2015 ACNS
Annual Meeting & Courses
February 3-8, 2015
JW Marriott Houston
Houston, Texas
www.acns.org
### ANNUAL COURSES OVERVIEW

**Tuesday, February 3, 2015**

<table>
<thead>
<tr>
<th>Time</th>
<th>Event</th>
<th>Location</th>
</tr>
</thead>
<tbody>
<tr>
<td>9:00AM - 5:00PM</td>
<td>Neurophysiologic Intraoperative Monitoring (NIOM): Part I</td>
<td>Salon B, 2nd floor</td>
</tr>
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**Wednesday, February 4, 2015**

<table>
<thead>
<tr>
<th>Time</th>
<th>Event</th>
<th>Location</th>
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<tbody>
<tr>
<td>7:00 - 8:30AM</td>
<td>EP Reading Session</td>
<td>Salon B, 2nd floor</td>
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<tr>
<td></td>
<td>Introduction to Stereo EEG</td>
<td>Salon C, 2nd floor</td>
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<tr>
<td>9:00AM - 5:00PM</td>
<td>Neurophysiologic Intraoperative Monitoring (NIOM): Part II</td>
<td>Salon B, 2nd floor</td>
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<tr>
<td></td>
<td>Electrocorticography and Intracranial EEG</td>
<td>Salon C, 2nd floor</td>
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**Thursday, February 5, 2015**

<table>
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<tr>
<th>Time</th>
<th>Event</th>
<th>Location</th>
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<tbody>
<tr>
<td>7:00 - 8:30AM</td>
<td>Neonatal EEG</td>
<td>Salon B, 2nd floor</td>
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<tr>
<td></td>
<td>EMG and EEG Technology</td>
<td>Salon C, 2nd floor</td>
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<tr>
<td></td>
<td>New Directions in Sleep Medicine</td>
<td>Bexar/Travis/Nueces, 2nd floor</td>
</tr>
<tr>
<td>9:00AM - 12:00PM</td>
<td>EMG</td>
<td>Salon C, 2nd floor</td>
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<tr>
<td></td>
<td>Video – EEG</td>
<td>Bexar/Travis/Nueces, 2nd floor</td>
</tr>
<tr>
<td>9:00AM- 5:00PM</td>
<td>ICU EEG</td>
<td>Salon B, 2nd floor</td>
</tr>
<tr>
<td>11:00AM — 2:30PM</td>
<td>Program Directors Symposium</td>
<td>Harris, 2nd floor</td>
</tr>
<tr>
<td>1:00 — 2:30PM</td>
<td>Applied Autonomic Neurophysiology</td>
<td>Bexar/Travis/Nueces, 2nd floor</td>
</tr>
<tr>
<td>1:00 — 5:00PM</td>
<td>Business in Clinical Neurophysiology</td>
<td>Salon C, 2nd floor</td>
</tr>
<tr>
<td>3:00 — 5:00PM</td>
<td>Case Studies in Peripheral Neurophysiology</td>
<td>Bexar/Travis/Nueces, 2nd floor</td>
</tr>
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**ACNS Executive Office**

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**MESSAGES FROM COURSE AND PROGRAM COMMITTEE CO-CHAIRS**

**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 2015 Annual Meeting and Courses in Houston, Texas.

The Annual courses, expertly fashioned by Dr. Tobias Loddenkemper will commence on Tuesday, February 3 and as always will be the best courses one can find on the latest clinical methods in both ICU and Intraoperative Monitoring; peripheral nerve studies; EEG, whether neonatal or invasive; Autonomic Neurophysiology; and in technology and other practical, business updates. This year’s new course; New Directions in Sleep Medicine hosted by Dr. Madeline Grigg-Damberger, will be a great educational addition to the Annual Courses program.

The Annual Meeting begins on Friday, February 6 and continues through Sunday, February 8. The Program Committee, led by Dr. William O. Tatum and Dr. Jaime Lopez, has assembled an impressive array of lectures and symposia on the latest innovations and developments in all forms of Clinical Neurophysiology brought to you from the world’s leading investigators and teachers. The roster of remarkably diverse topics underscores how rapidly our field is expanding. This year’s Joint International Symposia will bring a great new look into Clinical Neurophysiology from around the world. The variety of symposia, workshops and Special Interest Groups (SIGs) will provide something for everyone with a strong interest in Clinical Neurophysiology.

The ACNS Council and I want to extend a warm welcome to our international attendees and also to neurophysiology fellows and others new to the meeting. 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 to discuss very interests insights into the function of the human nervous system.

*Sincerely,*

**Tobias Loddenkemper, MD, FACNS**

**William O. Tatum, DO, FACNS**

**Jaime R. Lopez, MD, FACNS**

---

**PRESIDENT’S MESSAGE**

**Dear Friends & Colleagues,**

On behalf of the American Clinical Neurophysiology Society (ACNS), it is my great pleasure to welcome you to the 69th Annual Meeting & Courses in Houston, Texas.

The Annual courses, expertly fashioned by Dr. Tobias Loddenkemper will commence on Tuesday, February 3 and as always will be the best courses one can find on the latest clinical methods in both ICU and Intraoperative Monitoring; peripheral nerve studies; EEG, whether neonatal or invasive; Autonomic Neurophysiology; and in technology and other practical, business updates. This year’s new course; New Directions in Sleep Medicine hosted by Dr. Madeline Grigg-Damberger, will be a great educational addition to the Annual Courses program.

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*Aatif M. Husain, MD, FACNS*

President
ACNS COURSE & PROGRAM COMMITTEES

Course Committee
Chair:
Tobias Loddenkemper, MD, FACNS
Children’s Hospital Boston

Members:
Nicholas S. Abend, MD
Children’s Hospital of Philadelphia
Cecil D. Hahn, MD, MPH, FACNS
The Hospital for Sick Children

Lawrence J. Hirsch, MD, FACNS
Yale University
Marc R. Nuwer, MD, PhD, FACNS
UCLA Medical Center
Juan Ochoo, MD
University of South Alabama
Saurabh R. Sinha, MD, PhD
Duke University Medical Center

Program Committee
Co-Chairs:
Jaime Lopez, MD, FACNS
Stanford University
William O. Tatum, IV, DO, FACNS
Mayo Clinic Florida

