and Coding/Billing Issues
Marc Nuwer, MD, PhD

(ADs) and seizures; 7- Anesthesia Considerations. Important points of each technique will be exemplified by neurophysiologic recordings and pictures.

Monitoring of Peripheral Nerve Surgery
Leo Happe, PhD
Peripheral nerve injuries are common though they pose difficult decisions for clinicians. These questions include issues relating to the current status of the injury, whether the injury is recovering spontaneously and whether the injury will require surgical repair. In the early period after injury, little information is available to determine the extent of the injury and this compels intuitive decision-making. In the absence of hard information, such decisions may not lead to the best clinical outcomes. Proper use of operative stimulation and recording directly from the nerve injury site provides the necessary information to facilitate decision-making and lead to the optimal outcome for a particular injury.

Evidenced Based Studies in IOM
Jonathan Edwards, MD
Neuro-Intraoperative Monitoring (NIOM) continues to be a rapidly growing field in Neurophysiology. A significant body of peer reviewed clinical research studies have been published over the last decade, covering a wide range of NIOM procedures.

In this session, we will review the topic of Evidence-Based medicine, and we will discuss various kinds of evidence. We will review what we currently know regarding outcome data in NIOM, and will also explore some current challenges and opportunities for outcome studies and evidence in NIOM.

Troubleshooting During IOM
Brett Netherton, MS, FASNIM, CNIM
Many of the physiologic signals of interest in IOM are in the low microvolt range by the time they volume conduct to the surface of the body where we record them. A close look at the recording pathways of interest, from the source generator to the computer screen we view, reveals a fascinating world of engineering and technical accomplishment. In addition, it reveals areas of opportunity for improving what we do when performing IONM.

The goal of this presentation is to briefly review our most fundamental recording circuitry with focus on understanding the points where our troubleshooting can have the most impact. Particular attention will be devoted to optimizing the layout and application of recording and stimulating electrodes, learning the ideal way to run attachment leadwires, and understanding the role and limitations of the ground electrode. The dangers of capacitive coupling with the Electro-Surgical cabling leading to electrode burns will also be presented.

Many videos of demonstrations will be presented to illuminate not only the fascinating world of capacitance and other electromagnetic principles, but more importantly, to help the practitioner learn principles with which they can solve troubleshooting dilemmas and minimize risk for subdermal needle electrode burns.

UPDATES IN THE BUSINESS IN CLINICAL NEUROPHYSIOLOGY: FROM WASHINGTON TO YOUR OFFICE (7:00 — 8:30 AM)

Working with your local hospital in becoming a designated epilepsy center
Deborah Briggs, MD
The objectives of this presentation are to:
- Discuss the various types of accreditation and certification for epilepsy centers.
- Outline the cost and benefits of each type of accreditation.
- Will specifically address certification/accreditation from National Associate of Epilepsy Centers, American Board of Registration of EEG/EP Technologists, Joint Commission Accreditation of Healthcare Organizations — epilepsy disease specific certification.

Medicare Hospital Outpatient PPS Proposed Rule for 2014 Update
Marc Nuwer, MD, PhD
Update in the Business of Neurophysiology: Medicare Regulatory Update In 2013 several Advocacy initiatives were taken. Medicare (CMS) proposed a 50-60% reduction for office technical component payment for EEG and evoked potentials. After formal comments from the society, the final rule decreased that reduction to less than 20%, i.e. back to 2012 levels. Medicare in 2013 implemented deep reductions in EMG and NCV payments. After presentations to a special review panel, the office EMG pro fee payment was increased more than 20%. NCV payments remain unchanged from 2013. In hospitals, the proposed bundling of clinical neurophysiology tests was avoided. CMS originally proposed to pay nothing more for testing if the patient was seen elsewhere at the hospital that day. Further changes and new regulations also will be discussed.

Obama Care and its Impact on Neurophysiology
L. Elizabeth Mullikin, MPA, FACHE, FASET
Obama Care: Impact on Clinical Neurophysiology Author: L. Elizabeth Mullikin, FACHE The Affordable Care Act is accelerating the state of health care reform. What are the implications for clinical neurophysiology? With mounting focus on disease prevention, care coordination, and cost reductions substantial growth will occur in the outpatient ambulatory clinic practice and business that has been historically gained in the hospital setting will slow in pace. Reimbursement will continue to be pressured by policies designed to reduce costs with emphasis on efficiencies and a premium on value. Be prepared for full capitation models that are on the horizon. Clinical Neurophysiology is found in areas that are under tremendous scrutiny. It is not surprising that providers will be increasingly rewarded for not duplicating tests and performing unnecessary procedures. Consumers are rapidly becoming cost-conscious and looking for alternative treatment options. Every practitioner should understand the implications of patient experience.

EMG and EEG Technology (7:00 — 8:30AM)
Charles Epstein, MD and Susan Herman, MD
Digital technology has expanded the range of tools available to clinical neurophysiologists, and with it the multiple steps in signal processing that must be understood to use them optimally. Incorporating live demonstrations, this session incorporates aspects of the electrode interface, the different meanings of “ground”, electrical safety, differential amplifiers, common-mode noise rejection, analog-to-digital conversion, sampling and aliasing, filters, and fundamentals of frequency analysis.
Some key points that will be reviewed include: (1) connecting patients to true earth ground is dangerous because Earth is part of the AC power system. (2) The problems produced by imperfect electrode connections cannot be resolved simply by filtering the 60-Hz noise. (3) Artifacts can be generated both by using and omitting anti-aliasing filters. (4) EEG frequency analysis opens up a vast universe of potential applications that are becoming increasingly important in clinical practice.

---

**Assessment of Common Entrapment Neuropathies with EMG and Ultrasound**

Francis Walker, MD

Entrapment neuropathies are common problems in the general population and make-up a significant proportion of patients referred for electrodiagnostic testing. Recent advances in neuromuscular ultrasound have made it possible to routinely provide anatomic information regarding entrapped nerves that is complementary to information gathered from electrophysiologic testing. The most common entrapment neuropathies, median neuropathy at the wrist, ulnar neuropathy at the elbow, and peroneal (fibular) neuropathy at the fibular head involve superficial nerves that are easily investigated with high resolution ultrasound transducers. Typical findings in entrapment neuropathies include slowing of nerve conduction velocities or conduction block across affected segments and in more severe cases, reduction in motor or sensory response amplitudes along with spontaneous resting activity on EMG of distal muscles innervated by the entrapped nerves. By ultrasound nerves are enlarged and show reduced echogenicity in a fusiform distribution along several centimeters of nerve around the site of entrapment. There may also be changes in nerve vascularity and nerve mobility associated with entrapment, or the presence of anatomic variants, abnormalities in adjacent structures (e.g. tenosynovitis), or, less commonly, unexpected intraneural pathology. Ultrasound adds to the diagnosis and understanding of common nerve entrapments.

**Assessment of Radiculopathies and Plexopathies - EDX Approaches and Limitations**

Daniel Menkes, MD

Neurologists as well as other practitioners encounter patients with monomelic signs and symptoms. Most of these can be subdivided into mononeuropathies, radiculopathies orplexopathies in the order of their incidence. This requires a working knowledge of the brachial plexus and the lumbosacral plexus depending on the affected extremity. The first course of action is to determine if the lesion is restricted to one nerve territory. If so, then the diagnosis is mononeuropathy. If the patient has evidence of an objective deficit outside one nerve territory, then the most likely diagnosis is a radiculopathy. In the upper extremity, most radiculopathies affect one nerve root. In contrast, polyradiculopathies may be encountered in the lower extremity especially with centrally herniated discs. Notably, plexopathies rarely occur in the absence of a history of severe trauma or a neoplastic process. Notwithstanding, electrophysiologic studies are valuable for two main reasons. In some instances, the correct diagnosis cannot be ascertained on clinical examination alone such that electrophysiologic studies can render a more definitive diagnosis. In addition, electrophysiologic studies can characterize the chronicity and severity of the pathological process. These studies are of paramount importance when considering the best course of treatment.

---

**ICU EEG (9:00 AM — 5:00 PM)**

**Overview of ICU EEG Monitoring in Neonates, Children and Adults**

Nicholas Abend, MD

Critically ill patients of all ages are increasingly undergoing EEG monitoring. This talk will discuss the current use and impact of EEG monitoring, the epidemiology of electrographic seizures, the impact of electrographic seizures on physiology and outcome, the utility of quantitative EEG for seizure identification, and available guidelines/pathways addressing EEG monitoring in critically ill patients.

**The Ictal-interictal Continuum: Case Studies**

Suzette LaRocche, MD

Periodic and rhythmic EEG patterns are a common finding in critically ill patients, particularly those with acute brain injury. Although many of these patterns do not meet strict criteria for electrographic seizures, they exhibit features that are suggestive of ictal activity and hence have been termed “The Ictal-Interictal Continuum”. However, data on the association of these patterns with seizures, neuronal injury and outcome are lacking. Furthermore, there is no consensus on the approach to treatment of these patterns. This session will utilize case discussions to demonstrate the range of patterns encountered along the ictal-interictal continuum and discuss approaches to the management of patients exhibiting these findings including utilization of neuroimaging, multimodality monitoring and treatment trials.
to ICU patients and their potential diagnostic and prognostic implications. We will also address the controversy that surrounds the significance and management of periodic patterns.

Quantitative EEG for Seizure Identification
Cecil Hahn, MD, MPH

This presentation will provide an introduction to available techniques for quantitative EEG (QEEG) trending for seizure identification. I will review the concepts underlying various methods of quantitative EEG transformation, and discuss the potential applications of a variety of QEEG trends for seizure identification. I will review available data on the sensitivity and false positive rates of QEEG trends for seizure identification by expert neurophysiologists and ICU bedside caregivers. Finally, I will discuss how QEEG trends may be incorporated into an ICU EEG monitoring program to complement both live and post-hoc EEG review.

Treatment of Nonconvulsive Seizures and Status Epilepticus
Aatif Husain, MD

Generalized convulsive status epilepticus (GCSE) is readily recognized and is treated aggressively as it is a neurologic emergency. Nonconvulsive SE (NCSE) and nonconvulsive seizures (NCS) are more difficult to diagnose, and their contribution to neurologic morbidity is less clear. Usually the same antiepileptic drugs (AEDs) and the same treatment algorithm is used to treat GCSE and NCSE/NCS. However, it is unclear whether NCSE/NCS should be treated as aggressively as GCSE. Clinicians are more reluctant to use anesthetic agents for the control of seizures in NCSE/NCS than GCSE. Other AED options are available and may be appropriate in NCSE/NCS. These issues will be explored in this presentation. Thereafter a case will be presented which highlights the need for “appropriately aggressive” AED therapy.

The Business of ICU EEG Monitoring
Stephen Hantus, MD

Developing a Program and a Business Plan Each hospital across the country has patients with altered mental status, acute neurologic lesions and unexplained comas. Finding a way to build an ICU EEG program to support these patients requires planning and cooperation among departments. In addition to building clinical expertise and efficiency, a program must make business sense if it is to survive in the modern era of health care. In this session we discuss the process of building an ICU EEG program and developing a business model.

VIDEO-EEG (9:00 AM – 12:00 PM)

The Essentials of Video-EEG
Michael Sperling, MD

Video-EEG is performed to answer a focused question about the nature of paroxysmal events, classify seizures, and localize seizures. Video is useful for behavioral characterization of the episodes, and necessary for careful behavioral analysis. Not all seizures appear in the scalp or even intracranial EEG, and many artifacts can mimic seizures or obscure EEG interpretation. Visual inspection remains the standard for interpretation. Commercial manufacturers provide equipment that meets clinical and safety needs. Examples will be shown of technical setup, artifacts, and detail provided about technical specifications.

Applying Video-EEG in Practice (Focus on Pediatric Patients)
Tobias Laddenkerper, MD

Applying Video-EEG in practice (focus on pediatric patients) This presentation reviews lateralizing and localizing pediatric semiological signs during epileptic seizures with respect to prediction of the side of the epileptogenic zone and, therefore, presurgical diagnostic value. The lateralizing and localizing significance of semiological signs and symptoms can frequently be concluded from knowledge of the cortical representation. Visual, auditory, painful, and autonomic auras, as well as ictal motor manifestations, e.g., version, clonic and tonic activity, unilateral epileptic spasms, dystonic posturing and unilateral automatisms, automatisms with preserved responsiveness, ictal spitting and vomiting, emotional facial asymmetry, unilateral eye blinking, ictal nystagmus, and akinesia, have been shown to have lateralizing value. Furthermore, ictal language manifestations and postictal features, such as Todd’s palsy, postictal aphasia, postictal nose-wiping, postictal memory dysfunction, as well as per-ictal water drinking, per-ictal headache, and ipsilateral tongue biting, are reviewed. Knowledge and recognition of pediatric semiological localizing and lateralizing signs during seizures is an important component of the presurgical evaluation of epilepsy surgery candidates and adds further information to video/EEG monitoring, neuroimaging, functional mapping, and neuropsychological evaluation.

Video-EEG Pearls: A Case-Based Approach (Focus on Adults)
William Tatam, DO

This half-day course will address the critical need to enhance the skill sets of the neurologist and clinical neurophysiologist who perform video-EEG monitoring. The venue will include a didactic approach including the essentials of video-EEG prior to presenting case-based examples that address the pearls and perils that are involved for those performing evaluations in children and adults. The need for a definitive diagnosis, proper classification and characterization of patients with seizures and spells are apparent from the rate of misdiagnoses that occur in clinical practice. This course will provide practical information on the necessary aspects of video-EEG that will address the gaps in our current knowledge. The relevance to video-EEG will be enhance by including technical implications, the uses in pediatric and adulthood, and add critical safety issues involved with in the hospital as well as outside the epilepsy monitoring unit.

Relevance of Patient Monitoring: Sudden Unexpected Death in Epilepsy
Stephan Schuele, MD, MPH

Patients undergoing an elective admission to an Epilepsy Monitoring Unit (EMU) for the purpose of event recording while off medications should be continuously monitored by nursing staff and/or technologists. Staffing can be challenging and clear protocols and in-service training essential to maintain uninterrupted supervision. EKG telemetry looking for ictal tachycardia and EEG seizure detection software are both relatively sensitive and specific and might help to alert caregivers in a timely way.

Continuous monitoring is not only paramount for the quality of seizure testing, fall precautions and treatment interventions in the EMU, attendance and ability to provide resuscitation is also the most important factor to prevent Sudden Unexpected Death in Epilepsy (SUDEP). In the recently published MORTENUS study, timely resuscitation within 3 minutes separated the 9 patients experiencing near SUDEP from the 16 patients dying from SUDEP after a generalized convolution in the EMU. In the SUDEP patients, cardiorespiratory dysfunction was seen within the first 3 minutes after a
seizure and continuous heart rate and oxygen monitoring should be displayed at an EMU bedside to assist postictal supervision and resuscitation.

**Video-EEG Monitoring in the Home and Beyond — New Approaches**
*Steven Schachter, MD*

Ambulatory and home monitoring with simultaneous EEG and video may provide valuable diagnostic information in the evaluation of episodic behaviors in many patients. Associated challenges, technical limitations, and feasibility and safety issues have diminished with technological advancements and greater availability of ambulatory video-EEG systems as well as consumer video products. As technologies for ambulatory and home-based seizure monitoring further expand, their utilization will likely extend beyond short-term diagnostic evaluation to facilitate the development of systems that provide long-term monitoring to enhance patient self-management, reduce safety risks, diminish the possibility of SUDEP, track response to changes in therapy during drug trials, and increase our understanding of epileptogenesis.

**CASE STUDIES IN PERIPHERAL NEUROPHYSIOLOGY (2:30 – 5:00 PM)**

**Case Studies in Peripheral Neurophysiology**
*Elliot Dimberg, MD; Chris Nance, MD; Randa Jarrar, MD*

Utilizing a case-based approach, the faculty will present examples of various peripheral neuromuscular diseases. The focus will be on the neurophysiology of each disorder, but discussion will also include clinical and pathological aspects of each disorder, where applicable. Representative disorders will consist of primary disease of muscle including disorders of muscle membrane ion channels, the neuromuscular junction, peripheral nerve, motor neurons, and pediatric disorders. At the conclusion of the session attendees should be able to interpret patterns of clinical neurophysiological findings in peripheral nervous system disease and appropriately localize neuromuscular abnormalities according to the neurophysiological characteristics.
The functional significance of the brain’s thalamocortical activity will be discussed. Such activity will be addressed as the product of intrinsic electrical properties of neurons and the networks they weave. How this recurrent activity engenders motricity, as well as in the genesis of several neuropsychiatric ailments, will be discussed.

2013 Pierre Gloor Award & Lecture: Thalamocortical Dysrhythmia (TCD): Function and Dysfunction
Rodolfo Llinas, MD, PhD

The functional significance of the brain’s thalamocortical activity will be discussed. Such activity will be addressed as the product of intrinsic electrical properties of neurons and the networks they weave. How this recurrent activity engenders motricity and cognition, as well as in the genesis of several neuropsychiatric ailments, will be discussed.
Myotonia in Childhood
Peter Kang, MD

Clinical or electrophysiologic myotonia is a distinctive phenomenon at any age, including childhood and adolescence. Clinical myotonia can often be detected in school age children and adolescents. It may be more difficult to observe in infants, but some newborns with channelopathies such as paramyotonia congenita may display eyelid myotonia when they sneeze. In children and adolescents, electrophysiologic myotonia has been associated with channelopathies, myotonic dystrophy, congenital myopathies, and glycogen storage diseases. In addition, the presence or absence of myopathic motor units on needle examination can help distinguish further among these disease categories. Channelopathies such as myotonia congenita, paramyotonia congenita, and hyperkalemic periodic paralysis tend not to be associated with myopathic motor units. Myopathic motor units may or may not be seen in cases of myotonic dystrophy, congenital myopathies, and glycogen storage disorders. The needle examination is sometimes less comprehensive in children compared to adults, thus when electrophysiologic myotonia is not found, such results should be interpreted in light of the number of muscles examined and the degree of clinical suspicion. Thus, it is worth examining pediatric patients for clinical and electrophysiologic myotonia in the proper clinical contexts, as this finding can be very helpful in the diagnostic evaluation when detected.

Pediatric Neuromuscular Junction Disorders and the use of Stimulated SFEMG
Matthew Pitt, MD, FRCP

This presentation begins with a discussion of the different techniques available for analysis of the neuromuscular junction. It passes from nerve conduction studies and routine EMG to repetitive nerve stimulation before focusing on single fibre electromyography (SFEMG) and in particular stimulation SFEMG. The technique is demonstrated. There are problems with the analysis of the results, in particular the selection of potentials, which can be used. With the difficulties of identifying suitable potentials, a proposal is put forward that distinction between normal and abnormal responses is perhaps all that can accurately be achieved. Specificity is another problem that is considered. In contrast to the situation in adult patients where the differential diagnosis for myasthenia is relatively restricted the situation in children is much less easy with a wide range of clinical presentations being observed in myasthenic syndromes. To this end an investigative strategy using stimulation single fibre EMG is described, which allows differentiation between the common conditions entering the differential diagnosis such as bulbar palsy as well as myopathy with disordered neuromuscular junction function. Finally it is recommended that the technique should be called stimulation jitter analysis with concentric needle electrodes or StimJACE.

WIDE BANDWIDTH ELECTROPHYSIOLOGY AND EPILEPSY BIOMARKERS (10:30 AM – 12:30 PM)

International Electrophysiology Database and Collaborative Research
Brian Litt, MD

The International Electrophysiology Database (http://ieeg.org) is a free, cloud computing-based resource sponsored by the NIH/ NINDS. “The Portal” can be run from any web browser, it contains easy to use tools for viewing, annotating, analyzing and selectively sharing data, including multi-scale time series, imaging and metadata. The portal is set up to share actual algorithms and analysis code. Data can be selected and streamed to any computer for analysis from the portal, or distributed on Amazon’s Elastic Computing Cloud (EC2), for big analysis jobs. Each experiment is given a unique identifier linking data with processing algorithms that allows users to repeat and validate any experiment once it has been “published.” The portal makes multi-center studies, data collection and analysis extremely easy, as well as sharing educational materials such as teaching sets of EEG, evoked potentials, ICU monitoring, device recordings and intracranial data. The portal allows users to establish consensus/gold standard training and testing sets for evaluating software to interpret human and animal electrophysiology. In this lecture I will familiarize the audience the International Electrophysiology Portal, its capabilities and discuss the role of “Big Neural Data” in Translational and Basic neurophysiology research.

Origin of Pathological and Physiological Oscillations
William Stacey, MD PhD

For over a decade, researchers have evaluated the role of High Frequency Oscillations (HFOs) in the brain. Originally described in normal activity, HFOs were later found to have a strong correlation to epileptic tissue, and subsequent efforts have concentrated on the characteristics of this relationship. The origin of these oscillations is a complex problem, as there is evidence for a number of independent network processes. More importantly, there is great interest in identifying a difference between ‘normal’ versus ‘abnormal’ HFOs. Early work suggested stratifying by peak frequency, as HFOs > 250 Hz appeared to be more specific to epilepsy. However, more recent work has shown that the distinction is much more complicated. This talk will discuss the current theories on the generation of physiological and pathological HFOs, as well as the challenges we face and potential benefits of using HFOs as a biomarker of epilepsy.

BOTULINUM TOXIN: MECHANISM OF ACTION AND ULTRASOUND VS. EMG GUIDANCE (1:30 – 3:30 PM)

The Clinical Neuropharmacology and Neurophysiology of Botulinum Toxin
Francis Walker, MD

The basic pharmacology of botulinum neurotoxins is well delineated in that the toxins prevent the release of synaptic acetylcholine from terminal nerve endings at the neuromuscular junction. The drug interferes with the action of docking proteins on the neuronal membrane that allow the synaptic vesicle to fuse with the membrane and release their content. The clinical neurophysiology and pharmacology of the drug relates to its time course of action following intramuscular injections, the degree to which it spreads or is absorbed into the bloodstream, and its duration of action. Currently available toxins, although dosed differently in terms of units, seems to have similar biologic effects at equipotent doses. Studies with injections into small muscles show that the vast majority of the drug is active at the injected site, that diffusion is generally robust within a muscle, and that the reduction in compound muscle action potential amplitude that follows injections seems to begin before and last long after clinical effects are appreciated in patients with dystonia and spasticity. The cause of these differences remain unclear. Optimal use of these agents is likely enhanced by clear understanding of their biological properties.
Ultrasound Guidance of Botulinum Toxin Therapy
Katharine Alter, MD

Botulinum Toxins (BoNTs) are exotoxins of Clostridium Botulinum, an obligate anaerobic bacterium. BoNTs are known to be highly toxic but in minute quantities can provide significant relief to patients with a wide variety of clinical problems. The pharmacology of BoNTs and the principles of chemodenervation will be reviewed. When performing chemodenervation procedures, accurately targeting the structures to be injected is important for efficacy as well as to reduce potential risks of the procedures. Conventional guidance techniques including palpation/anatomic landmarks, EMG and electrical stimulation have recognized limitations. B-mode ultrasound is increasingly recognized as a more accurate imaging technique. This presentation will review ultrasound technology and scanning techniques required for performing ultrasound-guided chemodenervation procedures. A review of ultrasound imaging of muscles commonly targeted for botulinum toxin injections or nerve blocks will also be presented.

ISCHEMIA MONITORING IN CRITICAL CARE: EEG TREND ANALYSIS TO DETECT DEVELOPMENT OF AND RECOVERY FROM CEREBRAL ISCHEMIA (1:30 – 3:30 PM)

Continuous EEG monitoring (cEEG) in the intensive care unit has emerged as a tool for detecting delayed ischemic neurologic decline (DIND) following subarachnoid hemorrhage. This circuit functioning, and therefore the EEG, are tightly coupled to cerebral blood flow (CBF) and energy metabolism. Decreases in CBF produce predictable changes in EEG background rhythms, from the loss of fast oscillations to the emergence of high amplitude irregular or rhythmic slow oscillations, and ultimately to voltage attenuation and background suppression. Quantitative EEG (qEEG) measures have been shown to objectively capture these nearly instantaneous changes to the EEG in response to changes in CBF; several studies have demonstrated the use of qEEG parameters in detecting the slower ischemic changes associated with delayed cerebral ischemia after subarachnoid hemorrhage. We will review the pathophysiological processes that create changes to cEEG/qEEG during ischemia and discuss important groundwork that has paved the way for current efforts in using qEEG to detect ischemic injury before it becomes irreversible.