Members:
Nicholas S. Abend, MD
Children’s Hospital of Philadelphia
Imran I. Ali, MD, FACS
University of Toledo
Salah A. Almubarak, MD, FRCPC, FACS
Royal University Hospital
Anto Bagic, MD, PhD, FACS
University of Pittsburgh
Meriem Bensalem-Owen, MD, FACS
University of Kentucky
Bernard Allan Cohen, PhD, FACS
Neurological Monitoring Associates, LLC
Rafael de Castro, MD
Neurolife Natal
Frank W. Drislane, MD, FACS
Beth Israel Deaconess Medical Center
Jonathan C. Edwards, MD, FACS
Medical University of South Carolina
Ronald Emerson, MD, FACS
Hospital for Special Surgery
William B. Gallentine, DO, FACS
Duke University Medical Center
Gloria Galloway, MD, FACS
Ohio State University
Cecil D. Hahn, MD, MPH, FACS
The Hospital for Sick Children
Mark Hallett, MD, FACS
National Institutes of Health
Lawrence J. Hirsch, MD, FACS
Yale University
Aatif M. Husain, MD, FACS
Duke University Medical Center
Akio Ikeda, MD, PhD
Kyoto University Graduate School of Medicine
Mohammad MU Kabiraj, Sr., MBBS, PhD
Prince Sultan Military Medical City
Ekrem Kutluay, MD
Medical University of South Carolina
Suzette M. LaRoche, MD, FACS
Emory University School of Medicine
Jong Woo Lee, MD, PhD, FACS
Brigham & Women’s Hospital
Alan D. Legatt, MD, PhD, FACS
Montefiore Medical Center
Daniela N. Minecan, MD, FACS
University of Michigan Health System
Suraj Muley, MD, FACS
St. Joseph’s Hospital & Medical Center
Heidi Munger Clary, MD, MPH
Wake Forest University
Christos Papadelis, PhD
Harvard Medical School
Ex-Officio:
Tobias Loddenkemper, MD, FACNS
Children’s Hospital Boston

Eva K. Ritzl, MD
Johns Hopkins University
Mark Ross, MD, FACS
Mayo Clinic Arizona
Devon Rubin, MD
Mayo Clinic Florida
Raj D. Sheth, MD
Mayo Clinic Florida / Nemours Clinic-Florida
Saurabh R. Sinha, MD, PhD
Duke University Medical Center
John Stern, MD
UCLA School of Medicine
Amit Verma, MBBS
The Methodist Hospital
Francis O. Walker, MD, FACS
Wake Forest University
Greg Worrell, MD
Mayo Clinic
Courtney J. Wusthoff, MD
Stanford University
Ex-Officio:
Tobias Loddenkemper, MD, FACNS
Children’s Hospital Boston
Stephan U. Schuele, MD, MPH, FACS
Northwestern University
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. Koii, MD
1968  Gian-Ermiilio Chattian, 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. Lombaros, MD
1984  Robert J. Gunnitt, MD
1985  Andrew J. Gabor, MD, PhD
1986  John A. Wadd, MD,
1987  Frank W. Shatbrough, MD,
1988  Joan B. Crocco, MD, FACNS
1989  Barry R. Tharp, MD,
1990  Timothy A. Pedley, MD, FACNS
1991  Ernst Niedermeyer, MD, FACNS
1992  Barbara F. Westmoreland, MD, FACNS
1993  Jerome Engel, MD, PhD, FACNS
1994  Marc R. Nuwer, MD, PhD, FACNS
1995  Michael J. Aminoff, MD, FACNS
1996  John S. Ebersole, MD, FACNS
1997  Solomon L. Moshé, MD, FACNS
1998  Warren T. Blume, MD, FACNS
1999  C. William Erwin, MD, FACNS
2000  Michael R. Sperling, MD, FACNS
2001  Eli M. Mizrahi, MD, FACNS
2002  Bruce J. Fisch, MD, FACNS
2003  Charles M. Epstein, MD, FACNS
2004  Donald L. Schomer, MD, FACNS
2005  Ronald G. Emerson, MD, FACNS
2006  Richard P. Brenner, MD, FACNS
2007  Mark A. Ross, MD, FACNS
2008  Alan D. Legatt, MD, PhD, FACNS
2009  Garett J. Parry, MD, FACNS
2010  Peter W. Kaplan, MB, FRCP, FACNS
2011  Douglas R. Nordli, Jr., MD, FACNS
2012  Susan T. Herman, MD, FACNS
2013  Frank W. Drislane, MD, FACNS
*Deceased

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.
GENERAL MEETING INFORMATION

Business Center
The JW Marriott Houston offers a self-service Business Center, located in the main lobby next to Starbucks. It is accessible 24 hours/day. Services include pay-per-use computers, copiers, fax machine and internet access. It also includes a laptop station with wireless access including first 10 minutes at no cost.

Business Meeting
The ACNS Annual Business Meeting will be held in Salon B, from 7:00 – 7:30PM on Saturday February 7, 2015. This meeting is open to all attendees, but only ACNS Members may vote.

Cell Phone Protocol
Please ensure that cell phone ringers, pagers and electronic devices are silenced or turned off during all sessions.

Certificate of Attendance & CME Certificate
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 (included in this packet). 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, 2015.

Language
English is the official language of the ACNS Annual Meeting & Courses.

Lost & Found
Please notify staff at the ACNS Registration Desk (Salon B Foyer, 2nd floor) if you have lost or found an item during the course of the Annual Meeting & Courses.

Messages
A non-electronic message board will be available in the Registration Desk area for attendees to post notes or leave messages for other attendees. Please remember to check for any messages that may be left for you.

Photography and Recording Policy
Photography or video or audio recording of sessions, materials presented in session, or exhibits without written permission from ACNS is strictly prohibited. Please note that photographs and video taken by or on behalf of ACNS of event activities and attendees shall be property of ACNS.

Poster Sessions
Authors will be present during poster tours between 3:00 – 4:00PM on Friday, February 6 and 1:00 – 2:00PM on Saturday, February 7 for discussion. Poster abstracts and presentation dates can be found on page 54.

Friday, February 6, 2015
7:00AM – 5:00PM
Exhibit & Poster Hall, Liberty Hall

Saturday, February 7, 2015
7:00AM – 2:00PM
Exhibit & Poster Hall, Liberty Hall

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.

Smoking Policy
Smoking is not permitted during any meeting activity or event.

Special Needs
If you have any health issues for which you may require special accommodations or assistance, please notify the ACNS staff at the Registration Desk (Salon B Foyer, 2nd floor)
**GENERAL MEETING INFORMATION**

**JW Marriott Houston Floorplan**

**Nearby Restaurants**

**American**
- Grand Lux Café (W)
  5000 Westheimer Rd.
  713.626.1700
- Houston’s (V)
  5888 Westheimer Rd.
  713.975.1947
- Kenny’s & Ziggy’s Deli* (W)
  2327 Post Oak Blvd.
  713.871.8883

**French**
- Sage 400* (W)
  2800 Sage Rd.
  713.961.9566
- Yia Yia Mary’s* (V)
  4747 San Felipe St.
  713.840.8665

**Barbeque**
- Goode Company* BBQ (4.1 mi)
  5109 Kirby Dr.
  713.522.2530
- Luling City Market BBQ* (V)
  4726 Richmond Ave.
  713.871.1903

**Italian**
- Arco Doro* (W)
  5000 Westheimer Rd.
  713.621.6888
- Osteria Mazzantini* (V)
  2200 Post Oak Blvd.
  713.993.9898

**Steak**
- The Capital Grille (W)
  5365 Westheimer Rd.
  713.623.4600
- Morton’s the Steak House (W)
  5000 Westheimer Rd.
  713.629.1946
- Pappas Bros. Steakhouse* (V)
  5839 Westheimer Rd.
  713.780.7352

**Seafood**
- Truluck’s (W)
  5350 Westheimer Rd.
  713.783.7270
- Tex — Mex
  - Caracol* (V)
    2200 Post Oak Blvd.
    713.622.9996
  - Escalante’s* (V)
    4053 Westheimer Rd.
    713.623.4200
  - El Tiempo* (3.3 mi)
    3130 Richmond Ave.
    713.807.1600

(W) — Walking Distance
(V) — Hotel Valet Shuttle
(*) — Unique to Houston
CME INFORMATION

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, physiatrists, neuroradiologists, 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.

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, especially invasive EEG
- Sleep, Use of new scoring system, implications for patient care

Changes in Behavior/Practice
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.

Policy on Financial 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 are peer reviewed by two members of the ACNS CME Committee with no relationships. If bias is found, the presenter is asked to make changes to the presentation and it is 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 is printed in the final program for the activity. A learner may request additional information regarding the nature of a planner or speaker’s disclosure if “No Relevant Relationships” has been indicated below. To request additional information, contact the ACNS Executive office at info@acns.org.
**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 end 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.

Course-specific learning objectives are included on pages 15-20.

**Annual Meeting Learning Objectives**

At the end 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.

Session-specific learning objectives are included on pages 21-32.

**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 Polices 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.

**Important Dates**

<table>
<thead>
<tr>
<th>Event</th>
<th>Date</th>
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<tbody>
<tr>
<td>CME Certificate Program Opens</td>
<td>February 8, 2015</td>
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<tr>
<td>CME Certificate Claim Deadline</td>
<td>April 10, 2015</td>
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</tbody>
</table>
CONFLICT OF INTEREST DISCLOSURES

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<td>Alan Legatt, MD, PhD, FACNS</td>
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<td>Several companies that market health care goods or services; none of them are related to the subject of my presentation. (c)</td>
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<td>Charles Epstein, MD, FACNS</td>
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<td>MD Anderson Cancer Center</td>
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<td>Stanford University</td>
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<td>Erik Ortega</td>
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<td>Hiroshi Otsubo, MD</td>
<td>The Hospital for Sick Children</td>
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<td>Josef Parvisi, MD PhD</td>
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<td>Claudia Esther Paz Navarro</td>
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<td>Howard King</td>
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<td>Anthony Ritaccio, MD</td>
<td>Albany Medical Center</td>
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<td>Eric Rosenthal, MD</td>
<td>Massachusetts General</td>
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<td>Alexander Rotenberg, M.D., Ph.D.</td>
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<td>Eisai Co. Ltd. (a); Neuronmotion Inc. (c); NeuroRex Inc. (e)</td>
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<td>Steven Schachter, MD</td>
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<td>Sarah Schmitt, MD</td>
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<td>Reiner See, MD</td>
<td>Massachusetts General Hospital / Harvard Medical School</td>
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<td>Indranil Sen-Gupta, MD</td>
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<td>Mouhsin Shafi, MD PhD</td>
<td>Beth Israel Deaconess Medical Center and Harvard Medical School</td>
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<td>Deepal Shah, MD</td>
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<td>Parastou Shilian, D.O.</td>
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<td>University Of Michigan</td>
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<td>Adithya Sivaraja, MD</td>
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<td>Norman So, MD</td>
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<td>Janet Soul, MD</td>
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<td>Jeffrey Tenney, MD</td>
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<td>Elizabeth Thiele</td>
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<td>Agnes Trebuchon, Md, PhD</td>
<td>Universite Aix-Marseille</td>
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<td>Henrikas Vaitkevicius, MD</td>
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<td>Armando Valdes-Tello, MD</td>
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<tr>
<td>Michel van Putten, MD MSc PhD</td>
<td>University of Twente</td>
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<td>John Vargas, MD</td>
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<td>Renato Verduga</td>
<td>Clinica Alemana-Universidad del Desarrollo</td>
<td>See Addendum</td>
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<td>Joy Vijayan, MD</td>
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<tr>
<td>Courtney Wusthoff, MD</td>
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<tr>
<td>Ji Yeoun Yoo, MD</td>
<td>Mt. Sinai Hospital</td>
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</table>
AWARD RECIPIENTS & LECTURES

Friday, February 6, 2015
2015 Pierre Gloor Award Presentation & Lecture
“Cortical Activities Associated with Voluntary and Involuntary Movements”
Hiroshi Shibasaki, MD, PhD, FACNS

The Gloor Award is presented annually for outstanding current contributions to clinical neurophysiology research. Prof. Shibasaki will be recognized and will deliver the 2015 Gloor Address on Friday, February 6, 2015 in Salon B. Dr. Shibasaki is an Emeritus Professor at Kyoto University and former president of IFCN.

Saturday, February 7, 2015
2015 Herbert H. Jasper Award
A Personal Perspective on 50 Years of Clinical Neurophysiology
John Ebersole, MD, FACNS

The Jasper Award is presented annually to an individual who has made a lifetime of outstanding contributions to the field of clinical neurophysiology. Dr. Ebersole will be recognized during the general session on Saturday, February 7, 2015 in Salon B. Dr. Ebersole is Medical Director of the MEG Center at Overlook Hospital in Summit, New Jersey and a former president of ACNS.

2015 Robert S. Schwab Award
“Fasciculation Potentials: The Enigma”
Mamede de Carvalho, MD

The Schwab Award is presented annually to an individual who has made significant contributions in the area of clinical neurophysiology. Prof. de Carvalho will be recognized and will deliver the 2015 Schwab Address on Saturday, February 7, 2015 in Salon B. Prof. de Carvalho is Professor of Physiology at the Instituto de Medicina Molecular, University of Lisbon and former officer of IFCN.