Evolution of EEG Patterns after Global Cerebral Ischemia
Michael Van Putten, MD

In patients with postanoxic encephalopathy a rich phenomenology of EEG patterns is observed, ranging from iso-electric recordings, burst-suppression patterns or generalized periodic discharges to diffuse slowing, reflecting the extent of initial hypoxic injury and neural repair. These processes are well captured by continuous EEG monitoring, that typically evolve over time and have a strong predictive value for the neurological outcome. I will shortly review the differential sensitivity of neuronal processes and network activity to oxygen/glucose deprivation, and present examples of the evolution of EEG patterns, and their correlation with predicting poor and good outcome, based on our prospective cohort of postanoxic patients (n>150) treated in the ICU after cardiac arrest.

Emerging Methods for cEEG Ischemia Detection in Subarachnoid Hemorrhage
Eric Rosenthal, MD

Continuous EEG monitoring (cEEG) in the intensive care unit has emerged as a tool for detecting delayed ischemic neurologic decline (DIND) following subarachnoid hemorrhage. Its use as a continuous, noninvasive, and multiregional biomarker of end-tissue health is complementary to other tools focusing on uni-regional, temporally discrete, or inferred measures of blood flow. However, clinical practice in real time is labor-intensive, and the benefit of cEEG-guided management on clinical outcomes remains unexplored. Prospective clinical experience with SAH ischemia monitoring will be detailed; including 1) the impact of seizure and DIND rates on reading and reporting standards, 2) variations in practice among institutions, and 3) reasonable expectations of accuracy in practice compared with other methodologies, including timeliness, authority, and novelty. A proposed clinical guideline for the use of cEEG monitoring and reporting in clinical practice will be discussed as well as requirements for clinical trials assessing its benefit.

The EE of Cerebral Ischemia
Brandon Foreman, MD and Jan Claassen, MD, PhD

cEEG for seizure detection is well established; the use of cEEG to detect early ischemic changes has a solid basis in physiology, but has been underutilized in clinical practice. The EEG signal normally records voltage oscillations that reflect neocortical circuits in action. This circuit functioning, and therefore the EEG, are tightly coupled to cerebral blood flow (CBF) and energy metabolism. Decreases in CBF produce predictable changes in EEG background rhythms, from the loss of fast oscillations to the emergence of high amplitude irregular or rhythmic slow oscillations, and ultimately to voltage attenuation and background suppression. Quantitative EEG (qEEG) measures have been shown to objectively capture these nearly instantaneous changes to the EEG in response to changes in CBF; several studies have demonstrated the use of qEEG parameters in detecting the slower ischemic changes associated with delayed cerebral ischemia after subarachnoid hemorrhage. We will review the pathophysiological processes that create changes to cEEG/qEEG during ischemia and discuss important groundwork that has paved the way for current efforts in using qEEG to detect ischemic injury before it becomes irreversible.

THE CREATION OF EVIDENCE BASED MEDICINE IN INTRAOPERATIVE MONITORING (1:30 – 3:30 PM)

Grading of Evidence and the Creation of Trials
David Gloss, MD, MPH

In 2011, the Institute of Medicine published new standards for Clinical Practice Guidelines. The Guideline Development Subcommittee of the American Academy of Neurology (AAN) accepted these standards, and developed a new process for creating AAN guidelines. Previously, it was a two step process. 1. Creation of a systematic review based solely on AAN grading risk of bias in papers. 2. Development of recommendations based on the strength of evidence. This process has put intraoperative monitoring in a difficult position, since by AAN grading, most papers in intraoperative monitoring are considered low quality. The new process has four basic steps. 1. Slightly modified systematic review, 2. outcomes are abstracted from the papers, and a AAN-specific modified GRADE process is performed on them, 3. A modified Delphi process is performed on the resulting outcomes, looking at other factors such as variation in preferences, financial burden, magnitude of harm and benefit. With this new process in place, it may be time to propose a new guideline, using this new process. A new guideline may allow for more comprehensive community standards.

The Level of Evidence in Intraoperative Monitoring
Jay Varma, MD

In the first portion of the session, we review the studies used to create the evidence-based guideline for intraoperative spinal monitoring with somatosensory and transcranial electrical motor evoked potentials which was updated last year by the
Creation of Intraoperative Monitoring Guidelines
Marc Nutner, MD, PhD

Creating formal assessments involves several steps. A Panel of Experts devise one or several specific research questions. A literature search is based on relevant terms. Abstracts are screened for prospectively set inclusion-exclusion criteria. Literature is scored by pre-set criteria to determine evidence table data from all included studies. Studies’ class of evidence are graded. The assessment’s level of evidence is based on the studies’ classes of evidence. The assessment’s text addresses the methods, data found, conclusions drawn, and clinical relevance. The draft is reviewed by users, vendors, other specialties, and the sponsoring society’s committees and board of directors. After an extended period for all comments, questions, and conflicts to be resolved, the Panel, committee(s) and society’s board can endorse the new assessment. Setting the initial research questions well is a key to a good assessment. Problems include how to weigh animal studies, ethical issues about possible studies, heterogeneity of techniques and clinical applications, and lack of well designed class one studies. Recommendations are based on evidence, which can be constraining. Other comments can be considered in a discussion or commentary section, which may go beyond the evidence presented. Evidence-based Guidelines for clinicians are based on the outcomes of these assessments.

ADVANCED PRACTICE TECHNOLOGISTS IN THE NEW WORLD OF CONTINUOUS NEUROPHYSIOLOGICAL MONITORING (4:00 – 5:30 PM)

Eduction, Knowledge, and Credentials for Advanced Practice Technologists
Joshua Ehrenberg, BS, R EEG T, CNIM

Over the course of many years, the field of neurophysiology has developed significantly. With the growth in LTVM, ICU cEEG, and intraoperative monitoring, there has been a need for advanced practice technologists, and in many cases, these roles have been defined at the facility level. The ASET scope of practice has attempted to encompass this change. In instances where this has been implemented, and a differentiation has been made between procedural and advanced practice technologists, the qualifications have typically been subjective. There is a clear difference in some places between procedural technologists, who are at varying ranges of technical expertise, and advanced practice with clinical knowledge in non-traditional EEG areas. Due to the increasing need for this advanced practice role, and a cleaner definition of procedural technologists versus advanced practice technologists scopes of practice, formal and standardized programs of education were needed. Emory University, in conjunction with staff at other facilities where advanced practice roles are used, developed formal educational routes to advanced practice and the qualifications necessary to enter.

Current needs and usual practices will be discussed including proposed advanced practice requirements, the need for formal facility scope of practice differentiation, and the standardization of “advanced practice fellowship” programs.

Advanced Practice in the OR
Brett Netherton, MS, FASNIM, CNIM

As the neurodiagnostic specialty of IONM evolves, a correlate number of highly skilled IONM Technologists evolve with the field. The role of the Advanced IONM Technician (A-IONM tech) is explored by presenting the results of questionnaire responses by multiple A-IONM Techs. The A-IONM Tech routinely utilizes many different skillsets, tools, and relationships with others on the IONM team to accomplish their scope of practice. The goal of this presentation is to highlight the levels of achievement of modern A-IONM Techs. What are the challenges in their scope of practice? How are their skillsets evolving to further benefit the IONM team? What new electrodiagnostic and non-electrodiagnostic technologies are becoming part of their scope of practice? What are the challenges in their daily practice from remote versus onsite professional oversight? Attention will be given to differences in experience noted between A-IONM Techs working daily in the same hospital setting compared to those A-IONM Techs practicing between multiple facilities and settings. Additionally, what can we learn from the A-IONM Tech regarding how to recruit and train more like them? And what improvements in the overall IONM team function can the A-IONM Tech identify to further the ultimate goal of improved patient outcomes?

Utilization of Advanced Practice in EEG Monitoring
Sherry Nehamkin, R. EEG/EP T., CNIM, CLTM

The EEG Technologist Reader is an evolving advanced practice role that will help fill a tremendous need in the rapidly growing field of electroneurodiagnostics, especially when considering the rapidly increasing demand for ICU video EEG monitoring.

We propose that when carefully designed protocols are in place, experienced, credentialed technologists with advanced practice training should have the opportunity to be actively involved in the ongoing evaluation of electroclinical studies.

Taking on this new role in the Comprehensive Epilepsy Center, the Technologist Reader is responsible for evaluating long term video EEG files, including monitoring real time activity, and preparing reports for the epileptologists, attending daily patient rounds with the epilepsy team, and teaching health care workers who take care of epilepsy patients and other patients requiring long term video EEG monitoring.

SPIKES AND COGNITION: TO TREAT OR NOT TO TREAT? (4:00 – 5:30 PM)

Electrophysiological Assessment of Frequent Spiking and Non-Convulsive SE Syndromes
Iván Sánchez Fernández, MD

The relationship between interictal epileptiform activity and cognition could be better studied if quantified. While we can measure the cognitive function with standard cognitive tests, and express it numerically, the same does not apply for interictal epileptiform activity. We have no standard method of quantification of the interictal epileptiform activity, which makes comparisons between different patients and different studies challenging. Several reasons make quantifying epileptiform activity difficult: spikes have different morphologies and voltages, they are more or less localized or generalized, they occur mainly during wakefulness or mainly during sleep; should they be quantified equally? A relatively straightforward method of quantification is counting the number of spikes in a given EEG tracing. This can be
done as the percentage of seconds occupied by, at least, one spike or as the total number of spikes per unit of time. The latter method has no ceiling effect (can go over 100%) and therefore is able to distinguish between different degrees of severity in EEG tracings with many spikes. Future developments of spike quantification include the automatization of spike counting, and quantification of qualitative features such as voltage or distribution.

**Relationship Between Spiking and Cognition**

Kevin Chapman, MD

Concerns that interictal spikes may negatively affect cognition have been described since the late 1930s, but they have often been considered primarily of academic interest and without relevance to everyday function. This presentation will highlight findings from relevant studies describing the relationship of interictal discharges to decreased cognitive functioning that extends beyond simple reaction time prolongation. Differences between generalized and focal discharges have been observed in multiple reports, with focal discharges appearing to have more local effects in disrupting cognitive performance. The relationship between discharges and cognition may also be bidirectional in which decreased discharge frequency is present during task performance, with increased discharge frequency during periods of rest. Evidence also suggests that the disruptive effects of interictal discharges extend to the hippocampus, with a negative effect on Stenberg task short term memory performance recently has been described. Interictal discharge effects likely contribute to “noise” in the clinical characterization of epilepsy surgery patients and their outcomes. In addition to differences in cognitive effects associated with generalized vs. focal discharges, initial evidence suggests potential differential benefit following successful treatment of interictal spikes, although conclusive evidence is currently lacking.

**Should we Treat Spikes?**

Kevin Chapman, MD

Interictal spikes have been associated with cognitive impairment. This presentation will discuss which patients with interictal spikes should be considered for treatment. We will also review the available data studying the effect of anticonvulsant medications on interictal spikes.

---

**SPASTICITY - WHAT IS IT AND WHAT IS THE ELECTROPHYSIOLOGY (4:00 – 5:30 PM)**

**Storkes**

Cliff Klein, PhD

Weakness, muscle hypertonia, and spasticity contribute to poor hand function after stroke. These impairments may be attributed in part to altered excitability and behavior of spinal motoneurons. Spasticity may reflect increased excitatory synaptic input to the motoneurons and/or an increased intrinsic motoneuron excitability. Regardless of the origin, spastic paretic motoneurons may reside in a more depolarized (excitatory) state (Katz and Rymer, 1989). Axonal ion channels, membrane pumps, and axonal geometric properties play pivotal roles in axon function, and these components are derived in part from synthetic activities of the motoneuron. Hence, it is reasonable to assume that peripheral axonal properties may change when their central source of impulse activity and metabolic support is disrupted. This talk will provide an introduction to nerve excitability testing using threshold tracking (Bostock et al. 1998) and present some data using this technique in people with chronic stroke. Nerve excitability testing can indirectly assess excitability properties (ie., ion channels, membrane potential) of peripheral nerve fibers in-vivo, and by extension the motoneurons. Hence, the method may provide important insights into the processes underlying neuronal plasticity associated with disease and injury.

**Investigative Approaches to Characterizing Spasticity**

W. Zev Rymer, MD, PhD

Recently, there has been an increase in our knowledge concerning ways in which spinal motoneurons become more excitable after lesions of the neuraxis. Central to this knowledge is the discovery that spinal motoneurons display “voltage-gated conductances” — active sodium and calcium conductances that are switched on by ongoing membrane depolarization. Most importantly, these conductances are sensitive to the presence of neuromodulators such as serotonin or norepinephrine; these are released by brainstem pathways. Disturbances of excitability, such as spasticity may be linked, in part, to disruptions of these voltage-gated conductances. In complete spinal cord injury, spinal motoneurons lose their dependence on ambient serotonin, and respond as if serotonin receptors are highly active, giving rise to exaggerated excitability - this is responsible for hyperreactive stretch reflexes as well as spasms. In hemispheric brain injury, such as follows stroke, serotonin release may be increased, augmenting voltage gated conductances in spinal motoneurons, giving rise to exaggerated responses to muscle stretch. These various mechanisms can change reflex threshold by depolarizing motoneurons—as detected by clinical tests such as the Tardieu. Changes in reflex gain may also be manifested in an exaggerated response to tendon tap. We will describe ways to separate these two contributing mechanisms.
SEIZURES, SUDEP AND AUTONOMIC NERVOUS SYSTEM (8:50 – 10:10 AM)

Epilepsy and Autonomic Nervous System
Ekrem Kutluay, MD

Epilepsy is one of the most common neurological disorders. It is known that people with epilepsy have higher mortality rates than general population. Seizures, especially if they are poorly controlled, may have effects on an autonomic nervous system. These effects can be in the form of cardiovascular, respiratory, sexual or gastrointestinal. However, cardiovascular and respiratory changes during epileptic seizures are “hot topic” now considering their possible role in SUDEP. Several studies revealed different forms of cardiovascular changes associated with seizures. Ictal tachycardia is probably the most common one reported in 80% or more seizures. Ictal bradycardia and asystole is significantly less common but consequences are more threatening. Brain regions involved in autonomic control include limbic structures (amygdala, orbitofrontal cortex, insula and cingulate), hypothalamus, periaqueductal gray matter and autonomic medullary centers. Also, some cortical stimulation studies revealed possible lateralized representations with bradycardia elicited by left insular stimulation and tachycardia by right. Seizures are also associated with respiratory depression and apnea. Studies revealed prolonged ictal apnea and significant drop in oxygen saturation even in patients whose seizures did not generalize. Neurophysiological tests also revealed altered autonomic regulation of the cardiovascular system in patients with epilepsy during interictal state.

Neurophysiological Testing of Autonomic Nervous System
Safwan Jaradeh, MD

The Autonomic Nervous System (ANS) is responsible for homeostasis. It is divided into central and peripheral components, also known as pre-ganglionic and post-ganglionic. It is responsible for the innervation of multiple organs. It has 3 major components: Parasympathetic, Sympathetic and Enteric. Cardiovagal function testing consists mainly of Heart rate response to deep breathing (HRDB) and the Valsalva ratio (VR). HRDB variability also forms the basis for high frequency power spectral analysis.

Adrenergic function consists mainly of BP responses to the Valsalva maneuver and the BP responses to Head Upright Tilt. HRDB variability also forms the basis for low frequency power spectral analysis. Microneurography measures muscle sympathetic nerve activity. Sudomotor testing evaluates sympathetic cholinergic function. It consists of Sympathetic (galvanic) skin response (Electrodreemalactivity), Thermoregulatory sweat test, Resting sweat output, Quantitative sudomotor axon reflex test and Silastic sweat imprint method. Autonomic testing is important in the evaluation of suspected Autonomic dysfunction, Small fiber neuropathies (painful), Syncope, Orthostatic intolerance (hypotension, tachycardia), Sweating disorders, GI or GU motility disorders, Extrapyramidal and cerebellar disorders, dizziness with dysautonomia, vasomotor rhinitis, GERD, and sleep apnea. The value of testing as it relates to disorders, Extrapyramidal and cerebellar disorders, dizziness with dysautonomia, Autonomic dysfunction, Small fiber neuropathies (painful), Syncope, Orthostatic endurance is a tool to evaluate the cardiovascular-Respiratory Autonomic function and has been shown to be a useful complimentary tool when CEEG is not available, but if seizures are suspected on aEEG, CEEG should begin as soon as possible to confirm the diagnosis.

Interpretation of aEEG
Courtney Wusthoff, MD

Dr. Wusthoff will provide an introduction/refresher overview of how to interpret amplitude-integrated EEG. A tutorial will be given on the two most widely used background scoring systems for aEEG in term neonates. Patterns suggestive of seizures will be presented. Unique findings in preterms and special populations will be considered. These principles will be applied to example cases, leading to discussion of how neonatologists interpret aEEG in practice.

Current Applications of aEEG in the NICU
Tammy Ishida, MD PhD

Amplitude integrated EEG (aEEG) is being used more frequently in Neonatal Intensive Care Units (NICUs). aEEG enables the NICU to detect subclinical seizures, so that neonatal seizures can be identified and appropriately managed. In addition, there is literature to support the use of aEEG as a tool for prognosis after hypoxic ischemic injury. However, there are limitations to aEEG. In addition, the type of aEEG recording can affect the sensitivity of aEEG for seizures and background abnormalities. This talk will present the strengths and weaknesses of aEEG and optimal recording parameters.

Impact of aEEG Use in the NICU
Cecil Hahn, MD, MPH

Amplitude integrated EEG (aEEG) monitoring of newborn infants, originally pioneered in Europe, has now become commonplace in neonatal intensive care units (NICUs) throughout North America. In most NICUs, aEEG monitoring is applied by bedside caregivers and interpreted by neonatologists, with varying input from clinical neurophysiologists. This presentation will review the available evidence on the impact of aEEG use on clinical practice in the NICU, including the diagnosis of seizures, the use of antiepileptic drugs, the use of neuroimaging, and the frequency of neurology consultations. I will discuss how the availability of aEEG use has influenced the

Principles of aEEG
James John Riviello, MD

Continuous EEG (CEEG) is an essential tool for ICU monitoring. Digital trending analysis (DTA) compliments CEEG, using mathematical signal transformations based on amplitude, frequency, or power, displayed on a compressed time scale. DTA is useful to visualize long-term trends, especially seizures, EEG background, and sleep-wake cycling. aEEG modifies EEG data, filtering frequencies less than 2Hz and greater than 15Hz, rectifying and smoothing the signal, has a linear display for 0-10µV and logarithmic display for 10-100µV, on a time-compressed display at 6cm/hour. The leads are placed over the central (C3, C4) or parietal (P3, P4) regions. Frontal lead placement has more muscle artifact. Limitations include: limited electrodes, low amplitude or short duration seizures may be obscured by time compression; artifact contamination with “raw EEG” data needed to identify artifact; interpreter experience. aEEG machines now use dual channel recordings (C3,P3 and C4, P4), and display the “raw EEG” channel. CEEG is the gold standard for neonatal seizure detection and quantification and should be used whenever available. aEEG may be a useful complimentary tool when CEEG is not available, but if seizures are suspected on aEEG, CEEG should begin as soon as possible to confirm the diagnosis.
pattern of conventional EEG use, and reflect on the complementary roles of aEEG and continuous EEG monitoring.

**EEG AS A BASIC NEUROSCIENCE AND PSYCHOLOGY RESEARCH TOOL (10:30 AM – 12:30 PM)**

**ERPs from Basic to Clinical Research: Toward Biomarkers**
Greg Hajcak, PhD

This talk will focus on early electrocortical activity that has been shown to index response monitoring: the error-related negativity (ERN) is a negative deflection in the response-locked ERP that is maximal just 50 milliseconds following an erroneous response at midline frontal recording sites. Both source-localization and intracerebral recordings suggest that the ERN is generated in the anterior cingulate cortex (ACC)—a brain region that integrates information about negative emotion, pain, and cognitive control. An increased ERN has consistently been found in anxious patients—even after successful treatment. Anxiety, but not comorbid anxiety/depression, is characterized by an increased ERN, and this relationship emerges early in development. Unaffected first-degree relatives of anxious individuals also have an increased ERN. In addition to genetic mechanisms, I’ll describe learning-related differences may underlie the association between ERN and anxiety. In The ERN demonstrates excellent test-retest reliability, and appears to be a trait-like biomarker of certain forms of anxiety.

**Use of EEG and MEG in Studying Oscillatory in Normal and Pathological Brain States**
Michael Gandal, MD, PhD

Autism and schizophrenia are neurodevelopmental disorders characterized by deficits in social and cognitive function. The lack of efficacy of current medications for these impairments represents a significant obstacle for the treatment of these disorders. Developing novel therapeutics to target these symptoms requires appropriate neural biomarkers that can be investigated in model organisms; be used to track treatment response, and provide insight into pathophysiological disease mechanisms. A growing body of evidence indicates that neural oscillations in the gamma frequency range (30-80 Hz) are disturbed in autism autism schizophrenia. Gamma synchrony has been shown to mediate a host of sensory and cognitive functions, including perceptual encoding, selective attention, salience, and working memory. This talk will demonstrate that autism and schizophrenia are characterized by an elevation in baseline cortical gamma synchrony (‘noise’) coupled with reduced stimulus-evoked GBRs (‘signal’). This endophenotype can be recapitulated by acute or chronic NMDA-receptor hypofunction on pyramidal neurons in mice. Such signal-to-noise deficits are tied to changes in excitatory/inhibitory balance within glutamatergic afferents and can be reversed pharmacologically by boosting tonic inhibition. These data demonstrate a clinically relevant, highly translatable biomarker for preclinical therapeutic development across a host of disorders that share common endophenotypes.

**Development of Biomarkers and Clinical Tests from Basic EEG Research**
Joshua Even, MD

Clinicians who interpret clinical tests and those who order them must understand precisely what the test results mean—and with what level of confidence those results inform the care of the patient. Tests may be based on measurements whose relationship to the mechanism of disorder are clearly understood (such as presented by the previous two speakers) or on more obscure measurements; either way, a number of experimental design and statistical considerations must be taken into account when evaluating a diagnostic test. The goal of this talk will be to review a number of key aspects of diagnostic test/biomarker development and evaluation, as they relate to EEG-based methods.

**INTRAOPERATIVE NEUROPHYSIOLOGIC MONITORING DURING FUNCTIONAL NEUROSURGERY (10:30 AM – 12:30 PM)**
Mark Stecker, MD, PhD; Peter Dempsey, MD; Jay Shils, MD, PhD

The data obtained during the neurophysiologic acquisition process is needed to plan the next surgical steps (i.e., what would the recommendation be given a recording with minimal STN and a significant portion of thalamic activity—also, what role does the state of the patient and the specific disease have on the decision process). Also, how can the OR data be used in the post-operative management of the patient? These concepts will play a large role in the interactive elements of this session.

The neurophysiologists’ interpretive skills and how they convey information affects how the surgeon uses that data in their decision process. IONM data interpretation may cause the surgeon: (1) to stop the surgery; (2) modify their initial plan; (3) revise something they have done. Any of these changes are potentially beneficial or detrimental to the patient. Thus, communication and trust are critical factors in the surgeon-neurophysiologists relationship in the operating room and understanding these are key element to beneficial patient outcome.

This session will present a short theoretical basis for functional surgical approaches to treatment of movement disorders. The rest of the session will present case examples with an interactive discussion, involving all three speakers and the audience.

**EXTENDING CRITICAL CARE EEG MONITORING TO COMMUNITY-BASED PRACTICE (12:50 – 1:50 PM)**

**Mixing General Neurology and ICU Monitoring**
Jennifer Jones, DO

Abstract for Mixing general neurology and ICU monitoring.

This portion of the talk will include a review of issues relating to incorporating ICU EEG monitoring into a general neurology practice. Specifically, the topics to be covered include (1) what are the different models of employment and reimbursement for ICU monitoring (2) what are time management considerations in a general neurology practice including how to cover weekends and nights (3) how often and in what manner to communicate updated reports with ICU team (4) what is necessary to train EEG technologists to best utilize the technology?

**Starting an ICU Monitoring Program in a Community Hospital**
Evan Fertig, MD

Traditionally, ICU monitoring has been performed in intensive care units based in academic, tertiary medical centers. As private-practice based epilepsy programs grow, many groups are starting ICU monitoring programs in smaller, community-based hospitals, where there is a significant unmet need. This talk will focus on strategies to implement a successful program. Topics will include education of professional staff,
INTERESTING SPINAL CORD TUMOR CASES: A DISCUSSION BY SOME EXPERTS
(12:50 – 1:50 PM)

Interesting Spinal Tumor Cases: A Discussion by Some Experts
Francesca Sala, MD, PhD; David Gloss, MD; Mirela Simon, MD; Eva Ritzl, MD

Introduction: Intramedullary spinal cord tumors (ISCT) still represent a challenge for neurosurgeons. In the past 30 years, about 550 ISCTs have been operated on at the Institute of Neurosurgery in Verona. Since 2000, all ISCT surgeries have been performed with the aid of intraoperative neurophysiological monitoring (IONM). Material and Methods: We reviewed our experience in 240 ISCTs operated on with an IONM protocol including somatosensory evoked potentials (SEPs), transcranial muscle motor evoked potentials (mMEPs), spinal MEPS (D-wave) and the bulbocavernous reflex (BCR). Selected cases are presented for discussion.

Results and Conclusions: 1) The vast majority of patients with ISCT present some degree of transient neurological worsening early after surgery, in spite of the use of IONM. 2) The neuro-oncological aspects of ISCT surgery are relevant and the IONM professional must include these in establishing warning IONM criteria. 3) The goals of IONM should be tailored to different ISCT pathology (ependymomas, astrocytomas, hemangioblastoma, ...). 4) The main factor limiting the extent of resection of ISCTs remains the cleavage plane between tumor and spinal cord, yet IONM continue to play an essential role in this surgery.

SEMINIOLOGY OF STATUS EPILEPTICUS IN ADULTS (2:00 – 4:00 PM)

Semiology of Status Epilepticus in the Responsive Patient
Frank Drislane, MD

Nonconvulsive status epilepticus (NCSE) produces a remarkable variety of altered behavior – and prominent ‘non-behavior.’ This session will concentrate on the signs and symptoms of NCSE in patients who retain some, if limited, responsiveness. Before integrating EEG evidence, it is worthwhile to record the semiology precisely, to help avoid confusion between the seizure and the epilepsy syndrome. Some NCSE patients have seemingly global dysfunction, with immobility and staring. This can be of a primary generalized (absence) nature or focal-onset NCSE (often considered complex partial SE). The EEG, however, can be misleading as to the nature of the seizures. Simple and complex automatisms can occur in both focal-onset and generalized NCSE; careful description of the semiology may help define the syndrome. “Hypermotor” seizures may also occur without complete unresponsiveness.

Finally, some NCSE appears quite focal or regional in character, with perceptual and cognitive deficits very similar to those seen in patients with fixed focal lesions such as strokes and tumors. Failure to recognize that such semiology can result from NCSE can lead to major misdiagnosis. Among these cases are aphasia, amnesia, hemianopia, and neglect syndromes. Difficulties in diagnosis will be detailed.

Correlation of Semiology and EEG Pattern in Comatose Patients
Peter Kaplan, MD, FRCP

There is an extensive literature on EEG patterns in encephalopathy and comatose patients. EEG patterns range from reactive or unreactive patterns of theta, theta/ delta and delta; alpha and spindle coma tracings; and periodic waves in the form of triphasic waves. Various constellations of epileptiform activity occur that span the interictal-ictal continuum, with patterns of periodic discharges appearing focally, bilaterally, asynchronously and synchronously. Isolated seizures may appear focally or in a generalized pattern and when in the form of status epilepticus may be continuous or exhibit discrete seizures. Semiology during unresponsiveness varies from the minimally unreactive patient to the deeply comatose, the latter with either intact or variably absent brainstem reflexes, motor responses to pain and myoclonus. Clinical - EEG correlations in the context of etiology have prognostic import as well as providing a yardstick for management of coma and seizures or status epilepticus in the intensive care setting.

Intraoperative Neuromonitoring Below the Belt (2:00 – 4:00 PM)

Bulbocavernous Reflex; Anal Sphincter and Cremaster Electromyography; Pelvic Autonomic Monitoring
Stanley Skinner, MD

The great advantage of the BCR is its ability to detect low sacral segmental conduction block at both the peripheral somatic (afferent or efferent) and central levels. Because parasympathetic fibers are associated with sacral roots of the cauda equina, BCR affords surrogate testing of major bowel, bladder, and sexual vegetative functions. Obviously, for surgery within (or affecting) the lower pelvic periphery, sacral root/plexus, cauda equina, and conus medullaris... BCR implementation should be very strongly considered.

Extracellular recordings within the corpora (perivaginal and penile erectile tissue) may be tentatively termed “electromyography.” Although intriguing animal research has been published, rigorous but flexible thinking will be required 1) to exclude possible artifacts during slow wave recording (< 2 Hz) and 2) to accept the realities of unsummarized responses in the OR. Near field bipolar recording by electrodes that are shielded from skin activity and can reject other artifactual common mode signals will be required. The promised benefit of an electrophysiological approach lies in the recording of extracellular field potentials without waiting for a fickle end organ physical change (like strain gauges around the penis). Electromyographic recordings should be equally applicable in both sexes.

Pudendal Nerve and Sacral Root Evoked Potentials
Matthew Eccher, MD, MSPH

Originally described in 1982, scalp SSEP responses can be recorded following stimulation of multiple different pudendal-nerve- and sacral root-supplied structures. The resulting P40 response is usually highest amplitude at Cz. Responses are generally easy to resolve and therefore should be of equivalent ease to follow for neurophysiologic intraoperative monitoring versus lower limb peripheral nerve SSEPs (e.g., tibial or fibular [peroneal] nerves), but sizeable reports of pudendal SSEP monitoring are few. Direct orthodromic sensory nerve action potential (SNAP) recording from the cauda equina in response to single such sacral stimuli has been reported of utility for preserving roots that participate in urinary control during dorsal rhizotomy procedures for spasticity. Technical application of both techniques is quite
straightforward. As in most areas of NIOM, there are no well-constructed historical-control series informing use of these techniques, and certainly no clinical trials. Given the socially devastating consequences of urinary and anal continence disturbances, and a fairly high rate of functional postoperative disturbances when sacral roots are manipulated, this field begs more active clinical investigation.

Neurophysiology of Complex Spinal Cord Untethering
Francesco Sala, MD

Introduction: Intraoperative neurophysiologic monitoring (IOM) is nowadays extensively used to minimize neurological morbidity in tethered cord surgery. We present a 10-year clinical experience using a multimodal IOM approach in 47 patients with a tethered cord secondary to a variety of spinal dysraphisms. Material and Methods: Neurophysiological mapping of the cauda equina was performed through direct rootlet stimulation and bilateral recording from segmental target muscles in the lower limbs, and anal sphincters. Monitoring techniques included somatosensory evoked potentials (SEPs), transcranial motor evoked potentials (MEPs) from limb muscles and anal sphincters, and the bulbocavernosus reflex (BCR). Results: Neurophysiological mapping was successful in all cases. In six patients an unexpected muscle response was evoked by stimulating tissue macroscopically considered as not functional. The monitorability rate was 84% for SEPs, 97% for limb muscle MEPs, 74% for anal sphincter MEPs, and 59% for the BCR. SEPs, MEPs and BCR remained stable during surgery in all patients but one. This child showed worsening of a lower limb paresis and urinary/fecal incontinence, which completely recovered over 8 and 5 weeks respectively. Conclusion: Combining mapping and monitoring IOM techniques allow to safely address tethered cord surgery.

SPECIAL INTEREST GROUP: CRITICAL CARE EEG MONITORING AND OUTCOMES: DO WE HAVE ENOUGH DATA? (4:30 – 6:00 PM)

cEEG in the Adult ICU (Seizure Indications)
Jeffrey Kennedy, MD

The past few decades have witnessed a revolution within the fields of epilepsy and intensive care medicine through a significant increase and interest in continuous EEG monitoring (cEEG) in adult intensive care units. Advancements in technology leading to digital recording and remote review have allowed practical and feasible cEEG to be performed. Currently, the major application of cEEG in ICU is primarily to diagnose paroxysmal events, detect nonconvulsive seizures, and nonconvulsive status epilepticus (NCSE). cEEG monitoring has also revealed further understanding of associations of NCs and NCSE with several pathological processes commonly encountered in the ICU setting as well as identified several EEG patterns, whose clinical significance remains yet to be elucidated.

Update of Current Practices
Jay Gavvala, MD

The use of continuous EEG (cEEG) monitoring in the ICU has increased dramatically and is frequently used in a variety of clinical settings. Unlike other procedures or imaging modalities, at present time, there are no practice parameters or clinical guidelines to ensure a minimal standard for the completion and reporting of cEEG monitoring in addition to basic requirements for all staff involved. In a recent survey aimed to describe current practice among neurophysiologists and neurointensivists, practices are similar among neuro-intensivists and neurophysiologists regarding primary indications for utilization of cEEG. However, there is wide variability regarding recommended duration of monitoring and secondary indications for cEEG. Among neurophysiologists, there was large practice variability in the technical aspects of cEEG including frequency of review and use of EEG technologists in screening cEEG. These results have demonstrated that while use of cEEG has become more frequent with greater agreement for primary indications of cEEG monitoring, there is large practice variability in both use for indications other than seizures as well as cEEG reporting frequency, utilization of technologists processing time and other aspects of the practice of cEEG monitoring. Further large prospective multicenter studies are needed to clarify the optimal utilization of cEEG.

cEEG in the Neonatal ICU
Courtney Wusthoff, MD

The arguments for and against cEEG in the Neonatal ICU will be discussed, citing currently available evidence supporting each side of the debate. This session will critically evaluate the data regarding how cEEG monitoring for neonatal seizures and background predicts and impacts ultimate clinical outcomes. Priorities for future research will be considered by the group.

cEEG in the Pediatric ICU
Cecil Hahn, MD, MPH

In this presentation, I will review the current practice of continuous EEG monitoring in North American pediatric ICUs, summarize the available epidemiological data on the prevalence and risk factors for seizures among critically ill children, and discuss our current knowledge of the relationship between electrographic seizures and outcomes. I will highlight the gaps in knowledge regarding outcomes, and discuss research strategies to address these gaps.

EMG SIG (4:30 – 6:00 PM)

Conduction Block is Essential for diagnosis of MMN
Michael Cartwright, MD, MS

Multifocal motor neuropathy (MMN) is a rare condition with a prevalence of 1 to 2 per 100,000 individuals. It affects men more often than women and typically starts with slowly progressive asymmetric upper extremity weakness without sensory involvement. Differentiating MMN from motor neuron disease is critical, because MMN responds to immune modulating treatment, typically intravenous immunoglobulin. Conduction block is considered the electrophysiologic hallmark of MMN, but there is a lack of consensus as to whether conduction block is essential for the diagnosis of MMN. This presentation will review the data supporting and refuting the need to demonstrate conduction block prior to diagnosing and treating MMN.

Skin Biopsy is the Best Diagnostic Test for Assessment of Small Fiber Neuropathy
Morris Fisher, MD

Small fiber neuropathies are common. They are clinically important and frequently associated in an isolated fashion with prominent pain. The clinical findings in SFN may be minimal and the usual electrophysiologic studies unrevealing even if other electrophysiologic studies of autonomic dysfunction may be abnormal. Skin biopsies with evaluation of intraepidermal fiber densities (IEFD) are a well-established, safe, and readily available technique for defining SFN. The clinical features, evaluation, and
Ultrasound Improves Sensitivity in the Diagnosis of Entrapment neuropathy
Francis Walker, MD

Neuromuscular ultrasound is a recent addition to many EMG laboratories that provides complementary anatomic, and to some extent, physiologic information regarding entrapment neuropathies. Determining the value of this additional information is not as straightforward as it might seem because there is no gold standard for the diagnosis of nerve entrapment. It is tempting to use electrodiagnostic criteria as the gold standard, but then no test could be shown to add to sensitivity to electrodagnosis. Clinical criteria for entrapment neuropathies are problematic in that they are not particularly sensitive nor specific. In practice, ultrasound sometimes is positive in patients with classical clinical features of the disease when electrodiagnostic studies are normal; the reverse is also true. As ultrasound techniques have improved, there has been an improvement in the accuracy in diagnosing carpal tunnel syndrome, and in some studies, ultrasound shows superior sensitivity. For ulnar neuropathy at the elbow, ultrasound offers some advantages over electrodagnostic testing, particularly when there is ulnar nerve subluxation. Clearly, ultrasound adds information not routinely available by electrodiagnostic studies, particularly in terms of pathology in adjacent structures, anatomic variants, and dynamic aspects of nerve movement.
Cortical and Subcortical MEP Stimulation
Stuart Hoffman, DO
Despite ongoing advances in neuronavigation imaging tools to aid in tumor resection, neurophysiologic mapping and monitoring of motor pathways remains the gold standard when supratentorial tumors are located near the motor strip and subcortical motor pathways. Intraoperative neurophysiologic mapping and monitoring of these pathways aids the neurosurgeon by improving the odds of obtaining a total resection of the tumor while avoiding a new post-operative deficit. This talk will focus on the fast-train monopolar electrical stimulation technique for mapping and monitoring cortical and subcortical motor pathways during neurosurgical resection of tumors near these important pathways. The history of neurophysiologic mapping and monitoring of motor pathways will be briefly reviewed to put the current practice in context. Pertinent neuroradiology and neurophysiology regarding these techniques will be highlighted and the basic neurophysiologic set up will be presented. Other techniques, including 60 Hz bipolar stimulation will be reviewed and contrasted with fast-train techniques. Intraoperative alert criteria, subcortical intensity-distance mapping data, and the pitfalls and limitations of these neurophysiologic techniques will be presented.

DCS-MEP for Epilepsy Surgery: Current Evidence and Questions
Matthew Eccher, MD, MSPH
In the years since the first 1993 report of fast-train monophasic direct cortical stimulation, there have been several small reports regarding this technique in the epilepsy population. The clear advantage appears to be that there is a lower rate of induced seizures with this technique compared with biphasic 50- to 60-Hz long train stimulation. There are several comparative disadvantages, however, including inapplicability of this technique for sensory or language mapping, absence of FDA-approved equipment for this technique, and, perhaps most challenging, an absence of good head-to-head comparison of testing results between this technique and the older one. What literature there is in this area will be reviewed, some illustrative cases discussed, and next research steps for the field suggested.

DCS-MEP and CST Motor Threshold: Utility for Cerebral Hemispheric Lesion Surgery
Kathleen Seidel, MD
Mapping and monitoring are believed to provide an early warning sign to avoid damage to the corticospinal tract (CST) during brain surgery. We compared subcortical monopolar short train stimulation thresholds (MT) with direct cortical stimulation (DCS) motor evoked potential (MEP) monitoring signal abnormalities in 100 patients. At 3 months, 5 patients had a postoperative new motor deficit (lowest mapping MT 20mA, 13mA, 6mA, 3mA, 1mA). All showed DCS-MEP alterations (2 sudden irreversible threshold increases and 3 sudden irreversible MEP losses). Of these cases, 2 had vascular lesions (MT 20mA, 13mA) and 3 had mechanical CST damage (MT 1mA, 3mA and 6mA; in the latter two resection was continued after mapping and DCS-MEP alterations occurred thereafter). Mapping should primarily guide tumor resection adjacent to the CST. DCS-MEP is a useful predictor of deficits, but its value is limited because signal alterations were reversible in only approximately 60% of cases and irreversibility is a post-hoc definition. The true safe mapping MT is lower than previously thought. We postulate a mapping MT below 1 mA where irreversible DCS-MEP changes and motor deficits regularly occur. The limited spatial and temporal coverage of contemporary mapping may increase error and may contribute to false higher MTs.

Surgical Approach: Concept and Technique
Jorge Alvaro Gonzalez-Martinez, MD, PhD
In many centers in North America, invasive monitoring for medically intractable focal epilepsy is performed using subdural grids/strips, combined or not with depth electrodes. Most recently, the stereo-electroencephalography (SEEG) methodology has been applied in patients with difficult to localize seizures when a deep focus is suspected or a more wide spread epileptogenic network is responsible for the generation and early propagation of the epileptic activity. The SEEG method provides a 3 dimensional perspective that with the appropriate surgical hypothesis, encompasses necessary and previously untargetable brain regions. Since resective surgery has such potential for benefit, SEEG is an investigational modality that should complement traditional approaches and provide intractable epilepsy patients with surgical options otherwise unavailable to them.

Pediatric Stereo EEG: Challenges and Opportunities
Jonathan Miller, MD
Focal epilepsy in infancy and early childhood often is not associated with abnormalities on imaging because incomplete myelination interferes with the recognition of cortical dysplasia on MRI. Even when a lesion is present, MRI and video-EEG sometimes fail to adequately localize the epileptogenic zone. In these cases, invasive monitoring can be helpful to determine whether resection is feasible and to define the margins of resection. Stereo-EEG (SEEG) involves placement of multiple intracerebral electrodes to sample brain tissue directly. When thoughtfully planned, the individual contacts become “voxels” that can be used to plan the subsequent resection. Very young children present unique challenges related to the ability of the skull to accommodate anchoring devices and the tolerance of young children to prolonged invasive monitoring in general. Also, unlike widespread coverage with a subdural grid, each individual SEEG electrode samples only a single dimension, which can lead to a “tunnel vision” effect unless the arrangement of electrodes is carefully tailored according to a good working hypothesis defined by a multidisciplinary approach. When appropriately applied, SEEG can help to identify focal epilepsy in a majority of pediatric epilepsy patients with minimal morbidity.
**SUNDAY, FEBRUARY 9, 2014**

**CLINICAL NEUROPHYSIOLOGY TRIALS IN THE NEUROINTENSIVE CARE UNIT: FOCUS ON TRENDS (10:15 AM – 12:15 PM)**

**TRENDS: From Conception to Implementation**

Aatif Husain, MD

The Treatment of Recurrent Nonconvulsive Seizures (TRENDS) study is the first study of its kind in which the efficacy of two antiepileptic drugs (AEDs) is being compared for the treatment of electrographic seizures using continuous EEG monitoring. This is an industry sponsored, investigator initiated study (IIS). The challenges and rewards of designing and implementing a multicenter, randomized IIS will be discussed.

**Participating in a Clinical Trial**

Jong Woo Lee, MD, PhD; M. Brandon Westover, MD PhD

There are far too few evidence-based treatments for patients with critical neurological illness, including the treatment of nonconvulsive seizures. Clinical trials represent the best and most important method available to address these questions. This symposium will provide an overview of designing, implementing, and executing a clinical neurophysiology based trial. We will focus on the multicenter trial “Intravenous Lacosamide Compared with Fosphenytoin in the Treatment of Patients with Frequent Nonconvulsive Seizure (TRENDS).” Issues and challenges specific to neurocritical care patients will be addressed.

**Next Steps in Clinical Neurophysiology Trials**

Suzette LaRoche, MD

Over the past decade, there has been considerable increase in the utilization of continuous EEG monitoring in critically ill patients, particularly in patients with acute brain injury. However, in the age of rising healthcare costs, administrators demand evidence of “return on investment” prior to funding evolving technologies. Unfortunately, it remains unclear exactly what influence EEG monitoring for detection and treatment of secondary injuries such as seizures and ischemia has on outcome. Retrospective studies have shown that electrographic seizures are common, yet prospective data on how treatment of seizures affects outcome measures such as length of stay and functional recovery is lacking. Nonetheless, findings on EEG frequently result in treatment changes and can have a large impact on clinical decision making. Therefore, the challenge is to devise clinical trials that might provide better outcome data but also strive to provide more efficient and cost-effective EEG monitoring. This session will discuss the next steps that are necessary to design and implement neurophysiology trials that would answer these critical clinical questions.

**CRASHING THE CULTURES OF THE SOLE MEG OR EEG SOURCE MODELING: INSEPERABLE, NOT ONLY COMPLEMENTARY (10:15 AM – 12:15 PM)**

Anto Bagic, MD, PhD; Richard Burgess, MD, PhD; John Ebersole, MD

Even after the optimal combined acquisition of MEG and EEG for identification of epileptic foci, it is the prevailing practice in clinical MEG to perform source localization only of MEG, while source localization of EEG is usually an exception rather than the rule. Overall in fact, despite the recommendation and customary inclusion of simultaneous EEG during MEG, different centers use EEG quite variably, from being completely ignored, to being a pointer to “interesting” corresponding MEG discharges for source localization, to being used to corroborate authenticity and relevance of MEG discharges, and finally on rare occasions to being combined with MEG for electromagnetic source imaging. Furthermore, software to import and localize EEG and MEG may not be available from the MEG instrument manufacturer. Thus, while we profess the complementary nature of MEG and EEG, it is evident that this complementarity is exploited quite incompletely. To highlight the critical issues regarding EEG and MEG source localization, we will have an expert discussion of this issue in a classic debate format.

**NEONATAL AND PEDIATRIC EEG: PATTERNS OF EPILEPTIC ENCEPHALOPATHIES ACROSS THE AGE RANGE (10:15 AM – 12:15 PM)**

**Evolution Over Time in Neonatal Epileptic Encephalopathies**

Courtney Wusthoff, MD

Dr. Wusthoff will consider EEG patterns in the neonatal epileptic encephalopathies. Illustrations of specific findings will be presented, including those in KCNQ2 encephalopathy, Ohtahara syndrome, and congenital hemimegalencephaly. Typical evolution of EEG findings in the neonatal period and infancy will be considered, particularly in relationship to etiology and outcome. This session will include a focus on debunking myths about EEG findings in specific diseases.
**F1**

**Utility of Continuous EEG Monitoring in Acute Stroke**
Sanjay Menon, MD; Sandipan Pati, MD; M. Brandon Westover, MD PhD; Eric Rosenthal, MD

Objectives: 1) To assess the utility of continuous EEG monitoring in critically ill patients with acute ischemic stroke, and 2) its impact on clinical decision-making, especially with regard to antiepileptic drug management.

Study methods: Single centre, retrospective study involving 54 consecutive adult patients admitted to the neurosciences intensive care unit following acute ischemic stroke over the last three years who underwent continuous video EEG monitoring (cEEG).

Results: Fifty-four patients with the mean age of 67+/-11; half were men. Etiologies were: primary brain injury (25%; n=5); systemic illness (30%; n=6); cardiac arrest (35%; n=7); and other (10%; n=2). Most were comatose (55%; n=11). 35% (n=7) died; the majority had significant disability upon discharge (mRS 3-4; 55%; n=11). The ICC for main terms generalized and periodic was moderate (0.50; 95% CI 0.09-0.77). For 10 cases all raters agreed were GPDs, the ICC for the descriptor “triphasic” was 0.90 (0.78-0.97). For 3 raters who agreed all 20 cases represented GPDs, the ICC for the descriptor “triphasic” was 0.78 (0.53-0.91; Tables 1 & 2).

Experienced raters largely agree which discharges are “triphasic,” but they only moderately agree on what constitutes a GPD pattern. Concrete and quantitative criteria must be developed before GPDs (and therefore triphasic waves) are used for objective clinical interpretation.

**F2**

**Generalized Periodic Discharges: Inter-Rater Agreement**
Advait Mahulikar, MD; Prasanna Tadi, MD; Jonathan Halford, MD; Jan Claassen, MD, PhD; Suzette LaRoche, MD; Brandon Foreman, MD

Generalized periodic discharges (GPDs) are a common in the critically ill. Certain GPDs are described as “triphasic,” and are associated with a purely metabolic encephalopathy. However, the reliability of this distinction is not clear.

A prospective cohort of 79 patients with GPDs was collected. A representative cohort was chosen (n=20; Figure 1) and intraclass correlation coefficients (ICCs) were calculated across 11 raters.

Mean age was 69+/-11; half were men. Etiologies were: primary brain injury (25%; n=5); systemic illness (30%; n=6); cardiac arrest (35%; n=7); and other (10%; n=2). Most were comatose (55%; n=11). 35% (n=7) died; the majority had significant disability upon discharge (mRS 3-4; 55%; n=11). The ICC for main terms generalized and periodic was moderate (0.50; 95% CI 0.09-0.77). For 10 cases all raters agreed were GPDs, the ICC for the descriptor “triphasic” was 0.90 (0.78-0.97). For 3 raters who agreed all 20 cases represented GPDs, the ICC for the descriptor “triphasic” was 0.78 (0.53-0.91; Tables 1 & 2).

Experienced raters largely agree which discharges are “triphasic,” but they only moderately agree on what constitutes a GPD pattern. Concrete and quantitative criteria must be developed before GPDs (and therefore triphasic waves) are used for objective clinical interpretation.
Sensitivity of QEEG for Seizure Detection in the ICU

Hiba Anif, MD; Rosana Esteller, PhD; Suzette LaRoche, MD

AIM: Evaluate sensitivity of QEEG for seizure detection in the ICU.

Methods: EEG epochs were subjected to QEEG analysis based on amplitude, power, rhythmicity and asymmetry using Persyst® software. Each epoch was reviewed in 3 formats: Raw EEG(R), QEEG+Raw EEG(QR) and QEEG(Q). Neurophysiologists marked seizure onsets for each format. Sensitivity and false positive rates were calculated for Q and QR formats using seizures marked by raw EEG reviewers as gold standard.

Results: Mean sensitivity for QEEG alone ranged from 51-70% and from 63%-69% for Q and QR formats using seizures marked by raw EEG reviewers as gold standard.