NETWORKING AND SOCIAL EVENTS

Welcome Reception
Friday, February 6, 2015
Liberty Hall; 1st floor
7:00 – 8:00PM

Dr. Aatif Husain, MD, FACNS formally invites all Annual Meeting delegates to attend the ACNS Welcome Reception on Friday, February 6, from 7:00 – 8:00PM in ACNS Exhibit Hall, Liberty Hall, 1st 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
Sunday, February 8, 2015
Harris; 2nd floor
7:00 – 8:00AM

If you signed up to be a Mentor or Mentee, please join us at breakfast in Harris on the 2nd floor!
# PROGRAM AGENDA | ANNUAL COURSES

## Annual Courses Overview

<table>
<thead>
<tr>
<th>Day</th>
<th>Time</th>
<th>Event</th>
<th>Location</th>
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<tbody>
<tr>
<td><strong>Tuesday, February 3, 2015</strong></td>
<td></td>
<td><strong>9:00AM - 5:00PM</strong> Neurophysiologic Intraoperative Monitoring (NIOM): Part I</td>
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<tr>
<td><strong>Wednesday, February 4, 2015</strong></td>
<td></td>
<td><strong>7:00 - 8:30AM</strong> EP Reading Session</td>
<td>Salon B, 2nd floor</td>
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<td><strong>9:00AM - 5:00PM</strong> Neurophysiologic Intraoperative Monitoring (NIOM): Part II</td>
<td>Salon C, 2nd floor</td>
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<td><strong>Introduction to Stereo EEG</strong></td>
<td>Salon C, 2nd floor</td>
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<td><strong>9:00AM - 5:00PM</strong> Electroencephalography and Intracranial EEG</td>
<td>Salon C, 2nd floor</td>
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<td><strong>Thursday, February 5, 2015</strong></td>
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<td><strong>7:00 - 8:30AM</strong> Neonatal EEG</td>
<td>Salon B, 2nd floor</td>
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<td><strong>EMG and EEG Technology</strong></td>
<td>Salon C, 2nd floor</td>
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<td><strong>New Directions in Sleep Medicine</strong></td>
<td>Bexar/Travis/Nueces, 2nd floor</td>
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<td></td>
<td><strong>9:00AM - 12:00PM</strong> EMG</td>
<td>Salon C, 2nd floor</td>
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<td><strong>Video — EEG</strong></td>
<td>Bexar/Travis/Nueces, 2nd floor</td>
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<td><strong>9:00AM - 5:00PM</strong> ICU EEG</td>
<td>Salon B, 2nd floor</td>
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<td><strong>11:00AM — 2:30PM</strong> Program Directors Symposium</td>
<td>Harris, 2nd floor</td>
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<td><strong>1:00 — 2:30PM</strong> Applied Autonomic Neurophysiology</td>
<td>Bexar/Travis/Nueces, 2nd floor</td>
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<td><strong>1:00 — 5:00PM</strong> Business in Clinical Neurophysiology</td>
<td>Salon C, 2nd floor</td>
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<td><strong>3:00 — 5:00PM</strong> Case Studies in Peripheral Neurophysiology</td>
<td>Bexar/Travis/Nueces, 2nd floor</td>
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</table>
**Tuesday, February 3, 2015**

**Neurophysiologic Intraoperative Monitoring (NIOM) Part I**

9:00AM - 5:00PM  
Location: Salon B, 2nd floor  
*Co-Chairs: Jaime R. López, MD, FACNS and Michael McGarvey, MD, FACNS*

**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 the 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>
<th>Speaker</th>
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<tbody>
<tr>
<td>9:00AM</td>
<td>Welcome &amp; Introduction</td>
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<tr>
<td>9:05AM</td>
<td>BAEP Monitoring</td>
<td>Alan D. Legatt, MD, PhD, FACNS</td>
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<td>9:45AM</td>
<td>SEP Monitoring</td>
<td>Andres A. Gonzalez, MD, MMM, FACNS</td>
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<td>10:25AM</td>
<td>Break</td>
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<td>10:40AM</td>
<td>MEP Monitoring</td>
<td>Ronald Emerson, MD, FACNS</td>
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<td>11:20AM</td>
<td>EEG and Doppler Ultrasound Monitoring</td>
<td>Michael McGarvey, MD, FACNS</td>
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<td>12:00PM</td>
<td>Panel Discussion</td>
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<td>12:15PM</td>
<td>Lunch</td>
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<td>1:15PM</td>
<td>Mapping of Cortical and Subcortical Brain Structures</td>
<td>Mirela V. Simon, MD, MSc, FACNS</td>
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<td>1:55PM</td>
<td>Monitoring of Spinal nerve Roots</td>
<td>Monica Islam, MD</td>
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<td>2:35PM</td>
<td>Monitoring of Peripheral Nerve Surgery</td>
<td>Leo T. Happel, PhD</td>
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<td>3:15PM</td>
<td>Break</td>
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<td>3:30PM</td>
<td>Anesthetic Management and IOM</td>
<td>David Ferson, MD</td>
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<td>4:10PM</td>
<td>Case Presentations and Discussions</td>
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<td>4:50PM</td>
<td>Panel Discussion</td>
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**Wednesday, February 4, 2015**

**EP Reading Session**

7:00 - 8:30AM  
Location: Salon B, 2nd floor  
*Chair: Alan Legatt, MD, PhD, FACNS*

**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:**

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<th>Time</th>
<th>Activity</th>
<th>Speaker</th>
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<tr>
<td>7:00AM</td>
<td>Brainstem Auditory Evoked Potentials (BAEPs)</td>
<td>Alan D. Legatt, MD, PhD, FACNS</td>
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<td>7:30AM</td>
<td>Visual Evoked Potentials (VEPs)</td>
<td>Elayna Rubens, MD</td>
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<td>8:00AM</td>
<td>Somatosensory Evoked Potentials (SEPs)</td>
<td>Ronald Emerson, MD, FACNS</td>
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**Introduction to Stereo-EEG**

7:00 - 8:30AM  
Location: Salon C, 2nd floor  
*Co-Chairs: Stephan U. Schuele, MD, MPH, FACNS and Nitin Tandon MD, FAANS*

**Objectives:**
At the conclusion of this course, participants will be able to:
1. Understand the principles underlying Stereo EEG including patient selection and targeting electrode placement;
2. Demonstrate familiarity with the fundamentals of pre and postoperative image processing and co-registration;
3. Explain about the principles of stereotactic surgical implantation, pitfalls and complications.