Response Rates in Anticonvulsant Trials for Triphasic Waves

Deirdre O’Rourke, MB BC; Nicolas Gaspard, MD, PhD; Brandon Foreman, MD; Patrick Chen, AB; Lauren McClain, BA; Advait Mahulikar, MD; M. Brandon Westover, MD PhD

Background: Triphasic waves (TPWs) occur in metabolic encephalopathies and non-convulsive status epilepticus (NCSE). The utility of benzodiazepine (BZDs) or non-sedating anti-epileptic drug (NSAEDs) trials commonly used to differentiate is debated.

Methods: Three institutions within the Critical Care EEG Monitoring Research Consortium retrospectively identified patients with unexplained encephalopathy, TPWs, and results of BZD and/or NSAEDs trials done to differentiate ictal vs. non-ictal patterns. We assessed responder rates and compared metabolic profiles of responders and non-responders.

Results: 64 patients were identified. Most (71.9%) were admitted with metabolic derangements and/or infection. Positive clinical responses occurred in 8 (15.1 %) given BZDs. Responses to NSAEDs occurred in 19/45 (42.2%), being immediate in 6.7%, delayed but definite in 20.0%, and delayed but equivocal in 15.6%. Overall, 27/64 (42.2%) responded to either BZDs or NSAEDs. Metabolic differences between responders vs. non-responders were nearly all statistically insignificant (Figure 1).

Conclusions: Similar metabolic profiles in patients with encephalopathy and TPWs between responders and non-responders to anticonvulsants suggest predicting responders a priori is difficult. The high responder rate (42%) suggests trials of anticonvulsants indeed provide useful clinical information. The nearly 3-fold higher response rate to NSAEDs suggests this strategy may be preferable to BZDs. Further prospective investigation is warranted.

Continuous EEG monitoring in ICUs allows recognizing clinically invisible deteriorations. It is rarely used since manual review is time-consuming. NeuroTrend is a software developed for analysis of EEGs from critically ill patients. Main Terms, the Major Modifiers absolute amplitude and frequency, and sporadic epileptiform discharges according to the ACNS’ standardized critical care EEG terminology, and moreover rhythmic theta and alpha activity are determined and presented graphically.

EEGs from 10 ICU patients (avg. duration 19h, min. 11h, max. 30h) with an available clinical neurophysiological report were used. An EEG expert new to these EEGs was asked to write quick-reports using only 10 minutes of time per patient and NeuroTrend, which were then compared to the original clinical reports: In three out of five patients with reported seizures all seizures were found. One patient had 15 clinical subtle seizures (only two with clear EEG correlation), another patient had one seizure with unclear EEG correlation, which had been missed. LPD were correctly found in 4/5 patients, and GPD/GRDA was correctly found in 2/2 patients. In 7/9 patients slowing was also correctly reported. In two patients episodes of generalized rhythmic theta activity were found, which had not been mentioned in the original report.

Seizure and EEG in Autoantibody Positive Limbic Encephalitis

Ning Zhang, MD, PhD; Teddy Youn, MD; Emily Ho, MD, PhD; Shu-Ching Hu, MD, PhD; Shahin Hakimian, MD

Rationale: Seizures are commonly presentation in limbic encephalitis (LE) associated with neuronal auto-antibodies. Methods: We identified 13 limbic encephalitis patients with anti-NMDAR, 5 patients with anti-VGKC in a retrospective screening of all patients who underwent testing for auto-antibodies in serum/CSF from 2009-2012. Eight serology negative patients with clinical diagnosis of LE were also included in the analysis. Results: limbic encephalitis with anti-NMDAR had a more uniform course with sequential clinical manifestations. Two patients with anti-NMDAR had electrographic seizures or status, and five had “extreme delta brushes” on EEG. Limbic encephalitis with anti-VGKC had more variable clinical pictures with less severe symptoms. Seizures were the prevailing presentation at the onset of anti-VGKC limbic encephalitis, of which lateralizing or focal epileptiform discharges were well correlated with clinical semiology. In limbic encephalitis with anti-NMDAR, 57% patients had a distinct pattern of posterior hypometabolism in the occipital lobes and cerebellum on FDG-PET, whereas in other types of LE there was no distinct pattern. Conclusions: Limbic encephalitis with anti-NMDAR has characteristic multistage clinical presentations, and it is strongly associated with posterior hypometabolism on PET and with the extreme delta brush pattern on EEG. Seizures and focal/lateralized epileptiforms are more prevalent in patients with anti-VGKC.
**F9**

**Pentobarbital in Super-Refractory Status Epilepticus (SRSE)**

Trisham Gyang, MD; Michael Mendoza, MD; Julius Latorre, MD, MPH

Pentobarbital-induced electroencephalogram (EEG) burst suppression is a treatment for SRSE. Reported length of treatment varies from 7-244 hours with mortality of 77%. We present 3 patients with SRSE on prolong Pentobarbital with good outcome.

**Results:** When PP is the first finding in 542 patients, 45 (8.3%) subsequently developed EEGSZ. Of 543 who had EEGSZ as the first finding, 52 (9.5%) had PP on the same day or afterwards. Comparing PP characteristics in the first 24 hrs of detection, the presence of sharp or polyspike morphology strongly correlated with later seizure risk (p < 0.001) while TW and undifferentiated patterns did not. Etiology of hypoxia/anoxia correlated with death, and absence of EEGSZ a better outcome.

Conclusions: PP with sharp or spike/polyspike morphology correlated positively and TW negatively with seizure risk. Hypoxic/anoxic etiology was a major factor for death.

**Methods:** Retrospective identification of patients 18 or older, with PP, EEG seizures (EEGSZ), or both, studied by Continuous EEG between January 2011 and December 2012. PP included triphasic waves (TW), but excluded unilateral PLEDs. Selected 20 min files of PP on their first day of appearance were analyzed blindly for morphology, frequency, and topography in patients who later did (n=39) or never developed EEGSZ (n=101) and correlated to discharge outcome. Analysis was performed using JMP 9.0 (USA) statistical software.

**Results:** Twenty-three year-old male with lepto-meningoencephalitis of unclear etiology developed SRSE with sharp waves in the fronto-central region, treated with pentobarbital coma for 13 days and 4 antiepileptic medications (AEDs). He was discharged home AED mono-therapy. On follow-up he was back to previous functional.

Fifty-three year-old female with multiple acute infarcts developed SRSE with right central parietal spike waves, treated with pentobarbital coma for 19 days with 5 AEDS. She was discharged to rehabilitation facility on 3 AEDs. On follow-up she was back to functional baseline.

It was shown that poor outcome is related to etiology of seizure and comorbidities. This series reflect good outcome regardless of seizure etiology. With meticulous medical treatment of complications and appropriate AEDs, prolonged pentobarbital coma is therapeutic and safe.

---

**F10**

**EEG features of cyclic alternating pattern in sleep and coma**

Valia Rodriguez Rodriguez, MD, PhD

In this study we compared features of the cyclic alternating pattern observed in coma and NREM sleep. Features we used were source configuration, changes in synchronization features and graph theoretical properties of the EEG. Data was obtained from the slow-wave (SW) and non-slow-wave (NSW) periods observed during the continuous EEG monitoring of six critical ill patients -3 of them in coma. Results showed that: 1-configuration of source generators for delta and theta frequencies during SW and NSW was similar in both patient groups; 2- regional synchronization during SW and NSW tended to be higher in parietal and parieto-occipital regions of coma patients and in frontal regions of non-coma patients; 3- in both groups, SW had a more ordered network structure with higher clustering and smaller path length; 4- local clustering seemed to increase from NSW to SW but network global efficiency seemed to decrease; 5- coma and non-coma patients differed in local network efficiency; this did not vary in coma patients but increased during SW in non-coma patients. The results are preliminary but consistent at the patient level. Absence of regional synchronization in frontal regions and lower local efficiency may be important for the incapacity to wake from coma.

**F11**

**Periodic Patterns Related to Seizures, Etiology and Outcome**

Yod Pinnorj, MD

Introduction: Data on how periodic patterns (PP) correlate with seizure risk, etiology and outcome are needed.
Focal interictal epileptiform discharges (FIET) often consist of a train of peaks with a duration of 10 ms or less. They were examined for variation of FIET geometry with combined use of conventional polygraphic montages and polygraphic channel overlay (PGCO) with gain, frequency domain and temporal resolution maximized, could provide better temporal segmentation and definition of FIET against background (FIG). PGCO with gain, frequency domain and temporal resolution maximized, could provide better temporal segmentation and definition of FIET against background (FIG). PGCO with gain, frequency domain and temporal resolution maximized, could provide better temporal segmentation and definition of FIET against background (FIG).

We present the case of a 25 year old Hispanic female, who presented to the ED with status epilepticus. She has had frequent hospital admissions since her seizures started six months ago, described as tonic clonic jerking. Her episodes have been associated with receptive and expressive aphasia, changes in personality, aggression, and flat affect. Work up revealed a normal MRI, intermittent slowing on EEG (Fig. 1), and lymphocytic pleocytosis with 4+ oligoclonal bands. NMDA receptor antibody encephalitis was highly suspected. Intravenous Solumedrol was given, with minimal improvement. Serology sent came back positive for NMDA receptor antibodies. Anti-N-methyl-D-aspartate receptor (NMDAR) antibody encephalitis is a paraneoplastic syndrome affecting younger women, characterized by psychiatric symptoms, autonomic instability, neurologic abnormalities, and tonic-clonic type of seizures. CSF usually reveals lymphocytic pleocytosis, and MRI findings are non-specific. Diagnosis is confirmed with serology. A large percentage of patients diagnosed also present with a detectable tumor, the most common of which is ovarian teratoma. Anti-NMDA receptor antibody encephalitis is very responsive to treatment. The first line of management includes steroids, immunoglobulins, and plasma exchange. Rituximab and cyclophosphamide have shown to improve outcome in refractory cases.

Focal interictal epileptiform discharges (FIET) often consist of a train of peaks with a duration of 10 ms or less. They were examined for variation of FIET geometry with combined use of conventional polygraphic montages and polygraphic channel overlay (PGCO). PGCO with gain, frequency domain and temporal resolution maximized, could provide better temporal segmentation and definition of FIET against background (FIG). FIET varied in duration and propagation within each data set, including propagation between brain hemispheres and lobes. When short in duration, FIET varied in location without propagation. Complexity of FIET geometry reflects evolution of refractory partial epilepsy. Some FIET revealed pre-base shift prior to first peak. FIET in cursor-synchronized polygraphic montages (a1, b1) and PGCO (a2, b2) (AVG: common average reference). FIET peaks form true phase reversals in PGCO (b2). Compare with FIET (propagation 10 ms) in Abstract 1.079, Matsuo, 2012 AES Annual Meeting (www.aesnet.org).

F16
Yield of Ambulatory EEG: Not Beyond 13 Hours
Maria Siddiqui, MD; Jeffrey Jirsch, FRCP; S. Nizam Ahmed, FRCP
Purpose: This study aimed at evaluating the value added by 24 hour ambulatory EEG (AEEG) by comparing the presence of epileptiform discharges (EDs) between the first 30 minutes of recording vs. the following 23.5 hours.

Methods: A retrospective review of AEEGs of subjects divided into 2 groups, epilepsy and questionable epilepsy was conducted. AEEGs were divided into routine EEG equivalent (first 30 minutes) and extended EEG (remaining 23.5 hours). Extended EEGs were further divided into segments (S): 31st minute to 8th hour (S1), 9th to 16th hours (SII) and 17th to 24th hours (SIII). Each consecutive segment was reviewed to identify new EDs not seen previously.

Results: Fifty seven AEEGs were included, age range of subjects being 20 to 59 years. There were 38 (66.6%) females. In epilepsy group (46), additional yield of extended EEG was 34.1% (14/41) as opposed to 10.9% (5/46) in routine EEG equivalent. It was as follows: S1-29.3% (12/41), SII-6.9% (2/29), SIII-0/27 (Figure 1). The yield however, did not increase beyond 13th hour. In questionable epilepsy group (11), yield was 0/11 in all segments.

Conclusions: There was no value added for yield of EDs by extending the EEG recording: (1) Beyond 13 hours in epilepsy group. (2) Beyond 30 minutes in questionable epilepsy group.

F17
Demyelination Masked by Severe Axon Loss: Repeating NCS
Christina Chrisman, MD; Shafeeq Ladha, MD; Suraj Muley, MD
Background: In neuropathy patients with severe axonal degeneration but without demyelinating conduction slowing in the lower extremities and with normal nerve conduction velocities in the upper extremities, the presumed primary pathology is thought to be that of axonal degeneration.

Case Report: An 82-year-old man developed numbness from the feet to mid-calf level over 2 years. Examination showed normal strength and reflexes in the arms, areflexic legs, and impaired sensation to the mid-calf level. Nerve conduction studies showed reduced motor and sensory amplitudes and relative preservation of conduction velocities indicating an axonal neuropathy. Arm conduction velocities were normal.

Eighteen months later, he developed global weakness and numbness in all extremities. Examination revealed distally accentuated arm and leg weakness with areflexia. Sensation to all modalities was reduced distally in all limbs. Severe conduction slowing was seen in the arms consistent with demyelination. Laboratory testing showed elevated G01A IgM and IgG, as well as elevated CSF protein (105 mg/dl). A diagnosis of CIDP was made.

Conclusion: This case illustrates that in patients with severe axonal neuropathy, nerve conduction studies should be repeated since demyelinating changes that were initially masked by axon loss in severely affected nerves can become overt as the neuropathy evolves.
**F18**

**Predictors of Positive Repetitive Nerve Stim in Myasthenics**
Raghav Govindarajan, MD; Denny Reyes, MD; Efrain Salgado, MD

Background: There are few studies which have systematically analyzed the clinical and serological markers predicting positive repetitive nerve stimulation (PRNS) in a large newly diagnosed myasthenic cohort.

Methods: This is a retrospective chart review of all newly diagnosed adult myasthenics who had an RNS study with at least one year follow-up between 2001 and 2011. Each chart was carefully reviewed and myasthenic symptoms classified into one of the five: general, severe, axial, bulbar, ocular. RNS and Ach antibodies were classified either as positive or negative and where available Ach antibody levels were recorded.

Results: 44 patients were included (mean age: 60y, M: F 17:27, Caucasians: 68%, Hispanics: 16%). If age increases the odds of presenting with a proximal limb weakness decreases $\chi^2 = 10.17, p<0.01$. Proximal limb weakness had the strongest association with thymus hyperplasia/thymoma ($p=0.053$) and with Caucasian race $\chi^2 = 4.74, p<0.01$. If age increases, the odds of positive RNS decreases $\chi^2 = 7.41, p<0.01$. Irrespective of patient’s symptoms trapezius had the best yield for PRNS at 46%.

Conclusion: Age is the best predictor of PRNS. Trapezius had the highest yield for PRNS irrespective of presenting symptom including nonspecific symptoms such as generalized weakness and fatigue.

**F19**

**Predictors of an Incomplete Electrdiagnostic Study**
Raghav Govindarajan, MD; Katya Kurako, MD; Virgilio Salanga, MD, MS

Introduction: Incomplete electrodiagnostic (EDX=NCV/EMG) study can lead to inaccurate diagnoses and affect reimbursement.

Objective: Systematically and prospectively assess the predictors of performing an incomplete EDX in a large cohort of diverse patient population.

Methods: This was a prospective, non-randomized, single-blinded, self-administered, paper-based, close-ended and scaled questionnaire study of consecutively referred adult patients for an office EDX between August 2012 and November 2012. Patients were administered standard visual pain scale prior to EDX and then after nerve conduction studies and electromyography (EMG). A 4 item-3 factor Likert scale “EDX Awareness Questionnaire” was administered at the end of each study.

Results: 304 patients with 304 studies (mean age 95% CI:57.9 -1.41), 61% male, 54% Caucasians were included. 41% were referred for a radiculopathy, 50% of tests were ordered by non-neurologists. Age had an inverse correlation with pre-test EDX pain perception (p<0.05). 19 studies were incomplete with 67% of them not completing EMG. Patients who had higher pre-test pain perception for EMG were more likely to stop either of tests (p<0.001). Patients who had additional online pre-test information reported higher pre-test EMG pain perception and post-test EDX pain perception (p<0.05).

Conclusion: Patient’s pre-test pain perception of EMG appears to be a significant risk factor for incomplete studies with older patients reporting lower pre-test pain scores and patients who had online pre-test information reporting higher pain scores.

**F20**

**Automatistic Behaviors in Temporal Lobe Epilepsy**
Abuhuziefa Abubakr, MD; Ilse Wambacq, PhD

Purpose: To compare the clinical features of automatism between right and left TLE.

Method: We retrospectively reviewed the features of clinical seizures in 48 consecutive patients (27 females and 21 males) with TLE, age range 18–65 years. 24 patients had left and 24 had right TLE focus.

Results: Manual automatism is most frequent occurred in 83% and ipsilateral in 58% right and 62.5% left and bilaterally in 25% and 20.8% respectively. Pedal and oro-alimentary automatism occurred in both sides (20.8% and 20.4% vs. 16.7% and 14.6% respectively). Vocalization occurred in 33% of the left and 20.8% of the right TLE. Face wiping occurred in 6.25% of the cohort (1 right and 2 left), all are non-lateralizing (p = 0.16). Nose wiping is second frequent occurred in 45.8% of all patients and ipsilateral to the focus in 86.4% (70% left vs. 20.8% right), and significantly frequent in the right (p = <0.001). Genital manipulation occurred in two patient one on each side. Water drinking and retching plus vomiting occurred only in right TLE (4.1%).

Conclusion: Some automatistic behaviors may differentiate between right and left TLE, such as nose wiping, water drinking and retching plus vomiting to the right temporal focus.

**F21**

**A Retrospective Study of cEEG Monitoring in the NICU**
Arnold Sansevere, MD; Jacquelyn Klehrn, BA; Iván Sánchez Fernández, MD; Tobias Laddenkemper, MD

Purpose: To describe the main clinical and electroencephalographic (EEG) characteristics of neonates who underwent continuous EEG monitoring in the neonatal intensive care unit (NICU).

Methods: Retrospective study of 20 patients aged less than 1 month who underwent clinically indicated continuous video-EEG monitoring (cEEG) in the ICU at Boston Children’s Hospital during 2012.

Key findings: Of the 20 patients 65% were male, 85% were term, and monitoring began at a mean age of 2.31 days after admission/birth. Event characterization was the main indication for cEEG. The median (p25-p75) duration of cEEG monitoring was 1 (1-2) days. Mortality was high: five patients (20%) died prior to leaving the hospital. The most common EEG pattern consisted of multifocal spikes/sharp waves. Eleven (55%) patients had electrographic seizures, most of which were focal or multifocal and most lasted less than 5 minutes. Seven of 11 (63.6%) seizures had no clinical correlate.

Significance: cEEG provides important clinical information in selected NICU patients given the frequency of electrographic seizures without clinical correlates (35%).

**F22**

**Delayed Status Epilepticus After Acute Baclofen Overdose**
Fawad Khan, MD; Eugene Ramsay, MD

Background: Baclofen is widely used for the treatment of spasticity. Acute baclofen withdrawal has been reported to precipitate seizures in patients with no prior history of seizures.
Objective: We report a case of acute oral baclofen overdose resulting in excessive sedation and subsequent status epilepticus.

Results: A 50 year-old female presented to the emergency room after consumption of 25 baclofen tablets. Within 5 hours she became increasingly lethargic and hypotensive. Continuous EEG monitoring was initiated and approximately 48 hours after the overdose independent left and right lateralized epileptiform activity was noted. 59 hours after the overdose the patient was in non convulsive status epilepticus (NCSE). This was refractory to several doses of intravenous lorazepam. Three antiseizure medications and intravenous anesthetics (Propofol and Ketamine) successfully treated the NCSE. The patient was maintained on monotherapy with no further seizures.

Conclusion: Considering the short half life of baclofen (8 hours) and the 59 hours delay in onset of NCSE, we propose that the etiology was acute withdrawal of baclofen. Continuous EEG monitoring was valuable in early detection of the NCSE and is recommended in patients with overdose of baclofen.

F23
Risk Factors for Pediatric Convulsive Status Epilepticus
Iván Sánchez Fernández, MD; Kush Kapur, PhD; Jacquelyn Klehm, BA; Sookee An, BA; Dinesh Jillella, MD; Jaqueline Zelener, BS candidate; Alexander Rotenberg, MD PhD; Tobias Loddenkemper, MD

Purpose: To identify risk factors of pediatric convulsive status epilepticus (SE).

Methods: Retrospective cohort study of patients 1 month-21 years presenting with convulsive seizures.

Key findings: One thousand sixty-two patients (54% males) met inclusion criteria. Four hundred forty-four (41.8%) patients had SE ≥30 minutes. Compared to their respective control groups, patients with SE (defined with a cut-off value of either 5 or 30 minutes) were younger at the age of seizure onset and at the age of SE, were taking more antiepileptic drugs (AEDs) at baseline, had a higher rate of changes in AEDs during the three months prior to the episode, were more likely to have developmental delay at baseline, and had a higher mortality rate during comparable follow up intervals. Patients with SE had a higher baseline seizure frequency, and a higher increase in seizure frequency prior to the index episode that only reached statistical significance with the 5-minute cut-off.

Significance: This series identifies risk factors which independently predict convulsive SE in pediatric patients and that are similar when considering a 5-minute or a 30-minute cut-off for the definition of SE.

F24
Comparison Status Epilepticus 5-29 vs. More Than 30 Minutes
Jacquelyn Klehm, BA; Iván Sánchez Fernández, MD; Martina Vendrame, MD PhD; Kush Kapur, PhD; Seifee Uysal, MD; Mustafa Gedik, MD; Sookee An, BA; Dinesh Jillella, MD; Jaqueline Zelener, BS candidate; Sana Syed, MD; Vasi Gooty, MD; Alexander Rotenberg, MD PhD; Tobias Loddenkemper, MD

Objective: To compare the characteristics of pediatric patients with status epilepticus (SE) lasting 5-29 minutes (SE_5-29) with those lasting ≥30 minutes (SE_30).

Methods: Retrospective cohort study of patients 1 month-21 years presenting with seizures lasting at least 5 minutes.

Results: 445 patients (50.1% male) with a median (p 25-p 75 ) age of SE of 5.5 (2.8-10.5) years were enrolled. SE lasted for 5-29 minutes in 296 (66.5%) subjects, and for ≥30 minutes in 149 (33.5%). Patients with SE_30 were younger than patients with SE_5-29 at time of seizure onset and at time of SE episode. SE as first seizure presentation was more frequent in patients with SE_30 (Table 1). There was a tendency towards a higher rate of abnormalities in the magnetic resonance imaging at baseline in patients with SE_30 (Table 2). Differences were not detected in seizure frequency, seizure types, presence of developmental delay, and electroencephalogram abnormalities at baseline. On multivariate analysis, each additional minute of SE duration increased the odds ratio of death by 0.005 after adjusting for age and length of follow-up (Table 3).

Conclusions: Baseline characteristics were similar in patients with SE_30 and SE_5-29. Longer duration of SE correlated with higher mortality in this population.

F25
Sleep Misperception in Persons with Epilepsy
Marcus Ng, MD, FRCP; Matt Bianchi, MD, PhD

Being able to confidently ascertain the amount of sleep is critical to the clinical management of epilepsy. Sleep misperception is the phenomenon in which an individual underestimates the amount of time spent asleep. Little is known about sleep misperception in patients with epilepsy. We conducted retrospective chart reviews on individuals who self-identified as having epilepsy in a questionnaire database of patients who underwent polysomnography (PSG) in a sleep laboratory at a quaternary medical center. We confirmed 64 patients with epilepsy in the database. For total sleep time (TST) and sleep latency (SL), we calculated the difference between that reported by the patient on questionnaire and that measured by PSG. The median TST underestimation was 45 minutes (-180 to 60, p<0.05) and the median SL overestimation was 20 minutes (-10 to 170, p<0.05). This pattern of misperception is similar to that reported in patients with insomnia. There was no statistically significant difference based on categorical epilepsy refractoriness, cognitive impairment, or psychiatric comorbidity. Our findings suggest that sleep misperception is prevalent in patients with epilepsy which has important implications for the reliability of the clinical history for sleep assessment.