**Agenda:**

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<th>Time</th>
<th>Activity</th>
<th>Speaker</th>
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<tr>
<td>7:00AM</td>
<td>Patient and Electrode Selection</td>
<td>Stephan U. Schuele, MD, MPH, FACNS</td>
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<td>7:25AM</td>
<td>Questions</td>
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<tr>
<td>7:30AM</td>
<td>Image Processing and Surgical Planning</td>
<td>Giridhar Kalamangalam, MD, DPhil</td>
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<td>7:55AM</td>
<td>Questions</td>
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<tr>
<td>8:00AM</td>
<td>Nuts and Bolts of Surgical Implantation</td>
<td>Nitin Tandon, MD, FAANS</td>
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<td>8:25AM</td>
<td>Questions</td>
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Neurophysiologic Intraoperative Monitoring (NIOM) Part II

9:00AM - 5:00PM
Location: Salon B; 2nd floor
Co-Chairs: Jaime Lopez, MD, FACNS and Michael McGarvey, MD, FACNS

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 the 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:
9:00AM Monitoring Cerebral and Spinal Endovascular Procedures
Viet Nguyen, MD
9:40AM Electrocorticography During Pediatric Epilepsy Surgery
Jurriaan Peters, MD
10:20AM Break
10:35AM EMG Monitoring of Central Motor Pathways During Spine Surgery
Stan Skinner, MD, FACNS
11:15AM Regulatory, Medical-Legal and Coding/Billing Issues
Marc R. Nower, MD, PhD, FACNS
11:55AM Panel Discussion
12:10PM Lunch
1:10PM Monitoring of Spinal D-Waves
Eva K. Ritzl, MD
1:50PM Monitoring of Motor Cranial Nerves and Cranial Nerve Nuclei
Jaime R. López, MD, FACNS
2:30PM Evidenced Based Studies in IOM
Jonathan C. Edwards, MD, FACNS
3:10PM Break
3:25PM Troubleshooting During IOM
Brett Netherton, MS, FASNM, CNIM
4:05PM Case Presentations and Discussion
4:45PM Panel Discussion

Electrocorticography and Intracranial EEG
9:00AM - 5:00PM
Location: Salon C, 2nd floor
Co-Chairs: Stephan Schuele, MD, MPH, FACNS and Greg Worrell, MD

Objectives:
At the conclusion of this course, the learner should be able to:
1. Identify patients from Phase 1 evaluations that are good candidates for a Phase 2 evaluation;
2. Know what EEG patterns, seen on invasive EEG, are more likely to have a “good” surgical outcome and;
3. Have an updated understanding of ongoing research into basic physiology of focal onset epilepsy based on invasive EEG techniques.

Agenda:
9:00AM Introduction & Overview
9:15AM Phase 1 Evaluations that Lead to Phase 2 Testing
Giridhar Kalamangalam, MD, DPhil
9:45AM Choosing Phase 2 Electrodes
Stephan U. Schuele, MD, MPH, FACNS
10:15AM Utility of Source Localization and Magnetoencephalography
Richard C. Burgess, MD, PhD, FACNS
10:45AM Break
11:00AM Patterns of Seizure Onsets & Spread, Underlying Pathological Substrates, Surgical Outcomes in Adults
Lawrence J. Hirsch, MD, FACNS
11:30AM Discussions and Demonstrations of Patterns of Seizure Onsets & Spread, Underlying Pathological Substrates, Surgical Outcomes in Children
Tobias Loddenkemper, MD, FACNS
12:00PM Lunch
1:00PM The Use on Invasive Electrodes to Map “Epileptic Zones”
William C. Stacey, MD, PhD
1:45PM Wideband Intracranial EEG and Localization
Greg Worrell, MD
2:30PM Case Presentation I: Implantation Strategy
Jurriaan M. Peters, MD
3:00PM Break
3:15PM Case Presentation II: Implantation Strategy
Jay Gavvala, MD
3:45PM Functional Mapping
Nitin Tandon, MD, FAANS
4:30PM Case Presentation III: Mapping Strategy
Giridhar Kalamangalam, MD, DPhil
New Directions in Sleep Medicine
7:00 - 8:30AM
Location: Bexar/Travis/Nueces; 2nd floor
Chair: Madeleine Grigg-Damberger, MD, FACNS
Objectives:
At the conclusion of this activity, participants will be able to:
1. Realize obstructive sleep apnea (OSA) is highly prevalent in patients with stroke, associated with poorer outcomes after stroke, and prospective studies show OSA independently increases risks of incident ischemic stroke, composite risk of stroke, TIA and death;
2. Appreciate that Juvenile Myoclonic Epilepsy is an epilepsy syndrome profoundly affected by circadian rhythms and Nocturnal Frontal Lobe Epilepsy by sleep itself;
3. Know that the goals of chronopharmacology are to determine whether a particular drug or treatment is affected by endogenous circadian rhythms, and whether aligning it to endogenous circadian rhythms results in optimal levels, improved seizure control, and the least adverse or toxic effects;
4. Understand how short sleep duration, fragmented sleep, varying degrees of intermittent nocturnal hypoxemia, and excessive daytime sleepiness (EDS) in older adults are associated with Minimal Cognitive Impairment (MCI), incident Alzheimer’s disease (AD) and rates of cognitive decline.

Agenda:
7:00AM  Chronobiology and Chronopharmacology Applied to Epilepsy
         Tobias Loddenkemper, MD, FACNS
7:30AM  Sleep and Stroke
         Hrayr Attarian, MD
8:00AM  Sleep as a Robust Biomarker of Neurodegenerative Diseases
         Madeleine M. Grigg-Damberger, MD, FACNS

EMG
9:00AM - 12:00PM
Location: Salon C; 2nd floor
Chair: Francis O. Walker, MD, FACNS
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;

Agenda:
9:00AM  Motor Nerve Conductions and F-waves
         Paul E. Barkhaus, MD
10:00AM  Neuromuscular Ultrasound and Entrapment Neuropathies
          Francis O. Walker, MD, FACNS
11:00AM  Muscle Disease and EMG
          Elliot Dimberg, MD
Thursday, February 05, 2015, continued

**Video EEG**
9:00AM - 12:00PM  
Location: Bexar/Travis/Nueces; 2nd floor  
**Co-Chairs:** William Tatum, DO, FACNS and Tobias Loddenkemper, MD, FACNS