F26
Nonconvulsive Status Epilepticus Manifesting as Pure Alexia
Ning Zhong, MD, PhD; Jeremy Cholfin, MD, PhD; Jonathan Kleinman, MD; Dawn Eichasiv, MD; John Stern, MD

Pure alexia often results from lesions of the left angular gyrus or the left posteroior temporal lobe. We describe a case of pure alexia with visual symptoms due to nonconvulsive status epilepticus (NCSE). This 55-year-old man presented with sudden onset of confusion, not able to read, and a focus of bright light in his peripheral right visual field. His EEG was diagnostic of NCSE originating from the left temporo-occipital region, manifested as numerous 30 second to 2 minute runs of polyspike-wave complexes with evolvement to 5-6Hz disorganized sharply contoured theta activities intermixed with rhythmic 2Hz delta activities in the left parieto-occipital region. During the seizures, the patient complained bright flashing strobe light in right visual field, and that he could not read with preserved ability of communication, naming/repeat, and writing. His MRI showed hyperintensity bordering the sulci of the left temporal and occipital lobe with leptomeningeal enhancement. After
F27

SSEP in therapeutic Hypothermia Era: Still a Valuable Tool
Carolina Maciel, MD; Ching Tsao, MD; Elayna Rubens, MD

Objectives: The reliability of somatosensory evoked potentials (SSEP) in predicting outcome in comatose survivors of cardiac arrest undergoing therapeutic hypothermia (TH) has been questioned. We investigated whether the absence of cortical responses was a reliable predictor of nonawakening in the setting of TH. Materials and Methods: A retrospective review was conducted in cardiac arrest survivors admitted to a single tertiary care hospital from 04/2010 to 03/2013 treated with TH who had SSEP performed. N20 responses were categorized as normal, present but abnormal, and bilaterally absent. Neurologic outcome was assessed at time of discharge with Cerebral Performance Categories Scale (CPC). Results: Ninety-three SSEP studies were performed in 74 patients. Fifteen patients had absent N20 responses; all had poor outcome (CPC 4-5). Seven patients had absent N20s during hypothermia, 3 had follow up SSEPs after rewarming redemonstrating absent cortical responses. Sixty-seven patients had N20 peaks identified and had variable outcomes. Evaluation of one or more peaks was limited in 19% of SSEPs performed during cooling due to presence of artifact. Conclusion: SSEPs remain a reliable prognostic indicator in patients undergoing TH, even when performed during cooling. Technical challenges are commonplace during TH and caution is advised in interpretation of suboptimal recordings.

F28

Evaluation of P40 and CCT in 42 Patients with ALS
Teresa Maria Monte de Oca Dominguez, Dr.; Juan Manuel Rojas de Dios, Lic.; Idalma Padron Lopez, Lic.; Gladys Maya Morales, Lic.; Olga Gonzalez Perez, Lic.

ALS is a resulting upheaval of the progressive degeneration of the neurons of the motor cortex that give origin to the corticospinal tracts and motoneurons of the previous spares medular and the nuclei of the motor cranial nerves. The combination of clinical and electrophysiological criteria would allow demonstrating still subclinical alterations in the diagnosis of the ALS with use of PESS in early states. Objectives: Determine changes of latency P40 and CCT in lumbar area in ALS. Methods: Study of 42 patients (27 men, 15 female) with the criteria of inclusion, and diagnostic of ALS confirmed or probable criteria. Methods: Study of P40 and CCT localized in L1. Results: Normal latency of P40 in 10 patients (25%) and the absolute latencies were bilateral increased in 32 patients (75.6%). The CCT was absent in 7 patients (21.8%) and 5 patients (15.6%) with CCT prolonged. Conclusion: Presence of interconnections between the system and pyramidal neurons in the sensory areas, given the influence of the motor cortex in the ascending sensory pathways, we might explain these findings. The absent of CCT lumbar would have any relations with problems in medulla or encephalic area in specifics. The PESS1 would help in future to understanding the progress and possible cure to ALS.

F29

IONM Utility in Altering Operative Management of Aneurysms
Forough Ghavami, DO; Viet Nguyen, MD; Scheherazade Le, MD; Leslie Lee, MD; S. Charles Cho, MD; Gary Steinberg, MD, PhD; Richard Jaffe, MD, PhD; Michael Marks, MD; Robert Dodd, MD, PhD; Huy Do, MD; Jaime Lopez, MD

Methods: Retrospective study of 445 adult cerebral aneurysm cases. Surgical and endovascular cases were monitored using electroencephalography, somatosensory, brainstem auditory and motor evoked potentials. Critical IONM changes were based on accepted criteria in the literature. Postoperative neurologic deficits were identified through chart review.

Results: 87/445 (19.5%) procedures demonstrated IONM changes. 73/87 (83.9%) had transient IONM changes; 52 had no new deficits, 11 with new transient deficits, and 10 with new permanent deficits. 14/87 (16.1%) had persistent IONM changes; 2 had no new deficits, 2 with new transient deficits, and 10 with new permanent deficits. 358/445 (80.4%) cases had no IONM changes; of these 7 (1.9%) had new postoperative deficits (5 transient, 2 permanent). Operative management was altered in all instances of IONM changes. The sensitivity and specificity of predicting postoperative deficits was 82.5% and 87.1%, respectively.

Conclusions: Persistent IONM changes predict a high risk for new postoperative deficits, while transient changes predict a lower risk. In addition, the data indicates that identification of IONM changes allows for intraoperative interventions, likely associated with better patient outcome. These results support the usefulness of IONM as an adjunct in cerebral aneurysm treatment.

F30

Isolated Loss of tcMEPs with Intracranial Aneurysm Clipping
Leslie Lee, MD; Gary Steinberg, MD, PhD; Robert Dodd, MD, PhD; Steven Chang, MD; Jaime Lopez, MD

INTRODUCTION: Cerebral ischemia following aneurysm clip placement is a known procedural risk. Neurologic injury may result through compromise of blood flow directly from parent or adjacent branch vessels. The role of intraoperative neurophysiologic monitoring (IONM), and specifically transcranial motor evoked potentials (tcMEPs), in helping to prevent such injuries is highlighted.

METHODS: We present a series of seven surgical cases performed for treatment of middle cerebral (5), anterior communicating (1), and posterior communicating (1) artery aneurysms, where primary critical changes in tcMEPs occurred following clip placement.

Conventional techniques for acquisition of potentials were utilized. Multimodality IONM was employed in all cases, including transcranial motor evoked potentials, somatosensory evoked potentials, and electroencephalography.

RESULTS: In all cases critical loss of tcMEPs was observed following clip placement, without other IONM changes. Prompt identification of tcMEPs changes uniformly led to rapid surgical assessment, with eventual removal and/or repositioning of aneurysm clips in six cases, and an increase in cerebral perfusion in one case, which resolved neurophysiologic changes and correlated with no new sustained postoperative deficits.

CONCLUSIONS: This case series highlights the critical importance of tcMEPs in the early identification of potentially reversible IONM changes that may correlate with impending injuries related to aneurysm clip placement.
F31
Saphenous Nerve SSEP during Lateral Interbody Fusion
Lilit Mnatsakanyan, MD; S. Samuel Bederman, MD, PhD; Heriberto Guillen, CNIM; Daniel Yanni, MD

BACKGROUND: Lateral lumbar trans-psoas interbody fusion (LIF) is novel minimally invasive technique reducing risks related to traditional anterior and posterior approach surgeries. Despite advantages, trans-psoas exposure carries up to 30% risk of lumbar plexus injury. The intraoperative neuromonitoring (IONM) is utilized to prevent postoperative deficits, but to date there is no reliable technique to evaluate upper lumbar plexus.

Methods: Saphenous nerve SSEPs were obtained by stimulation of inferior medial thigh with needle electrodes and recording from scalp. Primary outcome was measured by testing reproducibility of SSEPs at baseline, changes during procedure and relevance to standard IONM modalities. Intraoperative changes were identified and correlated to primary outcome.

Results: Twenty-nine patients were included in the study. Reliable saphenous SSEP were recorded bilaterally in 26/29 patients. Reduction of amplitude > 50% in 2 cases was observed during expansion of the tubular retractor in psoas muscle. The posterior tibial SSEP remained unchanged. The saphenous returned to baseline after collapsing the retractor.

Conclusions: Saphenous SSEPs can be used to detect electrophysiological changes to prevent femoral nerve injury during LIF. Larger sampling size is underway as to validate whether this technique offers increased sensitivity/specificity, and correlates with the postoperative outcomes.

F32
Challenges in NIOM with Patients Undergoing TEVAR
Mohini Gurme, MD; Evgeny Tsimerinov, MD PhD; Jeffrey Chung, MD; Robert Zelaya, CNIM; Dawn Elashiv, MD

Severe neurologic deficit remains a significant concern in patients undergoing thoracic endovascular aortic repair (TEVAR). Spinal cord ischemia with subsequent peri-operative neurologic deficit occurs in 10-30% of TEVARs. One approach is to utilize neuro-intraoperative monitoring (NIOM) of motor and somatosensory evoked potentials (TcMEP and SSEP) to assess spinal cord function and guide protective surgical strategies.

Hypothesis: Combined TcMEP/SSEP monitoring is a useful approach that may prevent neurologic complications due to spinal cord ischemia during TEVAR but complication rates remain high.

Method: We reviewed the most current NIOM database in one center utilizing combined TcMEP/SSEP monitoring and standard surgical procedures, including hypothermia, to minimize peri-operative complications. We identified 13 consecutive TEVARs performed over a 4 month period.

Results: In two cases there was a significant change in both SSEP and MEP waveforms. In one case, there was post-operative focal neurologic deficit. Waveforms returned with rewarming in one case raising the issue of the effect of hypothermia on NIOM.

Conclusion: This study supports the importance of NIOM with TEVAR; however, a high neurological complication rate persists despite NIOM monitoring. Studies are needed to assess the threshold of changes and the effect of hypothermia on NIOM.

F33
ECoG-guided Cortical Resection: Outcomes in Children
Seema Bansal, MD; Sookyong Koh, MD; Douglas Nordli, MD; Andrew Kim, MD

Objective: To determine surgical outcomes and intraoperative interictal abnormalities in pediatric patients undergoing single-stage electrocorticography-guided cortical resection at a single institution.

Methods: We retrospectively analyzed 136 patients who presented with seizures at or before age 18 years. Comparisons were made between those patients who were and were not seizure-free at various time points following surgery.

Results: The mean duration of follow up was 3.2 years. At latest follow up, 76% of patients were seizure-free. Older age at onset of seizures and shorter duration of epilepsy prior to surgery were associated with a favorable outcome (p<0.05). Daily seizures prior to surgery were associated with worse outcomes (P=0.001). There was a trend toward better outcomes in patients undergoing temporal resections (p=0.082). Interictal abnormalities (attenuation and/or spikes) did not correlate significantly with outcome; however, of the patients with cortical dysplasia or frontal resection, those with attenuation tended to have better outcomes (p<0.1).

Conclusion: Longer duration of epilepsy, younger age at onset, and increased frequency of seizures are associated with worse outcomes following epilepsy surgery. While interictal spikes on intraoperative electrocorticography do not correlate with outcome, interictal attenuation may be useful in predicting seizure-freedom in certain patient populations.

F34
IONM Changes from Positioning: Acetabulum Fracture Cases
Shaile Gowda, MD; David Betts, CNIM, R. EEG/EP T.

Purpose: To describe intraoperative neurophysiologic monitoring (IONM) changes in acetabulum fracture repair cases due to positioning.

METHODS: We report two cases of acetabulum fracture repair treated with open reduction and internal fixation (ORIF). IONM involved Posterior tibial nerve (PTN) and Peroneal nerve (PN) SSEPs.

RESULTS: Case 1: 41 y/o male underwent left ORIF. Baseline SSEP responses were obtained from right lower extremity (LE). Thirty minutes into surgery, gradual sequential emergence of peripheral, spinal followed by cortical response was noted. It was felt acquisition of signals was due to reduction in edema of left LE due to limb positioning. SSEPs on the non-operative side remained stable during the entire course of the surgery. Case 2: 26 y/o female with left both column acetabulum fractures had left foot in traction boot. Baselines SSEP responses were established. Forty minutes later, there was loss of left PTN SSEPs. Traction boot was loosened and responses returned and fully restored upon complete removal of the boot.

CONCLUSION: Factors such as edema and positioning can cause absence or loss of SSEPs. Employing corrective measures can regain SSEPs allowing IONM to be continued during surgery thus preventing premature abortion of monitoring or suspecting neural injury.
F35
IONM of Adult and Pediatric Moyamoya Surgery
Viet Nguyen, MD; S. Charles Cho, MD; Leslie Lee, MD; Nadia Khan, MD; Gary Steinberg, MD, PhD; Richard Jaffe, MD, PhD; Jaime Lopez, MD; Scheherazade Le, MD

We examined the utility of intraoperative neurophysiologic monitoring (IONM) to detect markers of cerebral infarct or hemorrhage in the surgical revascularization of moyamoya.

700 moyamoya revascularization cases (435 patients, 1994-2010) were analyzed. IONM involved bilateral upper extremity SSEPs and 8-lead parasagittal scalp EEG. Postoperative events, including new strokes and hemorrhages within the first postoperative day, were analyzed. “Persistent” IONM changes did not resolve by the end of monitoring, whereas “transient” changes did.

Twenty-nine cases had new strokes, yet a correlating IONM change was present in only 4. Three cases developed intracerebral hemorrhage; one showed a correlating IONM change. Twenty-three cases (3.3%) had any IONM changes. All 4 cases with “persistent” changes had postoperative events (3 strokes, 1 hemorrhage). “Transient” changes correlated with an absence of postoperative events (17 of 19 cases).

IONM using bilateral upper extremity SSEPs and 8-lead parasagittal scalp EEG is a specific but not sensitive predictor of postoperative strokes or hemorrhages in moyamoya revascularization. Modification of monitoring techniques may be necessary. Persistent IONM changes correlated with postoperative stroke or hemorrhage; transient changes did not. It is possible that some events occurred postoperatively instead of intraoperatively, or that ischemic changes occurred beyond the territories monitored.

F36
Comparison of Seizure Outcome after Amygdalohippocampectomy
Aradia Fu, MD; Steve Chung, MD

Objective: The primary goal was to compare seizure outcome after selective amygdalohippocampectomy (SAH) for patients with temporal lobe epilepsy (TLE) based on age of seizure onset. The secondary goal was subgroup analysis of seizure outcome for patients with and without mesial temporal sclerosis (MTS).

Background: Previous studies have assessed seizure outcome in adult and pediatric patients after standard temporal lobeectomy, but there is a paucity of data comparing outcome after SAH.

Design/Methods: We retrospectively reviewed patients who underwent SAH. These patients were grouped into two: adult onset patients (AO) with age of seizure onset >18 and pediatric onset patients (PO) with age of seizure onset ≤18. We further divided the groups based on presence or absence of MTS (+ vs. -). Seizure outcome was measured one-year postoperatively according to the Engel’s classification.

Results: Total of 40 AO and 64 PO were identified. There was no significant difference in seizure outcome between AO vs. PO (p=0.65), +AO vs. +PO (n=77, p=0.43), and -AO vs. -PO (n=27, p=0.17).

Conclusions: We did not find significant difference in seizure outcome between adult and pediatric onset TLE after SAH. Furthermore, presence or absence of MTS prior to SAH had no significant impact on seizure outcome.

F37
A Method for Measuring Central Nervous System Motor Output
Barry McKay, BS, R.EEG.T; Joy Bruce, PT, PhD; Leslie Vanhuel, PT, D.Sc.PT; Raymond Alexander, D.P.T.; Keith Tansey, MD, PhD

Currently, clinical assessment of motor function is carried out using expert-examiner scales to categorically grade perceived muscle strength or complex sets of functional skills. Such scales have limited sensitivity and reliability, and differ across neurological diagnoses. A neurophysiological approach is being developed to augment those scales by quantitatively measuring features of motor control using surface electromyography (sEMG) as the closest non-invasive vantage point from which central nervous system motor behavior can be viewed. Multichannel/muscle sEMG registers the rate and distribution of changes in the excitability of the spinal motoneuron pools managing the contraction of the recorded muscles. Using a standardized protocol of reflex and volitional motor tasks, this method quantitatively describes central motor control across the spectrum from paralyzed to paretic and finally, to fully recovered using values calculated in relation to the patterns acquired from neurologically intact subjects. Pathophysiological changes in motor control such as spasticity are also captured using this method. Thus, the severity and form of motor control loss experienced after neurological injury or disease can be neurophysiologically quantified and tracked during recovery, exacerbation and treatment.

F38
Relationship Between Sleep Spindles and OSAS Severity
Yu Jin Lee, MD, PhD; Jong Won Kim, PhD; Yu-Jin Lee, MD; Do-Un Jeong, MD, PhD

Introduction: Synaptic plasticity is known to play a key role in the generation of sleep spindle, which suggests occurrence of sleep spindle might give some information on brain dysfunctions associated with obstructive sleep apnea syndrome (OSAS). Especially, fast spindles were reported to relate with the cognition more closely than slow spindles. In current study, we aimed to investigate spindle activities of sleep EEG in young (<30 years) and elderly (55>years) OSAS.

Methods: 72 EEG recordings of OSAS patients' data (young group: 36, mean age 25.8±5.6; elderly group: 36, mean age 57.6±1.1) during nocturnal polysomnography (PSG) were analyzed. Spectral analysis was performed with the qEEG-PSG program (CIRUS, Australia) for fast (13-17Hz) and slow (11-13Hz) spindle components.

Results: The OSAS severity (AHI) showed no significant difference between two groups (20.9±14.7 vs 27.0±17.8, p=0.114). There was no significant difference in the slow/fast spindle ratio (0.941±0.193 vs 0.999±0.166, p=0.174). The slow/fast spindle ratios were significantly correlated with AHI in young group (r=-0.005, p=0.012) but not in elderly group (r=-0.001, p=0.174).

Conclusion: Current results showed that sleep spindle activities were associated with OSAS severity in only young patients. This result suggests fast spindle might be a potential indicator of brain plasticity in young population.
F39
Automatic Artifact Removal for Long-Term EEG Monitoring
Manfred Hartmann, DI; Kaspar Schindler, PhD; Tineke A. Gebbink,;; Gerhard Gritsch, PhD; Tilmann Kluge, PhD

A method for automatic removal of artifacts from EEG recordings (PureEEG), which is based on an electrophysiological model, was developed and evaluated. In a validation study artifact removal performance and attenuation of true EEG patterns was investigated. In a multiple-choice questionnaire two independent reviewers assessed 102 twenty-second epochs from seizure onsets of 48 consecutive epilepsy patients. The amount of artifacts before and after PureEEG processing and attenuations of true EEG patterns were evaluated. “Major improvements” due to PureEEG were found by the reviewers in 59% and 49% of the EEG epochs respectively, “minor improvements” in 38% and 47% of the epochs. The answer “similar or worse” was chosen in 0% and 4% respectively. Neither of the reviewers found “major attenuations”, i.e., a significant attenuation of significant EEG patterns. Most EEG epochs were found to be either “mostly preserved” or “all preserved”. A minor attenuation was found in 0% and 17% by the reviewers, respectively. The PureEEG artifact removal algorithm effectively removes artifacts from EEGs and improves the readability of EEGs impaired by artifacts. Only in rare cases the algorithm attenuates EEG patterns slightly, but the clear visibility of significant patterns was preserved in all cases of this study.

F40
Timeliness of Initiation of Video-EEG Monitoring
Mark Callow, MD; Heather Hatton, R.EEGT; Meriem Bensalem-Owen, MD

Limitations of resources and hours of EEG laboratory operation can affect safety and quality of patient care. The purpose of this study was to assess timeliness of initiation of continuous video-EEG monitoring (cEEG) before and after extension of the EEG laboratory hours and acquisition of additional resources. All requests for cEEG of inpatients from October 2012 to February 2013 (phase one) and after EEG laboratory hour’s extension from September to October 2013 (phase two) were retrospectively identified. Patients for whom portable units and or EEG technicians’ availability to perform setup were not readily available at the time of the service request were identified. Time of initiation of cEEG and EEG findings were assessed. In the first phase of this study, 44 patients were identified. Among the patients for whom cEEG was eventually initiated, 33.3% had either electrographic seizures or epileptiform activity during monitoring. The average delay in initiation of cEEG was 14.9 hours. After extension of the laboratory hours and with acquisition of additional resources only 8 patients were identified. For this second phase the average delay of initiation of cEEG was 6.5 hours.

This study demonstrated that additional resources and extension of EEG laboratory hours in our institution shortened the time of initiation of cEEG by half.

F41
Seizures Without Awareness: A Pure Cohort
Michael Langston, BS; Kirsten Velvington, R.EEGT, CLTM; Jerry Shih, MD; William Tatum, DO

Objective: To profile patients with seizures without awareness (SWA).

Methods: 24 patients suspected of SWA were identified. Patients were case-matched by gender and age with seizure awareness (SA). A group always aware and one never aware of seizures was confirmed by video-EEG monitoring (VEM). Between group differences were addressed by Chi-square and Fischer’s Exact Test. Group significance was measured using the student t-test (p= <0.05)

Results: 11 patients with SWA and 11 case-matched SA were analyzed. SWA age 53 years v 33 years with SA (p=0.04). Six (55%) SA presented for new diagnosis v 3/11 (27%) with SA (X2=1.8, p=0.17). SA were on fewer AEDs (SA: 1.3; SD=0.8 v SA: 2.4; SD=1.1 (p=0.02). SA were more likely to report seizure freedom at follow up (X2=1.43, p=0.04) despite longer intervals (SA: 6.5; SD=5.1 vs SA: 2.8; SD=2.6) (p=0.049).

Conclusions: SWA is an at-risk subtype of TLE. Advancing age, presenting for diagnosis, subtle semiology, and left temporal ictal EEG are clues. VEM is essential for diagnosis.

F42
Electro-Clinical Evolution in West Nile Meningoencephalitis
Ning Zhong, MD, PhD; Dawn Eliasish, MD; Marc Nuewe, MD, PhD

West Nile meningoencephalitis represents a small fraction of cases of West Nile Virus infection. Organ transplantation is associated with increased risk of acquiring such invasive disorder. We report a 66-year-old woman patient with a history of liver and renal transplant who presented with persistent altered mental status when suffered with urosepsis and acute respiratory failure. Clinically she was noted to an episode of eye fluttering and jerky eye movements. The EEGs showed a non-specific moderate to severe diffuse slowing of the background rhythm. A week later, the EEGs evolved into persistent runs of bilaterally synchronous periodic epileptiform discharges (BiPLEDs) for over 24 hours, which was suppressed by benzodiazepine. Brain MRI initially showed hyperintense signal within the medial bilateral thalami and substantia nigra with contrast enhancement. Then it evolved to the tectum, and the middle cerebellar peduncles with new restrict diffusion when the patient EEGs showed BiPLEDs. The patient was aggressively treated with anti-epileptics in concerning for non-convulsive status. Two weeks later, the patient EEG returned to diffuse slowing and MRI showed prominent decreasing hyperintense signal. The EEG is helpful in the evaluation of patients with altered mental status when clinical exam is limited and can provide direction of management.

F43
EEG Features in Nocturnal Frontal Lobe Epilepsy (NFLE)
Trisham Gyang, MD; Robert Beach, MD PhD; Yaman Eksioglu, MD, PhD

Introduction: NFLE is a group of paroxysmal sleep related disturbance characterized by hypermotor behaviors arising during non-REM sleep. These spells are difficult to distinguish from non-epileptic sleep disorders due to limited and variable EEG findings.

Methods: Patients observed on video EEG monitoring.

Cases: Fourteen year-old female with presumed primary generalized seizures develops nocturnal episodes of sudden waking, crying and hypermotor activity. EEG revealed bifrontal predominant electrodecrement, and sharply contoured, rhythmic beta activity associated with these episodes.
Twelve year-old boy with history of developmental delay develops nocturnal episodes of sudden awakening, crying and violent behavior. EEG revealed bifrontal predominant electrodecrement with rhythmic sharply contoured beta activity, associated with these episodes. (Image3)

Thirty-nine year old male with history of traumatic brain injury and complex partial epilepsy develops nocturnal spells of sudden awakening and violent behavior. EEG revealed rhythmic bifrontal theta evolving into sharp and slow waves. (Image2)

Conclusion: Although clear electrophysiological characteristics of ictal events in NFLE are often lacking, electrographic findings are variable and can include diffuse relative voltage attenuation/electrodecrement with bifrontal rhythmic, sharply contoured beta following k-complex; and frontal rhythmic slowing with sharp and slow waves. These are associated with episodes of sudden arousal and hypermotor movements.

F44
Ictal Vital Signs in the EMU
William Tatum, DO; Emily Acton, BS; Michael Langton, BS

Objectives: Analyze ictal vital signs (i-VS) in epileptic seizures (ES) and non-epileptic seizures (NES).

Methods: 119/183 patients had video-EEG (vEEG) in 4/2010-4/2011. VS included heart rate (HR), oxygen saturation (O2), and systolic (S)/diastolic (D) blood pressure (BP). Peak i-VS during ES and NES were compared. A secondary analysis was based on motor semiology. Student T-test, Fischer’s Test and McNemar’s Test and linear regression correlated parameters.