**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:**  
9:00AM  Technical aspects of Video EEG  
Terrence D. Lagerlund, MD, PhD  
9:20AM  Epilepsy Surgery Evaluation in Pediatrics with VEM  
Phillip Pearl, MD, FACNS  
10:00AM  Video-EEG Pearls in pediatric patients  
Tobias Loddenkemper, MD, FACNS  
10:40AM  Scalp Epilepsy Surgery evaluation in adults with VEM  
Meriem Bensalem-Owen, MD, FACNS  
11:20AM  Video EEG Pearls in Adult Patients  
William O. Tatum, DO, FACNS

**ICU EEG**
9:00AM - 5:00PM  
Location: Salon B, 2nd floor  
**Co-Chairs:** Cecil Hahn, MD, MPH, FACNS and Lawrence J. Hirsch, MD, FACNS

**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:**  
9:00AM  Overview of ICU EEG monitoring in Neonates, Children and Adults  
Nicholas S. Abend, MD  
9:30AM  Q&A Discussion  
9:40AM  Guidelines and Nomenclature for ICU EEG Monitoring  
Susan T. Herman, MD, FACNS  
10:00AM  Q&A Discussion  
10:10AM  Coffee Break  
10:30AM  cEEG Interpretation: Assessment of Background, Sleep, Reactivity & Artifacts  
Nicolas Gaspar, MD, PhD  
10:50AM  Q&A Discussion  
11:00AM  cEEG Interpretation: Seizures and Periodic Patterns  
Suzette M. LaRoche, MD, FACNS  
11:20AM  Q&A Discussion  
11:30AM  cEEG Interpretation: Neonates  
Mark Scher, MD  
11:50 AM  Q&A Discussion  
12:00PM  Lunch  
1:00PM  Quantitative EEG for Seizure and Ischemia Detection  
M. Brandon Westover, MD, PhD  
1:20PM  Q&A Discussion  
1:30PM  Logistics of ICU EEG Monitoring  
Cecil Hahn, MD, MPH, FACNS  
1:50PM  Q&A Discussion  
2:00PM  Finances, Billing and Coding  
Marc R. Nuwer, MD, PhD, FACNS  
2:20PM  Q&A Discussion  
2:30PM  Coffee Break  
2:50PM  Treatment of Non-Convulsive Seizures and Status Epilepticus  
Jan Claassen, MD, PhD  
3:10PM  Q&A Discussion  
3:20PM  Treatment of Postanoxic Coma/Myoclonus  
Thomas P. Bleck, MD, FACNS  
3:40PM  Q&A Discussion  
4:00PM  ICU EEG Reading Session: Neonatal Cases  
Courtney J. Wusthoff, MD  
4:20PM  ICU EEG Reading Session: Adult Cases  
Elizabeth Gerard, MD  
4:40PM  ICU EEG Reading Session: Pediatric Cases  
Eric Payne, MD, MPH
### Program Directors Symposium

**11:00AM — 2:00PM**  
**Location:** Harris, 2nd floor  
**Chair:** Jeffrey W. Britton, MD, FACNS  

This symposium is the only one of its kind focused on Clinical Neurophysiology training programs and meets ACGME program requirement II.A.4, which advises that program directors attend one program director meeting per year. The objective of the symposium is to provide a forum for program directors to address challenges encountered in running a training program and in meeting accreditation expectations.

**Agenda:**

<table>
<thead>
<tr>
<th>Time</th>
<th>Session</th>
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</table>
| 11:00 - 11:45AM | CNP Program Milestones and Assessment Strategies  
Jeffrey Britton, MD |
| 11:45AM - 12:30PM | Quality Projects in CNP and ACNS Quality Module  
Jeffrey Britton, MD and Amy Crepeau, MD |
| 12:30 - 1:00PM | Break, Working Lunch  
1:00 - 1:30PM | Clinical Competency Committee and Program Evaluation Committee Processes  
Ruple Laughlin, MD |
| 1:30 - 2:00PM | ACGME Review Committee Q&A  
Imran Ali, MD |

### Applied Autonomic Neurophysiology

**1:00 - 2:30PM**  
**Location:** Bexar/Travis/Nueces, 2nd floor  
**Co-Chairs:** Jong Woo Lee, MD, PhD, FACNS and Claus Reinsberger MD, PhD

**Objectives:**

At the conclusion of this course, the learner should be able to:

1. Recognize the clinical features and patterns on autonomic testing in systemic and primary neurological disorders affecting central and peripheral autonomic pathways;
2. Understand an approach to the diagnostic evaluation and management of disorders of the autonomic nervous system.

**Agenda:**

<table>
<thead>
<tr>
<th>Time</th>
<th>Session</th>
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<tbody>
<tr>
<td>1:00PM</td>
<td>Introduction</td>
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</table>
| 1:05PM | Autonomic Testing  
Jeffrey Liou, MD |
| 1:35PM | Neurological Disorders with Central Autonomic Failure  
Alexandra Hovagimian, MD |
| 2:00PM | Peripheral Autonomic Failure  
Brent Goodman, MD |
| 2:25PM | Questions |

### Business in Clinical Neurophysiology

**1:00 - 5:00PM**  
**Location:** Salon C, 2nd floor  
**Co-Chairs:** Deborah Briggs, MD, FACNS and Yafa Minazad, DO, FACNS

**Objectives:**

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

1. Understand the current challenges facing neurologists/electrophysiologists employed in private practice and employed by hospital system;
2. Appreciate the changes and challenges that will be coming to how neurologists/electrophysiologists practice with the implementation of ACOs;
3. Understand the factors that need to be considered when negotiating with a hospital to bring in new technologies;
4. Know what neurologists/electrophysiologists should be focusing on when implementing EMRs, coding and billing systems given the changing field of reimbursements.

**Agenda:**

<table>
<thead>
<tr>
<th>Time</th>
<th>Session</th>
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</table>
| 1:00PM | Should I Stay or Should I Go?  
Yafa Minazad, DO, FACNS & Deborah Briggs, MD, FACNS |
| 1:50PM | Criteria for Performance Excellence — Concept for Professional & Group Practice in an ACO  
Elizabeth Mullikin, MPA, FACHE |
| 2:35PM | Break  
2:55PM | Operation Considerations in the New World  
John Vargas, MSL |
| 3:40PM | Panel Discussion  
Yafa Minazad, DO, FACNS; Deborah Briggs, MD, FACNS; Elizabeth Mullikin, MPA, FACHE; Elizabeth Delledera, MBA, MHA, BSN, RN |
| 4:25PM | Question & Answer |

### Case Studies in Peripheral Neurophysiology

**3:00 - 5:00PM**  
**Location:** Bexar/Travis/Nueces, 2nd floor  
**Chair:** Elliot Dimberg, MD

**Objectives:**

At the conclusion of the session, the learner should be able to:

1. Interpret patterns of clinical neurophysiological findings in peripheral nervous system disease;
2. Appropriately localize neuromuscular abnormalities according to the neurophysiological findings.

**Agenda:**

<table>
<thead>
<tr>
<th>Time</th>
<th>Session</th>
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</table>
| 3:00PM | Case Studies in Peripheral Neurophysiology  
Raghav Govindarajan, MD |
| 3:40PM | Case Studies in Peripheral Neurophysiology  
Randa Jarrar, MD |
| 4:20PM | Case Studies in Peripheral Neurophysiology  
Elliot Dimberg, MD |
## PROGRAM AGENDA | ANNUAL MEETING

### Annual Meeting Overview

**Friday, February 6, 2015**

<table>
<thead>
<tr>
<th>Time</th>
<th>Event</th>
<th>Location</th>
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<tbody>
<tr>
<td>7:00 - 8:00AM</td>
<td>Breakfast — Visit Exhibits and Posters</td>
<td>Liberty Hall, 1st floor</td>
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<tr>
<td>8:00 - 10:00AM</td>
<td>Concurrent Sessions:</td>
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<tr>
<td></td>
<td>SEEG Stimulation</td>
<td>Salon A, 2nd floor</td>
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<td>Beyond Seizure Detection in Critical Care EEG Monitoring</td>
<td>Salon B, 2nd floor</td>
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<td>Pelvic Floor Neuropathology (Joint ACNS/Mexican Society of Clinical</td>
<td>Salon C, 2nd floor</td>
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<td>Neurophysiology Symposium)</td>
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<tr>
<td>10:00 - 10:20AM</td>
<td>Coffee Break — Visit Exhibits and Posters</td>
<td>Liberty Hall, 1st floor</td>
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<tr>
<td>10:20 - 11:45AM</td>
<td>General Session: Presidential Lecture, Gloor Award Presentation &amp; Lecture</td>
<td>Salon B, 2nd floor</td>
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<tr>
<td>11:45AM - 1:00PM</td>
<td>Lunch — Visit Exhibits and Posters</td>
<td>Liberty Hall, 1st floor</td>
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<tr>
<td>1:00 - 3:00PM</td>
<td>Concurrent Sessions:</td>
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<tr>
<td></td>
<td>Four Ways of Looking at Seizure Networks</td>
<td>Salon A, 2nd floor</td>
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<td>Myoclonic Status Following Cardiac Arrest</td>
<td>Salon B, 2nd floor</td>
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<td>Advanced Electrodiagnostic Techniques</td>
<td>Salon C, 2nd floor</td>
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<tr>
<td>3:00 - 4:00PM</td>
<td>Coffee Break — Visit Exhibits and Poster Tours</td>
<td>Liberty Hall, 1st floor</td>
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<tr>
<td>3:00 - 4:00PM</td>
<td>Special Interest Groups</td>
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<tr>
<td></td>
<td>IOM</td>
<td>Salon A, 2nd floor</td>
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<td></td>
<td>ICU EEG: Periodic and Rhythmic Patterns</td>
<td>Salon B, 2nd floor</td>
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<td>Challenging EMG Cases</td>
<td>Salon C, 2nd floor</td>
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<tr>
<td>4:00 - 5:30PM</td>
<td>General Session: “Brain on Fire”</td>
<td>Salon B, 2nd floor</td>
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<tr>
<td>5:30 - 7:00PM</td>
<td>Neurophys Bowl</td>
<td>Salon B, 2nd floor</td>
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<tr>
<td>7:00 - 8:00PM</td>
<td>Welcome Reception</td>
<td>Liberty Hall, 1st floor</td>
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**Saturday, February 7, 2015**

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<thead>
<tr>
<th>Time</th>
<th>Event</th>
<th>Location</th>
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<tbody>
<tr>
<td>7:00 - 8:00AM</td>
<td>Breakfast — Visit Exhibits and Posters</td>
<td>Liberty Hall, 1st floor</td>
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<tr>
<td>8:00 - 9:30AM</td>
<td>Concurrent Sessions:</td>
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<tr>
<td></td>
<td>Innovative Electronic Devices in Clinical Neurophysiology</td>
<td>Salon A, 2nd floor</td>
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<td>Electroclinical Features of Autoimmune-Mediated Epilepsies</td>
<td>Salon B, 2nd floor</td>
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<td>Pedicle Screw Stimulation</td>
<td>Salon C, 2nd floor</td>
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<tr>
<td>9:30 -10:15AM</td>
<td>General Session: Jasper Award Presentation &amp; Lecture</td>
<td>Salon B, 2nd floor</td>
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<tr>
<td>10:15 -10:30AM</td>
<td>Coffee Break — Visit Exhibits and Posters</td>
<td>Liberty Hall, 1st floor</td>
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<tr>
<td>10:30 -11:15AM</td>
<td>General Session: Schwab Award Presentation &amp; Lecture</td>
<td>Salon B, 2nd floor</td>
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<tr>
<td>11:15AM -12:45PM</td>
<td>Concurrent Sessions:</td>
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<tr>
<td></td>
<td>Peripheral Nerve Injury and Evaluation</td>
<td>Salon A, 2nd floor</td>
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<td>Probing Cortical Physiology: The Use of TMS-EEG in Epilepsy and Psychiatry</td>
<td>Salon B, 2nd floor</td>
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<td>Epilepsy Case Study in Video EEG Monitoring (Joint ACNS/Canadian Society of Clinical Neurophysiologists Symposium)</td>
<td>Salon C, 2nd floor</td>
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<tr>
<td>12:45-2:00PM</td>
<td>Lunch — Visit Exhibits and Poster Tours</td>
<td>Liberty Hall, 1st floor</td>
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<tr>
<td>2:00-3:30PM</td>
<td>Concurrent Sessions:</td>
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<tr>
<td></td>
<td>Advanced Artifactology</td>
<td>Salon A, 2nd floor</td>
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<td>EEG — fMRI in Epilepsy (Joint ACNS/Brazilian Society of Clinical Neurophysiology Symposium)</td>
<td>Salon B, 2nd floor</td>
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<td>Evidence, Ethics and Epiphany in NIOM</td>
<td>Salon C, 2nd floor</td>
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<td>3:30 - 4:30PM</td>
<td>Special Interest Groups</td>
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<td>Economics of MEG: How to Keep a MEG Center Afloat?</td>
<td>Salon A, 2nd floor</td>
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<td>Intracranial EEG</td>
<td>Salon B, 2nd floor</td>
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<td>Neonatal and Pediatric EEG Patterns That May be Clues to the Specific Diagnosis: Case Examples and Discussion</td>
<td>Salon C, 2nd floor</td>
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### Saturday, February 7, 2015, continued