Results: 53 ES and 66 NES had VS and iVS. 23/53 (43.40%) ES had generalized motor seizures (GMS) with iVS greatest for this group. An iHR of 148 bpm in ES and 111 bpm in NES was seen (p<0.001). GMS had an iHR > focal seizures with impaired consciousness (p=0.0001). Higher iBP-S was relative to impaired consciousness in ES. Across groups iO2 reduction was greater for ES than NES with motor symptoms (p=0.01). In ES iHR was inversely proportional to iO2 reduction (R²=0.3262, p=0.02). In NES iHR was directly proportional to iBP-S (R²=0.38, p=0.0001) but not iO2.

Conclusions: iHR and iBP elevations in NES underscore the need for safeguards during vEEG. iVS are proportional to motor involvement in ES and iHR elevation is inversely associated with iO2 reduction supporting respiratory-cardiac dysfunction that may underlie SUDEP.

Saturday, February 8, 2014
Display Time: 7:00 AM – 2:00 PM
Poster Tours: 12:30 – 2:00 PM
Location: 200 Peachtree, 7th floor
Categories: S2 – S9

S2
Outcomes of ICU Pts Correlated to CC Research Terminology
Carlos Muniz, MD; Andrea Synowiec, DO; Kevin Kelly, MD, PhD

Background: The American Clinical Neurophysiology Society published in 2012 recommendations for standardized electroencephalographic terminology for EEGs performed in ICU patients. To date, there has not been published data on the outcomes of ICU patients whose abnormal EEGs have been reviewed using this new proposed terminology. Furthermore, the clinical significance of certain EEG patterns found in ICU patients has not been established.

Aim: To classify abnormal EEG patterns seen in ICU patients using the new proposed ACNS terminology and correlate these patterns with patient outcomes.

Methods: We will perform a keyword search in our EEG record database to select ICU patients who have had epileptiform abnormalities reported on EEG. The EEG tracings will then be reviewed and described using the new proposed ACNS terminology. Statistical analysis will be performed to assess the relation of specific abnormal EEG patterns on 1) mortality; 2) discharge disposition; 3) response to antiepileptic therapy; 4) length of hospital admission.

Hypothesis: The new ACNS standardized ICU EEG terminology can provide reliable information on patient outcomes.

S3
BurSIn Quantifies Burst Suppression in Status Epilepticus
Christos Papadelis, PhD; Chiran Doshi, MSc; Sigride Thorne, MD; Robert Tasker, MD; Tobias Loddenkemper, MD

Status epilepticus (SE) is a neurological emergency that requires prompt diagnosis and treatment. In order to control SE, short acting sedative drugs are administrated but the optimal treatment regimen remains unclear. Continuous EEG monitoring can be used to facilitate the better control of seizures and the administration of the drug. Methods: An automated EEG-based algorithm that identifies the occurrence of bursts and quantifies the power and temporal features of burst suppression in SE was developed. The algorithm, namely BurSIn (Burst Suppression Index), combines information from the amplitude, frequency content, and entropy of the EEG signals (figure). Results: The algorithm’s performance was tested on burst suppression EEGs (mean duration: 100.7 min (interquartile range: 6.4–186.3 min)) from 11 pediatric SE patients (3-17 years) with generalized SE treated mainly with pentobarbital (0.08-7 mg/kg/h) and isoflurane (0.1-1.5%). Independent EEG review was used as the gold standard in
estimating the algorithm’s sensitivity that was found to be 99.8% in the identification of burst occurrence and 97% in the quantification of the burst intervals. Conclusion: The algorithm identifies reliably burst suppression patterns and clinically relevant characteristics. This offers an evidence-based platform for further development of real-time monitoring in pediatric ICU patients with SE.

S4 Continuous EEG for All Grades of SAH Patients Developing DCI
Gregory Kapinos, MD, MS; Colleen U. Janson, PA-C; Uziel Yagudayev, PA-C; Willie Walker Jr., CEEGT; Cynthia Harden, MD; Raj K. Narayan, MD, FACS

BACKGROUND/OBJECTIVE: For poor grade patients developing delayed cerebral ischemia (DCI) after subarachnoid hemorrhage (SAH), neurointensivists, short of a clinical exam, rely on continuous quantitative EEG (cqEEG) to detect ischemic secondary injury. In good grade too, cqEEG is valuable. Right hemispheric dysfunction is notoriously misdiagnosed and undetected clinically. Large infarctions in this region can easily be missed.

METHODS: In one patient, cqEEG was used because of poor grade. Induced fluctuations in blood pressure (BP) allowed us to find patient’s daily ischemic threshold (IT). In a second patient, thought to be only mildly impaired, cqEEG was used after right hemispheric infarctions developed.

RESULTS: The 2 cases are presented in table 1. Figure 1 represents the poor grade patient’s CT with right thalamic infarction. Figure 2 represents the same patient’s cqEEG day 10 snapshot. Figure 3 represents the CT of the clinically “intact” second patient.

CONCLUSIONS: Liberal use of cqEEG is valuable not only for early detection of ischemia in clinically silent patients in stupor/coma (poor grade), but also for 1) adjusting BP goal to the electrographic IT and 2) early detection of ischemia in alert patients without motor deficits (good grade) but with cognitive/behavioral changes due to right hemispheric “sub/pauci-clinical” ischemia.

S5 Increase 5KΩ Guideline of EEG Electrodes During cEEG?
John Ives, BSc; Paul Dionne, R.EEG T; Jeremy Eagles, R.EEG T; Steven Bild, R.EEG/EP T, CNIM, BS; Joshua Ehrenberg, BS, R EEG T, CNIM

The 5KΩ impedance guideline for EEG electrodes during cEEG has its technical origin from the 1960/70s. This dogma has propagated into every EEG guideline since, but without reference to the source. Because of technical advances of EEG amplifiers, digital/software acquisition, and after 40-50 years, it may be an time to review the 5KΩ rational. There are two effects of high electrode impedance; distortion due to EEG signal amplitude attenuation because of mismatch of the electrode/amplifier input impedances. Even with a conservative modern EEG amplifier input impedance of 10MΩ, a 1% distortion would only occur with an electrode impedance of 100KΩ. Amplifier common mode rejection ratio, input impedance and differential electrode impedance spread, determines amount of 60Hz artifact on the EEG recording. With cEEG, a higher electrode impedance guideline would reduce scalp trauma and infections of multi-day recordings on the scalp of ICU patients, as well as reducing patient setup and multi-day maintenance time. We suggest systematic studies of higher electrode impedance: i.e. 5KΩ-10KΩ-20KΩ with 2KΩ-5KΩ-10KΩ spread, on clinical studies to evaluate the presents of 60Hz artifact. This new data could then be used to justify existing guidelines or new guidelines associated with modern digital EEG systems.

S6 Identification of Improper Train of Four (TOF) Stimulation
Laxmi Dhakal, MD; William Freeman, MD

Introduction: Intensive Care unit (ICU) EEG monitoring is continued to grow but the resources needed to monitor such.

Methods/Results: Case Report. A 54 year old male with aneurysmal subarachnoid hemorrhage with refractory intracranial hypertension underwent continuous 21 channel EEG monitoring with automated quantitative EEG (QEEG) seizure detection. He was treated with mild hypothermia, and neuromuscular blockade (NMBS). On hospital day 5, an automated alert was sent to the neurointensivist. The EEG showed a high amplitude electrical burst in the frontotemporal head region first at 50Hz stimulation, followed by a frequency of 2Hz x 2 seconds. This pattern was recognized as a tetanic stimulation followed by the TOF stimulation pattern near the patient’s temple at the orbicularis oculi. The nurse moved the TOF stimulation from the hand to the orbicularis oculi location because the median and ulnar locations became ‘unresponsive.’ No electrographic seizures or other complications occurred.

Conclusion: Continuous EEG with QEEG software monitoring often sends various ICU EEG artifacts, which are unrelated to true electrographic seizures. However, on this occasion the computer generated alert allowed near real-time feedback to proper TOF site stimulation. QEEG software remains an evolving area of research, yet may provide benefits beyond simple seizure detection.

S7 NCs in Children with Prolonged Febrile Seizures
Masahiro Nishiyama, MD; Tsukasa Tanaka, MD; Kyoko Fujita, MD; Azusa Maruyama, MD; Hiroaki Nagase, MD, PhD

Objective: To clarify the prevalence of nonconvulsive seizures (NCSs) in children with prolonged febrile seizure (PFS), the time to record the first seizure on EEG, and the relationship between NCS and neurological outcome.

Method: We studied 68 children with PFS. The children underwent continuous EEG monitoring on admission to a tertiary pediatric care center at Kobe Children’s Hospital between February 2007 and September 2013. Children with prior neurological abnormalities were excluded. Clinical profiles and prognosis at the discharge were compared between the patients with NCS and those without NCS (non-NCS).

Results: Of the 68 children, NCS occurred in 17 children (25%). Neurological morbidity was higher in NCS patients (7/17, 41.2%) than in non-NCS patients (3/51, 5.9%; p<0.001).

Conclusion: The occurrence of NCS in children with PFS is associated with short-term neurological outcome.

S8 Status Epilepticus Possibly induced by Varenicline
Perumpillichira Joseph Cherian, MD, PhD; Jeffrey Britton, MD

Varenicline is a α4β2-nicotinic acetylcholine receptor partial agonist, widely used for smoking cessation. A single published case (2010) as well as a report of 15 patients by the Australian Bulletin of adverse drug reactions (2008) suggest
POSTER ABSTRACTS — CONTINUED

Saturday, February 8, 2014

varenicline may be rarely associated with seizures. We report a patient who developed status epilepticus after being on varenicline for a month and also show the use of quantitative EEG trends for seizure monitoring.

A 31-year-old lady, previously in good health, developed headaches, insomnia and 10 days later, had four episodes of generalized tonic-clonic seizures within 30 minutes. Head CT scan and venogram were normal. Blood lactate and CK levels were mildly elevated. CSF study excluded CNS infections. Continuous video EEG monitoring showed seven subclinical seizures within three hours of left fronto-temporal onset, lasting 50-116 seconds. Majority of seizures were seen on qEEG trends. Seizures abated on fosphenytoin. Investigations including an MRI brain, drug and toxin screens as well as paraneoplastic and autoimmune panels were negative. Varenicline was discontinued and levetracetam was started. She was discharged three days later, after 40 hours of seizure freedom. She has remained seizure-free.

S9

Relationship of Highly Epileptiform Bursts to Seizure
Stephen Thompson, MD; Stephen Hantus, MD

Rationale: The ACNS nomenclature of critical care EEG introduced the term Highly Epileptiform Bursts (HEBs). The predictive value of HEBs for seizure is unknown. Knowing their predictive value would inform the therapy of refractory status epilepticus.

Methods: Consecutive patients classified as having burst suppression over a period of 3-4 months were located in our database. Those of an anoxic etiology were excluded. Available EEG records were reviewed, both visually and quantitatively (Persyst), and burst suppression was dichotomized as HEBs or not. Times of transition out of burst suppression were identified, and whether burst suppression was followed by seizure or by a continuous slow (CS) EEG was determined.

Results: 24 adult patients meeting these criteria were identified, with some patients having multiple transitions out of burst suppression. HEBs were noted to be followed by seizure in 11 cases and by CS in 6 cases. Seizure did not follow burst suppression that was not of epileptiform morphology.

Conclusions: HEBs are associated with seizure. Whether or not HEBs can be used to predict the risk of seizure requires further prospective study.

S10

QEEG in Stroke Patients under rTMS Therapy
Geno Estrada, Dr.

Objective: A double-blind prospective study was carried out to assess the QEEG in a sample of 11 subjects with chronic stroke after the application of rTMS (1Hz).

Methods: The sample in study was randomly divided into two groups: 5 patients received sham rTMS and 4 patients received real rTMS both for 20 days. EEG was recorded before and after rTMS. The neuropsychophysiological measures used were the resting EEG power spectrum, Delta/Alpha ratio (DAR), the spike frequency-amplitude.

Results: 1 Hz rTMS caused a increase (p=0.06) in the Alpha and decrease Delta power spectra in both brain hemispheres. DAR diminished 23 % more in the 1 Hz rTMS group than in the sham rTMS group, and the spike-frequency also increased in 1 Hz rTMS group after stimulations.

Conclusions: Stroke patients who received 1 Hz rTMS sessions experienced modifications on qEEG, suggesting a propensity to the cortical activation in both brain hemispheres and the increment of cortical excitability. 1 Hz rTMS group had a better clinical recovery and of the brain electrical activity, reflected in the modifications of the Scandinavian Scale and DAR.

S11

Neurofeedback Protocol for Internet Addiction
Hyang Lim, M.A.; Hyerim Lee, M.A.; Jinkyung Oh, M.A.; Yunnha Kwan, M.A.; Sungwon Choi, PhD

The present study was a case study aimed to explore the effect of neurofeedback training (NFT) on frontal scalp area for reducing Internet overuse behavior.

One female undergraduate student who participated the study fulfilled inclusion criteria of above 80 at Young Diagnostic Questionnaire for Internet Addiction (YDQ) and not any history of brain damage. In the present study, the NFT protocol was administered to decrease the amplitude of theta wave band(3–7Hz) and maintain the amplitude of alpha wave band(8–12Hz) on Fz with seven 15-minute sessions of training. A self-administered daily time record of Internet use, Go/No-go Task and Stroop Task were used to evaluate the changes at pre and post-treatment.

During total training sessions, the amplitude of alpha wave band was maintained well. On the other hand, the amplitude of theta wave band showed observed high variance, but lowest amplitude of the wave band descended. Therefore, it is suggested that the participant’s control over theta wave activity was improved.

The finding of the present study raise the possibility that theta inhibition training on prefrontal cortex cause beneficial effect on Internet addiction patients.

S12

A Novel Seizure Pattern on Quantitative EEG (QEEG)
Ching Tsao, MD; Suzette LaRoche, MD; Shanaz Merchant, REEGT

The classic seizure pattern often seen on QEEG consists of an abrupt increase in power and amplitude of high frequency, rhythmic activity followed by a more gradual downsloping deflection as there is evolution to lower amplitude, slower frequencies and eventual resolution of the seizure.

We present a case of multiple, brief, focal seizures presenting with a unique QEEG seizure pattern. Simultaneous increase in amplitude and power at multiple frequencies resulted in several distinct horizontal bands with sudden offset. This pattern was best appreciated on compressed spectral array (CSA) and relative asymmetry measures. On raw EEG, the various frequency bands correlated to high frequency spikes followed by delta frequency slow waves over the left frontal region while the remainder of the left hemisphere contributed to the appearance of distinct alpha and theta frequency bands.

Further studies need to be conducted to appreciate the full spectrum of seizure presentations on various QEEG parameters, but we hope that the awareness and recognition of this atypical seizure pattern will increase sensitivity of the detection of seizures when utilizing QEEG.
S13
Postsurgical QEEG in refractory Temporal Lobe Epilepsy
Genco Estrada, Dr.

Purpose: To assess evolutionary changes in the QEEG in patients with refractory Temporal Lobe Epilepsy who underwent Epilepsy surgery.

Method: A prospective and longitudinal study was carried out to assess the QEEG before and after Epilepsy surgery in 13 patients with refractory TLE. Presurgical EEG recording was performed at 6, 12 and 24 months after surgery. QEEG measures evaluated were: Delta/Alpha Index (DAI), Brain Symmetry Index (BSI), Spectral Powers Index (SPI), total discharges frequency (TDF) and interictal epileptiform activity amplitude (IEA). These findings were correlated with clinical evolution using a seizures severity scale.

Results: We found a DAI decrease (p = 0.026), and BSI increase (p = 0.038). On the other hand a decrease of DTF and IEA (p = 0.024, p = 0.04) respectively were also found. These changes were gradually established until two years after surgery.

Conclusions: Refractory TLE patients undergoing epilepsy surgery, evidenced reorganization and cortical activation of both hemispheres, proven by the increased interhemispheric symmetry in QEEG and decreased IDA. The group of patients also showed a significant decrease in cortical excitability and epileptogenicity level, expressed by the DTF and IEA decreases at two years of evolution.

S14
Rapidly Evolving EEG in a Case of Autopsy Confirmed sCJD
Adam Juersivich, MD; Michel Berg, MD

Sporadic Creutzfeldt-Jakob Disease (sCJD) is a fatal prion disease, affecting 0.5 to 1.5 per 1,000,000/year. sCJD consists of a constellation of a rapidly progressive decline of neurological and psychiatric function, myoclonus, and various degrees of visual, cerebellar, and motor dysfunction. A diagnosis of probable sCJD may be made by a combination of clinical, EEG, MRI, and CSF findings. Characteristic EEG findings of periodic sharp wave complexes (PSWC) have a specificity for sCJD of 92%, which is higher than other non-invasive diagnostic tests. There is a high degree of variability in the sensitivity of EEG, with reports ranging from 44% to 94%. We present a case of an 85 year-old man with rapidly progressive dementia and myoclonus who died eight weeks after a precipitous decline in function. Autopsy specimens sent to the National Prion Disease Pathology Surveillance Center confirmed the presence of prion protein. Serial EEGs performed over the course of 11 days, beginning 5 weeks into the rapid phase of the illness, demonstrated progression from nonspecific slowing to clear PSWC. This case demonstrates the rapid progression of EEG changes in sCJD and helps to explain the wide variability in reported EEG sensitivity.

S15
Quality Assessment of EEG From “Dry Electrode” System
Jonathan Halford, MD; Gabriel Martz, MD; Ekrem Kutluay, MD; Chad Waters, MS; Brian Dean, PhD; Walid Soussou, PhD; Eric Duff, MBA

RATIONALE: A prototype dry electrode EEG system developed by Quantum Applied Science and Research (QUASAR), Inc. for Advanced Neumetrics, Inc. (ANI) utilizes a “dry electrode” system that allows recording of EEG data without use of collodion or gels between the electrode and the scalp.

METHODS: Twenty-one EEGs were acquired using both the standard scalp system (XLTEK) and the QUASAR system at the same encounter. Three ABCN-certified clinical neurophysiologists rated technical quality on a five-point scale for each EEG recording.

RESULTS: After the recording, 12 of 21 subjects rated QUASAR as “very comfortable”, 8 of 12 subjects “somewhat comfortable”, one subject as “mildly uncomfortable but OK”, and none rated it as painful. On a five point scale (1 — best quality, 5 — worst quality), average technical quality was 1.66 (SD 0.84) for standard EEG and 2.64 (SD 1.11) for QUASAR (paired t-test; p=0). The time to put on the QUASAR headset was much quicker (average 5.67 minutes) than standard EEG (average 21.1 minutes).

CONCLUSIONS: The QUASAR dry electrode EEG recording system offers quick and easy setup and is well tolerated. The technical quality of the recordings is less than standard EEG, although most recordings are interpretable.

S16
Effects of Tobacco Smoking on EEG
Lingling Rong, MD; Alfred Friontera, MD; Selim Benbadis, MD

Objective: To determine the effects of tobacco smoking on EEG in humans

Methods: The recordings from all adult patients who underwent routine EEG at the Tampa General Hospital (TGH) from January 1<sup>st</sup> to June 30<sup>th</sup> 2012 were analyzed for the effects of tobacco smoking on EEG.

Results: A total of 807 patients were included. The mean age was 58.401 (49.7%) were male. The numbers of smokers, non-smokers and patients of unknown smoking status were 263 (32.6%), 260 (32.2%) and 284 (35.1%) respectively. Smokers were male. The numbers of smokers, non-smokers and patients of unknown smoking status were 263 (32.6%), 260 (32.2%) and 284 (35.1%) respectively. Smokers were further divided into active smokers 134 (50.9%) and remote smokers 129 (49.1%). 165 smokers (62.8%) had abnormal EEG (generalized or focal slowing, spike or sharp waves, alpha coma, electrical silence). Among them, 14 (5.3%) showed epileptiform discharges (spikes or sharp waves). Of these 14, 1 patient (0.8%) was remote smoker, 13 (9.7%) were active smokers. 156 non-smokers (60%) had abnormal EEG. 20 of them (7.7%) had epileptiform discharges. In patients with an unknown smoking status, 175 (61.6%) had abnormal EEG and of them, 15 (5.3%) had epileptiform discharges.

Conclusions: The preliminary data showed that active smoking may increase, remote smoking may decrease the rate of EEG with epileptiform discharges. Further study with increased power is warranted.

S17
Parietal – Premotor Connectivity in Writer’s Cramp Patients
Nivethida Thirugnanasambandam, MBBS, PhD; Ajay Pillai, PhD; Jessica Shields, BS; Mark Hallett, MD

Patients with writer’s cramp (WC) demonstrate highly task-specific dystonic symptoms of the hand during free handwriting, while no symptoms are observed during other similar distal motor tasks. Recent studies postulate that impaired cortical connectivity could be partly responsible for WC symptoms and its task specificity. Functional MRI studies have shown that the left premotor and posterior parietal cortices are part of the task-specificity network and that the resting state connectivity between these 2 regions is decreased in WC patients. However, the connectivity between these regions has not been explored while the patients performed free handwriting.
In the current study, we aimed to determine the task-related coherence (TRCoH) between left premotor and posterior parietal cortices while the patients performed one of the following tasks: free handwriting or sharpening a pencil or imagining writing/sharpening. 32-channel EEG was recorded while the subjects performed the tasks.

Our preliminary results show that TRCoH in the β-frequency range between left premotor and posterior parietal electrodes is reduced in writer’s cramp patients when compared to healthy controls. This result is consistent with the idea that reduced connectivity contributes to the pathophysiology of WC. Future studies should explore if restoring β-coherence would improve behavioral symptoms.

**S18**

**Action Observation Training in Prosthesis Users**

**William Cusack, MS; Scott Thach, MS; Rebecca Patterson, MS; Robert Kistenberg, MPH, CP, LP, FAAOP; Lewis Whealton, PhD**

Our previous work demonstrated that the action encoding parietofrontal network, which is crucial for planning and executing motor tasks, is less active in prosthesis users who imitate movements of intact actors (mismatched limb) versus prosthesis users (matched limb). The current study investigates the cortical activity and motor behavior in prosthesis users trained with either matched limb imitation (MAT) or mismatched limb imitation (MIS). Intact subjects donned a “fictional amputee model system” (FAMS) to simulate the kinematic restrictions of a transradial amputation.

The hypothesis is that MAT would show greater engagement of the parietofrontal network and reduced movement variability compared to MIS. Training elapsed over three days and comprised of matched or mismatched video observations followed by action imitation. In order to track changes in cortical activity and movement variability, subjects performed cued movements while electroencephalography and electrogoniometry were collected. MAT showed greater engagement of the parietofrontal network and lower movement variability, while MIS showed greater engagement of the parietooccipital system. Results suggest that type of limb imitated plays an important role in the neurobehavioral process of learning to use a novel prosthesis and may have important implications on occupational therapy, which involves amputees imitating the mismatched limbs of intact therapists.

**S19**

**Adult Onset Startle Epilepsy Arising from Mesial Structures**

**Brian Moseley, MD; Cheolsu Shin, MD**

Introduction: Startle epilepsy is a form of reflex epilepsy, or condition in which seizures are induced by sensory stimuli. Startle epilepsy typically arise during childhood. The structures which generate startle-provoked seizures have not been fully elucidated. We present a case highlighting that startle epilepsy can be diagnosed in adults and may arise from mesial structures.

Methods: Case-report/literature review.

Results: A 45 year old gentleman presented with spells. For 8 years, the patient had experienced startle-induced spells. He would develop an epigastric rising sensation, lose awareness for seconds, develop tonic stiffening of his extremities, and fall. He had previously been diagnosed with paroxysmal kinesigenic dyskinesia and unsuccessfully treated with clonazepam, levetiracetam, and carbamazepine. The patient’s neurologic examination and MRI brain were normal. He was admitted for video EEG monitoring. During startle-induced events, his EEG revealed brief, fast beta activity in the midline central head region, consistent with seizures. Although lamotrigine improved his seizure control, he continued to have seizures a year later.

Conclusions: Startle epilepsy can be diagnosed at any age. The location of our patient’s ictal discharges further implicates mesial structures in the generation of startle-provoked seizures. Proper diagnosis is important, as its prognosis/treatment differs from other startle syndromes.

**S20**

**Diagnosis and Management of Status Epilepticus**

**Jessica Templer, MD; Thomas Bleck, MD; Bichun Ouyang, PhD; Adriana Bermeo-Ovalle, MD**

Rationale: Status epilepticus (SE) is a neurological emergency with major morbidity and mortality. This study describes the demographics, risk factors, and clinical characteristics of SE patients, their treatment, and their seizure recurrence risk.

Methods: We retrospectively reviewed records of SE patients >18 years old.

Results: We included 47 patients; 46% had a seizure history, and 48% were on antiseizure medications at SE onset. The majority had focal motor signs at presentation (43%), with behavioral changes in 32%, and bilateral motor phenomena in 17%. SE was suspected prior to continuous EEG recording in only 36%. It took an average of 3 days until SE was controlled (1-102 days). 11 (23%) had recurrent seizures after SE was controlled, 10 of whom had recurrent SE during the same hospital stay.

Conclusions: Almost one-half of patients with SE had a history of seizures and were on antiseizure medications at the time of SE onset. SE was suspected in a minority. Clinicians should consider SE in all patients with unexplained behavior changes, and be cognizant that SE recurrence is relatively frequent in the hospital setting.

**S21**

**Nystagmus as the Presenting Sign of Subtle Convulsive Status**

**Jigar Rathod, MD; Noemi Rincion-Flores, MD; Selim Benbadi, MD; Alfred Frontera, MD; Ali Bozorg, MD; Curtis Keller, MD**

Background: Up to 50% of patients with generalized convulsive seizures will have persistent electrographic seizures detected on EEG after overt convulsive activity has stopped. Epileptic nystagmus, a form of rhythmic eye oscillations, may be the only motor manifestation of ongoing electrographic seizure activity. Although it is a well recognized phenomenon, there are few cases mentioned in literature that have confirmatory video EEG.

Method: We present a case where nystagmus was the presenting sign of subtle convulsive status epilepticus confirmed by continuous EEG video monitoring.

Results: A 56 year old man with cirrhosis secondary to hepatitis C and alcohol use was admitted for liver transplant evaluation. He became confused and found to have elevated ammonia level. He later had a generalized tonic-clonic seizure which was treated with lorazepam leading to cessation of “clinical seizure activity”. Neurological evaluation revealed horizontal nystagmus. Head CT showed a calcified cystic lesion near the left posterior lateral ventricle. Due to continued nystagmus he was presumed to be in subtle convulsive status epilepticus which was confirmed on video EEG tracing.

Conclusion: Nystagmus can manifest as subtle convulsive status epilepticus. This case emphasizes the importance of video EEG monitoring to confirm the diagnosis and direct treatment.
S22
Paraneoplastic Epilepsy in Endometrial Adenocarcinoma
Noair Zaher, MD; Andrew Zillgitt, DO
71 y.o female was admitted for evaluation of right arm twitching. PMH is significant for hypertension and MVA in 1968. Clinically, she was noted to have multiple spells: right and left facial twitching, expressive aphasia, left eye twitching, left head deviation and stiffening of her arms. EEG showed on average eighteen seizures per day for a total of five days. Electrographically, there was prolonged focal seizures arising independently from the left posterior quadrant, right fronto-central region, and left fronto-central region. MRI brain W/O contrast did not show any significant findings. Extensive blood workup and CSF analysis including cytology was performed and was negative. Due to the lack of a clear etiology, screening for occult malignancy was performed. Mayo Clinic paraneoplastic panel was sent and later came back negative. CT abdomen revealed intraventricular mass extending from the fundus to the cervix. A biopsy was diagnostic for poorly differentiated endometrial adenocarcinoma grade III. Patient reached seizure freedom on Dilantin, Keppra,Topamax, and Vimpat but continued to be severely encephalopathic. Following a total abdominal hysterectomy, her mental status improved significantly and was discharged four days later.

S23
IGE vs. FLE: A Common Dilemma at Epilepsy Centers
Noemi Rincon Flores, MD; Ali Bozorg, MD; Alfred Frontera, MD; Selim Benbadis, MD; Fernando Vale, MD; Valerie Kelley, RN; Rathod Jigar, MD
Methods: We reviewed patients whose EEG-video monitoring and pre-surgical evaluation led to the question of Idiopathic Generalized Epilepsy (IGE) vs. Frontal Lobe Epilepsy (FLE).
Conclusions: These patients present with generalized tonic clonic seizures, early age of onset, and various amounts of asymmetries clinically or electrically, but in the absence of an MRI lesion, it is difficult to prove whether they have a focal onset that could be amenable to surgery.

S24
Ictal Tachyarrhythmia on a Patient Presenting with Syncope
Rafael Lopez-Baquero, MD; Indranil Sen-Gupta, MD; Adrianne Keener, MD; James Chen, MD PhD
Cardiac arrhythmias during epileptic seizures are uncommon; however the incidence of ictal arrhythmias appears to be more common in patients with intractable temporal lobe and generalized seizures. Information on isolated ictal arrhythmias is scarce. We report the case of a patient without previous history of seizures who was found with bicusynchronous temporal intermittent rhythmic delta activity and diffuse onset subcortical electrographic seizures, on initial evaluation for a syncope episode. EEG was ordered due to family history of epilepsy and febrile seizures history. Continuous EEG was remarkable for an ictal atrial tachyarrhythmia episode that was seen to stop synchronously with the electrographic seizure. After 3 months, patient has not had any syncope event since placed on Lacosamide, Keppra and Depakote. Patient under Cardiology follow up, pending pacemaker implantation versus cardiac ablation. Atrial tachyarrhythmias have been associated to autonomic dysfunction, here we discuss the potential factors influencing EEG as part of the initial workup of patients presenting with syncope and/or supraventricular arrhythmias.

S26
Allopregnanolone to Treat Refractory Status Epilepticus
Wendell Bobb, MD; Bradley Kolls, MD; Monika Ummat, MD; Aatif Husain, MD
Allopregnanolone is a neuroactive steroid metabolized from progesterone. Prolonged seizure activity results in internalization of the synaptic benzdiazepine-sensitive GABA<sub>A</sub> receptors. However, extrasynaptic receptors are not internalized and are therefore the primary means through which allopregnanolone is proposed to treat refractory status epilepticus. We report a case of a 28 year-old male with refractory status epilepticus. He was previously healthy and suddenly developed encephalopathy and meningismus and progressed to unresponsiveness within one week. Imaging remained normal throughout. He initially had infrequent, bihemispheric, multifocal seizures which became continuous with intermittent generalized periodic discharges. Over 25 days, numerous anticonvulsants and treatments were initiated including ketamine, propofol, midazolam, pentobarbital, immunoglobulins, ketogenic diet, and cooling. Although burst suppression was achieved on pentobarbital, the bursts consisted of high amplitude sharp activity, and three attempts to wean off pentobarbital were unsuccessful. Allopregnanolone was administered over five days. From about 60 hours after the initial infusion, the EEG showed progressively fewer sharps within the bursts and began to resemble normal waveform morphology. This continued until seven days after the allopregnanolone was stopped at which time his seizures recurred as primarily right temporal seizures. The infusion was well tolerated with no overt complications attributable to the infusion seen.

S27
Ictal and Postictal Asystole
Wendell Bobb, MD; Rodney Radtke, MD; Mariam Wasim, MD; Aatif Husain, MD
Ictal asystole is considered a rare phenomenon and has been implicated as a possible cause for SUDEP. It is usually seen in temporal lobe seizures and less commonly in focal extratemporal lobe seizures although most reports have not found a predominant lateralization of the focus. Sinus tachycardia is the most common arrhythmia observed with seizures, but severe bradycardias due to sinus node arrest generally precede ictal asystole. We describe four cases of ictal asystole ranging from five to 20 seconds in addition to a case of postictal asystole. Among the four ictal asystole cases, two originated from the left temporal lobe, one from the right temporal lobe and the other's origin unclear because of myogenic artifact. The case of postictal asystole occurred 46 seconds after a generalized-onset tonic-clonic seizure with a three-second followed by a five-second cardiac pause. Four of the patients with temporal lobe epilepsy received pacemakers. The patient with postictal asystole did not receive a pacemaker because the asystole appeared to occur during a postictal apnic episode and not immediately following her seizure. Further studies and guidelines are needed to better establish the need for pacemaker implantation in ictal versus postictal asystole.

S28
Evidence of Functional Connectivity Between Right and Left
Nuria Lacuey, MD; Jonathan Miller, MD; Hans Luders, MD PhD
Objectives: to investigate the functional connections between right and left mesial temporal structures. Previous studies of cortico-cortical evoked potentials (CCEPs) only reported positive results in 1 out of 28 patients.
PGES and Bradycardia after Seizure in Dravet Syndrome
Se Hee Kim, MD; Linda Laux, MD; Douglas Nordli, MD

Incidence of sudden unexpected death in epilepsy (SUDEP) is exceptionally high in Dravet syndrome (DS). Postictal generalized EEG suppression (PGES) followed by acute cardiorespiratory dysfunction always preceded arrests in recorded SUDEP cases. We sought to identify PGES and following bradycardia after convulsive seizures in 8 patients with DS (9.6 ± 0.9 years old), in comparison to 5 patients with myoclonic-ataxic epilepsy (MAE) (10 ± 2.1 years old).

Methods: The material consisted of 6 patients with drug resistant focal epilepsy explored with depth electrodes implanted in the mesial temporal structures on both sides. All patients also had CCEPs evoked by stimulation of the fornix and hippocampus as part of a research project evaluating fornix stimulation for control of hippocampal seizures.

Results: Stimulation of the fornix elicited responses in the ipsilateral hippocampus in all patients, while 2 out of 6 (33.3%) also had contralateral hippocampus response, without involvement of the contralateral temporal isocortex or amygdala.

Conclusion: This study confirms the existence of functional connectivity between bilateral mesial temporal structures. These connections explain the frequent spreading of mesial temporal epileptiform discharges to contralateral mesial temporal structures without prior involvement of neocortical temporal structures.

S29

Functional Connectivity Between Mesial Temporal Structures
Nuria Lacuey, MD; Bilal Zonyj, MD; Emiré Kahrmán, MD; Jonathan Miller, MD; Hans Luders, MD PhD

Objectives: to investigate the functional connections between right and left mesial temporal structures. Previous studies of cortico-cortical evoked potentials (CCEPs) only reported positive results in 1 out of 28 patients.

Methods: The material consisted of 6 patients with drug resistant focal epilepsy explored with depth electrodes implanted in the mesial temporal structures on both sides. All patients also had CCEPs evoked by stimulation of the fornix and hippocampus as part of a research project evaluating fornix stimulation for control of hippocampal seizures.

Results: Stimulation of the fornix elicited responses in the ipsilateral hippocampus in all patients, while 2 out of 6 (33.3%) also had contralateral hippocampus response, without involvement of the contralateral temporal isocortex or amygdala.

Conclusion: This study confirms the existence of functional connectivity between bilateral mesial temporal structures. These connections explain the frequent spreading of mesial temporal epileptiform discharges to contralateral mesial temporal structures without prior involvement of neocortical temporal structures.

F30

PGES and Bradycardia after Seizure in Dravet Syndrome
Se Hee Kim, MD; Linda Laux, MD; Douglas Nordli, MD

Incidence of sudden unexpected death in epilepsy (SUDEP) is exceptionally high in Dravet syndrome (DS). Postictal generalized EEG suppression (PGES) followed by acute cardiorespiratory dysfunction always preceded arrests in recorded SUDEP cases. We sought to identify PGES and following bradycardia after convulsive seizures in 8 patients with DS (9.6 ± 0.9 years old), in comparison to 5 patients with myoclonic-ataxic epilepsy (MAE) (10 ± 2.1 years old).

More patients with DS had convulsive seizures with both tonic and clonic components (p=0.01), lasting 1 – 3 minutes (p=0.05), compared to patients with MAE. PGES was found only in DS (3/8 vs. 0/5, p = 0.12). One patient with DS showed persistent bradycardia after a tonic-clonic seizure with prolonged PGES (43 seconds) (Fig.1c). Two patients with DS or MAE without PGES showed bradycardia briefly at the seizure end, but developed tachycardia immediately thereafter. Tachycardia persisted throughout the initial 2 – 4 min postictal period in the others (6/8 DS vs. 4/5 MAE, p = 0.84) (Fig.1c).

Persistent, postictal bradycardia happened rarely, possibly in association with PGES, and only in patients with DS. Larger studies are warranted to confirm these findings and to clarify the relationship between PGES and bradycardia.

S31

HFO Rate in Different Structures of the Brain
Vojtech Svehlik, MD; Cimbalko Jan, MS; Vlastimil Sulc, MD; Greg Worrell, MD; Benjamin Brinkman, PhD; Vincent Vasoli, MS

High frequency oscillations (HFOs) are potentially useful biomarkers of seizure onset zone (SOZ) and epileptogenic brain. The HFO characteristics in anatomical structures, however, are not known. The aim of this work was to assess the rate of HFOs in SOZ and non-SOZ in medial temporal lobe structures.

We analyzed 25 patients with drug-resistant focal epilepsy who were implanted with depth electrodes as part of their evaluation for epilepsy surgery. The electrodes that were localized within four distinct medial temporal lobe structures, were classified as within SOZ or non-SOZ using co-registered post-implant CT and pre-operative MR images.

We statistically evaluated HFO rate in different temporal lobe structures using ANOVA (p < 0.01). Subsequently, we performed Wilcoxon rank-sum analysis on all the pairs separately. We found a significant difference between SOZ and non-SOZ in all structures but hippocampus. There was no significant difference between individual SOZ structures. Notably, SOZ amygdala was not significantly different from nonSOZ hippocampus.

While HFOs appear to be a good biomarker of epileptogenic tissue, the presented result suggests that HFO rates depend on the structure which generates them and that the researchers should take this into account when localizing pathological brain based on HFO rates.

S32

Clinical Application of fMRI/EEG of Patients in the EMU
John Ives, BSc; Jean Gotman, Phd; Mukund Balasubramanian, Phd; Simon Tousseyn, Phd

It has been 20 years since the technique of fMRI/EEG was demonstrated followed by validation of the hypothesis correlating BOLD/fMRI with epileptic activity. Technical advancements permitting continuous fMRI/EEG acquisition with improved computer analysis have now been established and employed in multi-center studies. What we need for moving fMRI/EEG towards a routine clinical tool is an approach that permits studies of EMU patients to be obtained efficiently. Electrode-caps are not compatible with LTM while traditional EEG electrodes are not MRI compatible. An electrode system designed for LTM/fMRI/EEG was originally employed. The EEG electrodes are designed for LTM recording but are imaging compatible. They can be disconnected at the head-end because of simple, small mass-connectors. The patient can be taken to the fMRI/EEG unit, connected via the same electrode/connector system. At the end of the study the patient can return to the EMU, reconnect permitting continuation of LTM. An EEG technologist is not usually required as it is no longer necessary to remove LTM electrodes, install an EEG cap, remove cap and replace LTM electrodes. Besides efficiently making fMRI/EEG studies routinely possible on a wide-range of patients, there is reduced wear on patient’s scalp and less down-time of LTM.
**S33**

**RF Heating of Gold and Plastic EEG Electrodes during MRI**

Mukund Balasubramanian, PhD; William Wells, PhD; John Ives, BS; Patrick Britz, PhD; Tobias Loddinkemper, MD; Padmavathi Sundaram, PhD; Robert Mulkern, PhD; Darren Orbach, MD, PhD

The EEG electrodes used in clinical settings are often removed prior to MRI scanning at 3T due to safety/heating concerns. It would be advantageous to leave these electrodes on during routine clinical MRI scans and to be able to record from them during fMRI/EEG studies. Previous studies of the heating of EEG electrodes by radiofrequency (RF) pulses have either been at 1.5T or have employed EEG caps that are not typically used in clinical settings. We therefore measured the temperature changes under two types of clinical electrodes (gold cup and conductive plastic) during a variety of MRI scans at 3T, using watermelons as phantoms. For both electrode types, we found little heating for all scans when the wire lengths were multiples of 1/2 the RF wavelength and substantial heating for scans with high specific absorption rate (SAR) when the wire lengths were near 1/4 wavelength (~2ft at 3T), consistent with the idea that RF standing waves established on the wires are the main cause of heating. Our results suggest that these electrodes could be used safely in 3T MRI scanners, as long as the EEG wire length and the SAR of the MRI scans are both carefully taken into consideration.

**S34**

**IONMAssociated with Delayed Cerebral Ischemia and Infarct**

Forough Ghavami, DO; Viet Nguyen, MD; Scheherazade Le, MD; Leslie Lee, MD; S. Charles Cho, MD; Michael Marks, MD; Jaime López, MD

A 38 year old male with subarachnoid hemorrhage from a right posterior cerebral artery aneurysm, without deficits, underwent endovascular coiling with IONM. Electrocencephalography, brainstem auditory, motor, and somatosensory evoked potentials (SSEP) were all symmetric and reproducible at baseline. After detachment of 8 coils, a 75% amplitude reduction in the left upper and lower extremity SSEPs was noted. This reduction did not resolve by the end of the case, despite increases in the mean arterial pressure. All other modalities remained unchanged. Post-procedure, the patient had full sensation and strength in all extremities until post-operative day 3, when he developed acute left sided weakness and numbness after being transferred out of intensive care off phenylephrine. MRI brain showed a right thalamic infarct. Treatment of cerebral aneurysms carries the risk of intraoperative cerebral ischemia. In our experience, persistent IONM changes typically correlate with immediate post-operative new neurologic deficits. Our case suggests that the significant intraoperative decline in cortical SSEPs was indicative of cerebral ischemia that persisted post-operatively. The spinal cord can be tethered by the filum terminale. Often this is the result of excessive or additional fat within the filum which may cause it to adhere to the dura. Although less common than in humans canines also suffer from tethered spinal cords, with similar clinical symptomatology. We report what we believe to be the first occurrence of a canine tethered spinal cord release with the assistance of neuromonitoring.

**S35**

**Does IONM Cause Delays in Surgical Cases?**

Forough Ghavami, DO; Viet Nguyen, MD; Scheherazade Le, MD; Leslie Lee, MD; S. Charles Cho, MD; Richard Jaffe, MD, PhD; Gary Steinberg, MD, PhD; Jaime López, MD

Background: There is debate that the time needed to setup patients for IONM delays the start of surgery. To our knowledge, this has not been evaluated.

**S36**

**Intraoperative Monitoring of SSEP in Diabetes. A Pilot Study**

Ilrun Cho, MD; Young Jin Ko, MD, PhD

Introduction: Conduction abnormalities of somatosensory evoked potential in diabetes have been reported. The aim of this study is to investigate effect of diabetes on intraoperative monitoring of somatosensory evoked potential.

Methods: We retrospectively reviewed data of bilateral median nerve somatosensory evoked potential from 7 patients with diabetes more than 5 years (5-20years, average 10.5years) and 7 age- and sex-matched patients without diabetes. All patients underwent craniotomy and unruptured cerebral aneurysm clipping without proximal vessel clamping under total intravenous general anesthesia. There were no complications during and after the surgery. Patients with brain lesions which could affect somatosensory evoked potential and peripheral neuropathy due to other causes except diabetes were excluded. We compared cortical latency (N20) and central conduction time (N20-P25) between two groups during the surgery.

Results: Cortical latency (N20) was significantly delayed (P < 0.05) symmetrically in diabetic patients compared with non-diabetic patients. N20-P25 inter-peak latency representative of central conduction time was also increased (P < 0.05) in diabetic patients. During the operation, there were no significant changes of somatosensory evoked potential in both groups.

Conclusion: We conclude that central conduction abnormalities may be present in intraoperative monitoring of diabetic patients without definite central nervous system lesions.

**S37**

**Neuromonitoring of Canine Tethered Cord Surgery**

Jonathan Norton, PhD; Kathy Linn, DVM

The spinal cord can be tethered by the filum terminale. Often this is the result of excessive or additional fat within the filum which may cause it to adhere to the dura. Although less common than in humans canines also suffer from tethered spinal cords, with similar clinical symptomatology. We report what we believe to be the first occurrence of a canine tethered spinal cord release with the assistance of neuromonitoring.
Clinical history and neuroimaging (MRI) were both suggestive of a fatty filum tethering the spinal cord in a Boston Terrier. Under standard surgical conditions a L7 laminectomy was performed. We instrumented the anal sphincter as well as hindlimb muscles using a Neurolog system (Digitimer, UK) and custom-build display hardware and software (Norton). Stimulation of nerve roots and the filum was performed with a custom built probe connected to Digitimer DS7A stimulator. Stimulation parameters were 0.1-5mA for threshold stimulation of nerve roots. No response to stimulation of the filum at 10mA was observed.

This report demonstrates the feasibility of neuromonitoring of canine (and presumably other animal) surgical procedures. Within highly specialised centres this new field of IOM offers the potential to improve animal care.

**S38**

**Comparison of BIS and Entropy Interference During IOM**

Shaila Gowda, MD; David Betts, CNIM, R.EEG/EP T.; William Nantau, CNIM; Kaveh Aslani, MD; Roy Sato, MD

**Objective:** Intraoperative somatosensory evoked potential (SSEP) recordings are obtained in a hostile environment due to electrical noise and artifacts from other equipments. BIS and Entropy monitors (EM) are used to measure the depth of anesthesia in patients undergoing surgeries. Aim of this study was to compare the interference of BIS and EM during surgeries with simultaneous SSEP recordings.

**Method:** Sixteen patients underwent spine surgery; 8 had BIS and 8 had EM with simultaneous SSEP recording. Comparison of degree and improvement in artifacts was tested for both monitors by: 1. Increasing the separation between devices 2. Routing FPz only electrode 3. Routing all electrode opposite to BIS connection and 4. Bipolar recording modification - moving FPz electrode farther away from BIS or EM.

**Result:** SSEP traces of all patients with EM were artifact free requiring no intervention. All patients with BIS monitors showed noise in SSEP recording. Channels most affected included FPz electrode. In 2 patients SSEP recording significantly improved after FPz electrode was moved 2-3 cm posteriorly away from the BIS montage. Other interventions did not help.

**Conclusion:** In our experience EM had no interference and appears to be an optimal choice compared to BIS for intraoperative SSEP recording.

**S39**

**EEG Monitoring During Cerebrovascular Surgery**

William Nowack, MD

During intraoperative monitoring (IOM) of cerebrovascular surgeries EEG is monitored. EEG can be assessed visually or digitally.

Patients: During seven cerebrovascular IOM procedures (three cerebral aneurysm clippings, two carotid endarterectomies, one arteriovenous malformation repair and one revascularization in a patient with sickle cell) EEG was evaluated in real time on a Natus or Cadwell machine, using simultaneous visual and digital (density spectral array, compressed spectral array and spectral edge) analyses.

Results: In five cases changes were detected on digital but not visual analysis and in two on visual but not digital analysis. The changes were reversed by mild blood pressure elevation or by repositioning temporary aneurysm clips.

**S40**

**Jeavons Syndrome: Seizing the Light. A Case Series**

Ashok Yadav, MD; Prashant Rai, MD; Ingrid Tuxhorn, MD

**RATIONALE:** JS is characterized by eyelid myoclonias +/- absences and photosensitivity. A few series have been published on JS but it is as yet not featured in the epilepsy Classification of ILAE.

**METHODS:** We analyzed 5 children with JS over a period of 3 years at RB&C Hospital, with EM, and photosensitivity.

**RESULTS:** All girls, ages of onset from 5-14 years. Daily EM, obsessive induction of seizures - waving a hand in front of one eye (with secondary eyebrow alopecia), seeking out lights, looking into the glare of snow or sunshine on water, were seen. Ictal EEG - spontaneous, forced eyelid closure induced generalized spikes and PPR, Intermittently normal and reactive PDR & generalized EDs. One had focal interictal discharges over the occipital regions bilaterally. EM and/or paroxysmal EDs were induced by photic stimulation. Diagnosis delayed - up to 8 yrs and earlier diagnosed as absence(generalized). Misdiagnosed as Facial tics, Syncope and OCD.

**SIGNIFICANCE:** JS may be more frequent than assumed and also under and misdiagnosed. Comorbidities & Photostimulation suggests dysfunction in the midbrain visual system and connected occipital and frontal networks. Further studies are needed to better delineate it.

**S41**

**Ictal Cardiac Asystole Due to a Hippocampal Seizure**

Edilberto Amorim, MD; Steven Factor, MD; Alexandra Popescu, MD; Gena Ghearing, MD

Cardiac arrhythmias are a well-known complication of seizures and might be associated with sudden death in epilepsy patients. Ictal- asystole is a rare complication of epilepsy, and its management remains uncertain. A 47-year-old man with refractory epilepsy was admitted to the epilepsy-monitoring unit with a history of seizure-related falls and pericardial cyanosis for several years. Following a focal seizure that evolved to a bilateral convulsive seizure, an ictal cardiac asystole of 34 seconds duration was observed. Scalp EEG monitoring revealed a right-frontal seizure onset. A brain MRI showed an area of heterotopic gray matter adjacent to the right lateral ventricle and involving the mesial temporal lobe. Further monitoring after placement of subdural and depth electrodes revealed consistent right anterior hippocampal ictal onset with initial spread to the inferior right frontal lobe. Depth electrodes placed in the occipital lobe section of the heterotopic gray matter lesion were not activated during the seizures. A right anterior temporal lobectomy was performed, and the patient has been seizure free on a similar medication regimen. Pacemaker implantation is often recommended in patients presenting with ictal-asystole, however its clinical efficacy on preventing sudden unexplained death in epilepsy remains uncertain in surgically treated patient.
S42
MAE Gender Preference and Mean Age of Onset: Literature Review
Jenny Guerre, MD
Myoclonic absence seizure is a rare type of generalize seizure with variable prognosis. It represents 0.5 to 1% of the cases of epilepsy, the association of characteristic myoclonic jerks with axial hypertonia, brief loss of awareness with 3Hz generalize spike and wave during ictal EEG are the corner stone of the diagnosis. Because of its rarity not many cases have been reported. We did a review of the medical english literature from 1950 to 2013 in pubmed to assess the gender preference and mean age of onset of myoclonic absence seizure.

Results: 230 articles were retrieved in Pubmed using key words: epilepsy,myoclonic absence,cases,humans. After review of their abstracts we selected for complete review the articles which contained any reference to clinical case(s) of myoclonic absence epilepsy. 11 articles met the criteria. They described a total of 109 cases of confirmed myoclonic absence epilepsy. The analysis of their data showed a male predominance (62%) and a mean age of onset of 4 years old.

Conclusion: Myoclonic absence epilepsy mean age of onset is likely lower than previously estimate with an age of onset ranging from 3 months old to 12 years and 5 months old. There is a male predominance (62%).

S43
Frontal Lobe Epilepsy Mimicking Myoclonic Absence Seizure
Jenny Guerre, MD
Myoclonic absence seizure is a rare type of generalize seizure. Generalize symmetric synchronous 3Hz SW in strict relation with characteristic myoclonic jerks with axial hypertonia and brief loss of awareness are the corner stone of the diagnosis. We present the case of a 6 years old girl who presented the clinical characteristics of MAE. Her video EEG showed frequent MJ, brief loss of awareness synchronous with 3Hz generalize spike and wave. Late in the recording she was found to have left frontal lateralisation of the epileptic discharge with rapid spread to the contralateral side. We believe that the generalize discharge was related to bisynchrony phenomenon. We did a review of the english literature from 1950 to 2013 in pubmed to determine how frequently MAE semiology associated with 2.5-3.5 Hz generalized spike and wave discharge have been reported in childhood onset frontal lobe epilepsy.

Preliminary Results: 568 articles were retrieved using key words: epilepsy, frontal, lobe, cases, humans. 125 articles reviewed. 12 articles described clinical semiology and ictal EEG of 175 patients with childhood onset FLE. 2 had myoclonic jerks with unilateral frontal SW discharge, one had MJ with reported generalize SW discharge.

Conclusion: Myoclonic jerks is a rare semiology in published FLE. Generalized SW discharge on ictal EEG are a rare findings in published FLE. This is the first reported case of MAE mimicking FLE.

S44
Epileptic Aphasia Managed by Continuous Video EEG
Jeremy Cholfin, MD, PhD
Epileptic aphasia is a rare clinical presentation. We describe here a 61 year-old right handed man with a history of left parotid gland tumor and metastasis to the left parietal lobe, status-post resection and focal radiation, presenting with acute onset of global aphasia. Continuous video EEG revealed frequent electrographic left temporal-parietal seizures superimposed on a background of periodic lateralized epileptiform discharges. Administration of IV lorazepam abolished the seizures and the patient converted from a mixed aphasia to a predominantly Wernicke’s type. Uptitration of levetiracetam and addition of lacosamide resulted in further improvement on the EEG, and despite continued periodic lateralized discharges, there was significant clinical improvement. By the time of discharge, the patient’s aphasia had largely resolved. To our knowledge, this is the first reported case of epileptic aphasia monitored and managed using continuous EEG monitoring.
EXHIBIT HALL & EVENING PROGRAMS

Exhibit Hall Hours
Friday, February 7, 2014 7:00 AM – 4:00 PM
Friday, February 7, 2014 6:45 – 8:00 PM (Welcome Reception)
Saturday, February 8, 2014 7:00 AM – 2:00 PM

EXHIBIT HALL FLOORPLAN

EXHIBITORS

<table>
<thead>
<tr>
<th>Exhibitor</th>
<th>Booth #</th>
</tr>
</thead>
<tbody>
<tr>
<td>ABCN/ABRET</td>
<td>Table A</td>
</tr>
<tr>
<td>ACMEGS</td>
<td>Table B</td>
</tr>
<tr>
<td>Alpha Omega Co. USA</td>
<td>24</td>
</tr>
<tr>
<td>ASET</td>
<td>Table C</td>
</tr>
<tr>
<td>Austrian Institute of Technology</td>
<td>6</td>
</tr>
<tr>
<td>Cadwell Laboratories, Inc.</td>
<td>5/8</td>
</tr>
<tr>
<td>Compumedics USA, Inc.</td>
<td>21</td>
</tr>
<tr>
<td>Consolidated Neuro Supply, Inc.</td>
<td>4</td>
</tr>
<tr>
<td>Demos Medical Publishing, LLC</td>
<td>20</td>
</tr>
<tr>
<td>DigiTrace EEG Services</td>
<td>23</td>
</tr>
<tr>
<td>Electrical Geodesics, Inc.</td>
<td>12</td>
</tr>
<tr>
<td>Elekta</td>
<td>22</td>
</tr>
<tr>
<td>Ives EEG Solutions</td>
<td>14</td>
</tr>
<tr>
<td>Moberg</td>
<td>2/3</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Exhibitor</th>
<th>Booth #</th>
</tr>
</thead>
<tbody>
<tr>
<td>mytuMed, Inc.</td>
<td>1</td>
</tr>
<tr>
<td>Nihon Kohden America</td>
<td>15</td>
</tr>
<tr>
<td>Persyst Development Corporation</td>
<td>18</td>
</tr>
<tr>
<td>PMT Corporation</td>
<td>13</td>
</tr>
<tr>
<td>Rhythmlink International, LLC</td>
<td>9</td>
</tr>
<tr>
<td>Ripple, LLC</td>
<td>10</td>
</tr>
<tr>
<td>Rochester Electro-Medical, Inc.</td>
<td>19</td>
</tr>
<tr>
<td>RSC Diagnostic Services</td>
<td>16</td>
</tr>
<tr>
<td>Signal Gear, LLC</td>
<td>11</td>
</tr>
<tr>
<td>UCB</td>
<td>17</td>
</tr>
<tr>
<td>Visualase, Inc.</td>
<td>7</td>
</tr>
<tr>
<td>Wolters Kluwer Health</td>
<td>26</td>
</tr>
<tr>
<td>WR Medical Electronics Co.</td>
<td>25</td>
</tr>
</tbody>
</table>
American Board of Clinical Neurophysiology, Inc. (ABCN)
2509 W. Iles Ave, Suite 102
Springfield, IL 62704
Phone: 217.726.7980
Fax: 217.726.7989
www.abcn.org

The American Board of Registration of EEG and EP Technologists (ABRET, Inc.)
2509 W. Iles Ave, Suite 102
Springfield, IL 62704
Phone: 217.726.7980
Fax: 217.726.7989
www.abret.org

American Clinical MEG Society (ACMEGS)
555 E. Wells Street, Suite 1100
Milwaukee, WI 53202
Phone: 414.918.9804
Fax: 414.276.3349
www.acmegs.org

American Clinical MEG Society is a non-profit trade association that includes the membership of clinical magnetoencephalography (MEG) facilities in the United States. Founded in 2006 by physicians committed to setting a national standard for high quality care of patients with epilepsy, ACMEGS now advocates for all individuals with neurological conditions who would benefit from MEG by educating policymakers and regulators about current and recommended standards of care, financial reimbursement, and health care provider regulations.

ASET - The Neurodiagnostic Society
402 E Bannister Rd, Ste A
Kansas City, MO 64131-3019
Phone: 816.931.1120
Fax: 816.931.1145

ASET - The Neurodiagnostic Society is the largest national professional association serving neurodiagnostic practitioners. ASET provides educational resources in all neurodiagnostic modalities, sets standards and competencies in neurodiagnostic technology and provides governmental advocacy to preserve the practice of neurodiagnostics. ASET’s membership represents over 4,000 neurodiagnostic professionals including technologists, students, physicians and institutions. The mission of ASET is to provide education and advocacy, creating greater awareness of the profession, and establish best practices to ensure quality patient care.

ASET provides its members practical guidance and helps them stay abreast the latest advances in the field through education programs, publications, and its member network. Resources and additional information are available at www.aset.org.

Alpha Omega Co. USA
5755 North Point Parkway
Suite 229
Alpharetta, GA 30022
Phone: 770.521.2049
Fax: 877.471.2055
www.alphaomega-eng.com/

Alpha Omega has been known in the neuroscience industry for two decades, offering clients breakthrough technology, unrivaled dependability, and dedicated service. A global leader in neural acquisition and analysis technology, Alpha Omega provides the latest Microelectrode Recording technology, which incorporates innovative software, hardware, mechanical designs, electrodes, and arrays. Alpha Omega’s competitive edge is in its custom design, manufacturing, and internationally marketing of research products that provide customizable solutions for neuroscientists and neurosurgeons. The company’s products can be found in advanced research institutions, hospitals, and universities across the globe. Alpha Omega maintains offices in Israel and Germany, and U.S. offices based in Atlanta, GA.

Austrian Institute of Technology
Donau City Strasse 1
Vienna 1220
Austria
Phone: +43.664.8251449
Fax: +43.505.5504125

The Austrian Institute of Technology provides easy-to-use EEG solutions for various areas of electrophysiology, including long term EEG monitoring for epilepsy and critical care. A new important highlight of our portfolio is NeuroTrend, the first EEG bed-side monitor for the ICU that implements the new ACNS standardized critical care EEG terminology. It enables in one glimpse reading off trends in the functional state of the patient’s brain. Further solutions are the epileptic seizure detection EpiScan with the verifiable highest reliability available on the market or the artifact removal PureEEG discovering the EEG even in heavy distorted brain signals.

Cadwell Laboratories, Inc.
909 N. Kellogg Street
Kennewick, WA 99336
Phone: 509.735.6481
Fax: 509.783.6503

Cadwell has developed useful and innovative devices for clinical neurophysiology. Neurologists, neurophysiologists and technologists who want reliable devices for research, academia and clinical uses have come to rely on Cadwell since 1979. Products include the Cascade® for IONM, Easy® for EEG/ICU/LTM and PSG and HST and the Sierra® range for EM/EP and NCV.
Booth # 21
Compumedics USA, Inc.
6605 W. WT Harris Blvd.
Charlotte, NC 28269
Phone: 704.714.3200
Fax: 704.714.3298

Compumedics Neuroscan is world-leader in the development of hardware and software for measuring and integrating all forms of brain activity. The systems developed by Compumedics Neuroscan have applications in all aspects of cognitive neuroscience and in medical diagnostics focused on sleep and neurology. The Company’s premiere product, the Curry NeuroImaging Suite, can integrate and co-register data from all neuroimaging modalities including EEG, MEG, MRI, fMRI, PET, SPECT, CT, DTI, and ECOG. Compumedics Neuroscan also has hardware and software solutions for the simultaneous acquisition of EEG and fMRI, now offering a simple solution with a broad EEG frequency band, with high quality data.

Booth # 4
Consolidated Neuro Supply, Inc.
6682 Waters Edge
Loveland, OH 45140
Phone: 513.722.2999
Fax: 866.270.4065

Consolidated Neuro Supply, Inc. is a leading provider of healthcare products used to diagnose neurological disorders and monitor critical neurological structures during high-risk surgical procedures. With nearly $5 million in annual sales, product offerings include EMG needle electrodes, subdermal needle electrodes, and pedicle screw and direct nerve stimulators. Located in Loveland, OH, the company has been in business since June 2003.

Booth # 20
Demos Medical Publishing, LLC
11 W. 42nd Street
New York, NY 10036
Phone: 212.804.6316
Fax: 212.941.7842
www.demosmedpub.com

Demos Medical is a publishing leader in clinical neurology and related disciplines. Visit us at ACNS 2014 to preview our list of premier print titles and exciting new digital products and apps. www.demosmedpub.com

Booth # 23
Digitrace EEG Services
200 Corporate Place, Suite 5
Peabody, MA 01960
Phone: 978.536.6101
Fax: 978.536.6401

Digitrace EEG products and services are used by dozens of comprehensive epilepsy centers throughout the US. In addition, there are more than 40 locations around the country where physicians can refer their patients for ambulatory testing. We are noted for unique capabilities, including our lightweight head-mounted preamplifier that minimizes motion artifact, on-line spike and seizure detection, multiple day monitoring capabilities and high-resolution synchronized video.

Booth # 12
Electrical Geodesics, Inc.
1600 Millrace Drive
Suite 200
Eugene, OR 97403
Phone: 541.687.7962
Fax: 541.687.7963
www.egi.com

EGI brings next generation clinical EEG systems, tools, and workflows to hospitals and clinics worldwide. EGI's EEG Systems feature EGI's revolutionary Geodesic Sensor Net for unprecedented patient comfort and rapid application, removal, and clean-up. Your choice of systems with 32 channels for routine 10-20 EEG in ambulatory or stationary mode, or dense array systems with 64, 128, or 256 channels for source estimation and other applications requiring high spatial resolution. EGI also offers software for spike and seizure detection and for source estimation. Visit EGI's booth to see this in action! www.EGI.com.

Booth # 22
Elekta
50-400 Perimeter Center Terr.
Atlanta, GA 30346
Phone: 770.300.9725
info.america@elekta.com

The Elekta Neuromag® TRIUX is the most advanced magnetoencephalography (MEG) system available. It takes functional brain mapping to the next level. MEG is rapidly becoming an indispensable brain imaging technology. Using sophisticated instrumentation, MEG detects the weak magnetic activity emanating from groups of neurons in the brain. Only MEG can precisely localize and record these millisecond phenomena that produce signals approximately a billion times smaller than the earth’s magnetic field. The TRIUX is a vital tool for improving patient management in the evaluation of epilepsy as well as pre-surgical mapping of motor cortex, visual, auditory, somatosensory and language functional areas.
Never have to remove EEG Electrodes again for imaging purposes. The Ives Imaging Friendly™ Electrode System provides a comprehensive selection of three separate imaging friendly EEG electrode types to record the patient’s EEG in the EMU or cEEG in the ICU.

The Moberg Component Neuromonitoring System (CNS Monitor) is the only EEG system specifically designed for the needs of the ICU. Only the CNS Monitor collects data from over 25 other monitoring and therapeutic devices and accurately synchronizes it in real time with quantitative EEG trends. The CNS Monitor is a full-function video/EEG monitor with remote review of EEG and other physiology using Persyst software. The future of integrated ICU neurophysiological monitoring is available today with the CNS Monitor.

We founded mytaMed, inc. in 2006 to provide great products to the IONM community at great prices. Providing off the charts value is our number one goal each and every day, and we live by the following formula: Value = Quality / Price. To increase value one of the following has to happen: quality increases or price decreases (preferably both). All of our neuro stimulation probes are very well made right here in the USA. We also run a lean operation, so you won’t find any mahogany desks or big expense accounts at mytaMed. Please stop by our booth. If you miss us in Atlanta, feel free to call us at 877.444.6982, email us at info@mytamed.com or visit our online store at www.neurostimprobes.com.


For more than twenty years Persyst has produced the most trusted and innovative EEG analysis products. Today, Persyst is the world’s leading supplier of seizure detection, spike detection, and quantitative analysis software to every leading EEG manufacturer for continuous EEG monitoring, LTM and ambulatory EEG.

PMT® Corporation is the premier supplier of an extensive line of neurosurgical products. PMT® offers Cortac® cortical surface electrodes and Depthalon® depth electrodes for epilepsy monitoring and microsurgical instruments. PMT® also offers high-end neck braces, including halo systems and orthotic vests for cervical, thoracic and lumbar spinal immobilization. Our product quality and dependable customer service makes us an industry leader. With a large sales force spread throughout the U.S. and distributors around the world, we can be on-site to work with you to define products to match your specific requirements.
**Booth # 9**  
**Rhythmlink International, LLC**  
1140 First Street South  
Columbia, SC 29209  
Phone: 803.252.1222  
Fax: 803.252.1111

Rhythmlink International, LLC designs, manufactures and distributes neurodiagnostic, intraoperative monitoring, critical care and polysomnography devices. Rhythmlink is the first to receive FDA clearance of Disposable MR Conditional Electrodes. Visit our booth to learn about the Disposable MR Conditional Cup Electrodes along with the innovative Disposable PressOn™ Electrodes and soon to market Disposable MR Conditional PressOn Electrodes. Recognized as a leader within its field, Rhythmlink provides the important physical connection between patients and the diagnostic equipment.

**Booth # 10**  
**Ripple, LLC**  
2015 South 1100 East  
Salt Lake City, UT 84106  
Phone: 801.413.0139  
Fax: 801.413.2974  
www.rpl.com

Ripple provides high performance neurophysiology data acquisition systems for recording and stimulation. Our systems are compact, portable, and heavily optimized for real-time, closed-loop control applications with up to 512 channels of EMG, EEG / ECoG and microelectrode data. Our software is cross platform, and can be run on Windows, Mac OS X, and Linux.

**Booth # 19**  
**Rochester Electro-Medical, Inc.**  
4212 Cypress Gulch Drive  
Lutz, FL 33559  
Phone: 800.328.5544  
Fax: 800.545.0845  
www.rochestermed.com

For over fifty years, Rochester Electro-Medical has been your best source for affordable, high-quality Neurodiagnostic Supplies and Customer Service with a personal touch. Featured products include Surgical Probes, fully customizable lines of Horizon™ Single, Paired, Twisted and RIBBON Subdermal Needle Electrodes, and GoldSelect™ Disposable Electrodes that offer unbeatable value and performance. Contact us for information on our full range of Disposables, Electrodes, Needles, Sensors and Accessories.

**Booth # 16**  
**RSC Diagnostic Services**  
101 E. Park Blvd.  
Suite 910  
Plano, TX 75074  
Phone: 877.333.2575  
Fax: 800.840.8626

RSC Diagnostic Services is a leading provider of EEG services, specializing in video ambulatory EEG, hospital ambulatory EEG partnership programs and continuous EEG monitoring in the ICU. The leadership of RSC Diagnostics Services has over 30 years of experience, delivering the highest level of quality and professional customer service in neurodiagnostics. The foundation for our success has been our commitment to develop dedicated teams whom deliver our promise to provide unparalleled customer service focused on our patients, our physician’s practices, and our hospital partners.

**Booth # 11**  
**Signal Gear, LLC**  
27 Sweetwater Drive  
Prosperity, SC 29127  
Phone: 630.272.5812

SIGNAL GEAR® is a medical device company focused on designing and developing neurodiagnostic accessories. We currently offer products for IOM and EMG/NCS settings, but are introducing many new products soon. Founded on the premise that passionate, thoughtful study is key to innovation and creativity, we study the scientific literature, the patient and clinical practice. Our goal is to provide the optimal patient product for each specialty, from the clinical office setting to the operating room, by tirelessly testing our products in our electrical, mechanical, and clinical test labs. All SIGNAL GEAR® products will be the best or we won’t sell them.

**Booth # 17**  
**UCB**  
1950 Lake Park Drive  
Smyrna, GA 30080  
Phone: 770.970.7500  
uscommunications@ucb.com

UCB Inc is a global biopharm company that aspires to be the patient-centric global biopharmaceutical leader, transforming the lives of people living with severe diseases. VIMPAT (Lacosamide) tablets and oral solution are indicated as adjunctive therapy in the treatment of partial-onset seizures in patients with epilepsy aged 17 years and older.
**EXHIBITORS — CONTINUED**

Booth # 7  
**Visualase, Inc.**  
8058 El Rio Street  
Houston, TX 77054  
Phone: 832.577.7773  
Fax: 713.741.0122  

The Visualase Thermal Therapy System is an MRI-guided, minimally invasive laser ablation system which allows for continuous monitoring of an ablation in real-time. The system is FDA-cleared for the ablation of soft tissue including for use in neurosurgical procedures (see www.visualaseinc.com for complete indication). More than 25 US centers have performed >250 epileptogenic foci and >225 brain tumor procedures.

Booth # 26  
**Wolters Kluwer Health**  
Two Commerce Square  
2001 Market Street  
Philadelphia, PA 19103  
Phone: 612.259.8114  
Fax: 612.677.3059

Booth # 25  
**WR Medical Electronics Co.**  
1700 Gervais Avenue  
Maplewood, MN 55109  
Phone: 651.604.8400  
Fax: 651.604.8499

WR Medical Electronics specializes in autonomic function labs for quantitative assessments of the sympathetic and parasympathetic nervous system. Perform head-up tilt analysis, QSART studies, and beat-to-beat blood pressure and heart rate measurements. Non-invasive, standardized, and simple-to-perform examination of diabetic neuropathy and orthostatic intolerance. Measure sweat output, heart rate and BP variability, and thermal/vibration thresholds. Featuring the Q-Sweat™ for Quantitative Sweat Measurement, the HRV Acquire and the CASE IV™ Quantitative Sensory Testing System.
ACNS is pleased to introduce the following Evening Programs. Each two-hour session is supported and programmed by a single supporting company and will feature presentations on topics and technologies selected by the company. Beverages and snacks will be served. CME credits are NOT available for the Evening Programs.

Evening Programs will be held on Thursday, February 6 from 5:30 – 7:30 PM.

**DENSE ARRAY EEG IN CLINICAL PRACTICE**
Presented by: Electrical Geodesics, Inc. (EGI)
Room: International C, 6th floor
Instructors: Susan T. Herman, MD; Phan Luu, PhD; Don Tucker, PhD

**Overview of Dense Array EEG in the ICU and Neurosurgical Planning**
*Susan T. Herman, MD*

Advances in sensor application allow up to 256 channels to be recorded in continuous monitoring, providing greater information on cerebral pathology, and allowing registration with MRI for relating EEG pathologies to structural imaging.

**Routine Dense Array EEG: From Rapid Overview to High Resolution Analysis**
*Phan Luu, PhD*

Conventional clinical review of the Ten-Twenty montage can be facilitated by automated dense array detection of possible pathological features, such as spikes, which can then be clustered and registered with the MRI for source localization.

**Sensitivity of dEEG vs. MEG Compared with Intracranial Recording in Neurosurgical Planning**
*Don Tucker, PhD*

Simultaneous recording of intracranial EEG (icEEG) has now been conducted with non-invasive scalp dEEG and with whole head MEG. For mesial temporal spikes identified in icEEG, 50% are detected in dEEG, whereas 25% are detected in whole head MEG.

**EEG REVIEW AND ANALYSIS USING PERSYST qEEG TRENDING, ARTIFACT REDUCTION, SEIZURE DETECTION, AND SPIKE DETECTION**
Presented by: Persyst Development Company
Room: International B, 6th floor
Instructors: Mark L. Scheuer, MD

<table>
<thead>
<tr>
<th>Time</th>
<th>Event</th>
</tr>
</thead>
</table>
| 5:30 – 5:55 PM  | Introduction to EEG Trending, Seizure Detection, and Spike Detection using Persyst  
Mark L. Scheuer, MD |
| 5:55 – 7:05 PM  | Hands-On Interactive Case Studies  
Mark L. Scheuer, MD and Persyst Staff |
| 7:05 – 7:30 PM  | Case Presentations  
Attendees should bring their own laptops and arrive early to the session (approximately 5:00 PM) to directly participate in the interactive cases. Persyst staff will install Persyst software onto attendee PCs prior to the program. |
The Component Neuromonitoring System
Continuous EEG and Integrated Physiology for the ICU

Introducing a Systems Approach to Brain Monitoring:

Integrated EEG (iEEG)
is the real-time integration of EEG and other physiology.

The CNS Monitor offers the entire “EEG alphabet.”
- cEEG: Full-function, continuous EEG
- vEEG: Synchronous video recording
- qEEG: Powerful EEG processing
- aEEG: Amplitude integrated EEG for neonatal monitoring
- iEEG: EEG trends time synchronized with other physiology

CNS-200 Multimodal Data Integration System

CNS EEG Display & Persyst Remote Review

CNS-200 Multimodal Data Integration System

Physiology & EEG Display

2-Channel aEEG Display

Contact Us:
224 S. Maple St.
Ambler, PA 19002
info@moberg.com
215-283-0860

Visit us at ACNS 2014 in Atlanta: Booth #2-3

MOBERG ICU Solutions
www.Moberg.com
SAVE the DATE!
2015 ACNS Annual Meeting & Courses
JW Marriott Houston - Houston, Texas
February 3-8, 2015