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<tr>
<th>Time</th>
<th>Event</th>
<th>Location</th>
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<tbody>
<tr>
<td>4:30 - 6:00PM</td>
<td>Concurrent Sessions:</td>
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<td></td>
<td>Reflex Epilepsy: How to Define and Determine It</td>
<td>Salon A, 2nd floor</td>
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<td>Electrocardiography Overview and Future Directions</td>
<td>Salon B, 2nd floor</td>
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<td>Autonomic Function (Joint ACNS/IFCN Latin American Chapter Symposium)</td>
<td>Salon C, 2nd floor</td>
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<td>6:00 - 7:00PM</td>
<td>Research Highlights</td>
<td>Salon B, 2nd floor</td>
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<tr>
<td>7:00 – 7:30PM</td>
<td>Annual Business Meeting</td>
<td>Salon B, 2nd floor</td>
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### Sunday, February 8, 2015

<table>
<thead>
<tr>
<th>Time</th>
<th>Event</th>
<th>Location</th>
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<tbody>
<tr>
<td>7:00 - 8:00AM</td>
<td>Breakfast</td>
<td>Salon B Foyer, 2nd floor</td>
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<tr>
<td>8:00 - 9:30AM</td>
<td>Concurrent Sessions:</td>
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<td></td>
<td>Intraoperative Communication Strategies: Case Workshop</td>
<td>Salon A, 2nd floor</td>
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<td>Skills Workshop: How to Record and Analyze Wide-Band EEG in Clinical Epilepsy: Slow Shifts and HFO</td>
<td>Salon B, 2nd floor</td>
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<td>Minimally Invasive Epilepsy Surgery: Case Based Discussion on the Role of Laser Ablation</td>
<td>Salon C, 2nd floor</td>
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<tr>
<td>9:30 – 10:00AM</td>
<td>Coffee Break</td>
<td>Salon B Foyer, 2nd floor</td>
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<tr>
<td>10:00 - 11:30AM</td>
<td>Concurrent Sessions:</td>
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<td></td>
<td>Clinical Neurophysiology Trials and Tribulations in the ICU</td>
<td>Salon A, 2nd floor</td>
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<td>A Practical Approach to Stereo EEG for Different Types of Focal Epilepsy</td>
<td>Salon B, 2nd floor</td>
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<td>When Surgery is not an Option: Neurostimulation and Beyond</td>
<td>Salon C, 2nd floor</td>
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<td>11:30AM</td>
<td>Adjourn</td>
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**PROGRAM AGENDA | ANNUAL MEETING**

**Friday, February 6, 2015**

**BREAKFAST – Visit Exhibits & Posters**
7:00 – 8:00AM  
Location: Liberty Hall, 1st floor

**CONCURRENT SESSIONS**
8:00 - 10:00AM

**SEEG Stimulation**  
Location: Salon A, 2nd floor  
Chair: Norman So, MD, FACNS

**Objectives:**
At the conclusion of this activity, participants will be able to:
1. Describe the potential and limitation of electrical stimulation to reproduce seizure symptoms and signs;
2. Describe techniques and paradigms to localize motor, sensory, and other functions;
3. Recognize language networks in the brain;
4. Review how CCEPs can elucidate cerebral connectivity.

**Agenda:**
- 8:00AM Stimulation for Auras and Seizures  
  Patrick Chauvel, MD
- 8:30AM Stimulation Mapping of Non-language Functions  
  Juan Bulacio, MD
- 9:00AM Stimulation mapping of Language  
  Agnes Trebuchon, MD, PhD
- 9:30AM Cortico-Cortical Evoked Potentials  
  Dileep Nair, MD

**Beyond Seizure Detection in Critical Care EEG Monitoring: Background Matters**  
Location: Salon B, 2nd floor  
Chair: Nicolas Gaspard, MD, PhD

**Objectives:**
At the conclusion of this activity, participants will be able to:
1. Describe the EEG and quantitative EEG changes encountered in patients with sepsis, and relate these changes to clinical neurological function and outcome;
2. Discuss the physiological mechanisms of EEG reactivity and variability, how they can be quantified and how this quantification may assist in the assessment and prognostication of brain dysfunction and/or injury;
3. Describe the EEG signatures of consciousness and sedation, and how these vary depending on sedative pharmacology and in the setting of medical comorbidities and encephalopathy;
4. Discuss the assessment of sleep in the ICU setting, and the prevalence and risk factors of sleep disruption in critically ill patients.

**Agenda:**
- 8:00AM EEG Reactivity and Variability: Standardization, Quantification and Significance  
  Nicolas Gaspard, MD, PhD
- 8:40AM Continuous EEG for Monitoring of Pain Control and Sedation  
  M. Brandon Westover, MD, PhD
- 9:20AM Delirium and Encephalopathy in Critical Patients: Quantitative EEG Correlates and Role of Sleep Disturbances  
  Emily J. Gilmore, MD

**Sociedad Mexicana de Neurofisiología Clínica A.C**

**Pelvic Floor Neurophysiology**  
Joint ACNS/Mexican Society of Clinical Neurophysiology (SMNFC) Symposium  
Location: Salon C, 2nd floor  
Co-Chairs: Claudia Esther Paz Navarro, MD and Armando Valdes-Tello, MD

**Objectives:**
At the conclusion of this activity, participants will be able to:
1. Understand the general neuroanatomy of the pelvic floor;
2. Describe the utility of evoked potential testing in assessing pelvic floor neurophysiology;
3. Describe the EMG and nerve conduction findings in pelvic floor disorders;
4. Understand how IONM can alter surgical management and help preserve urinary and anorectal function in pelvic floor surgery.

**Agenda:**
- 8:00AM Introduction & Anatomy: Autonomic and Somatic Sacral Reflexes  
  Jaime Ramos Peak, MD
- 8:35AM MEPs of the Pelvic Floor Muscles, Pudendal SEPs: EMG of Anal and Urethral Sphincters, Pudendal Nerve Terminal Motor Latencies  
  Armando Valdes-Tello, MD
- 9:10AM Intraoperative Neurophysiological Monitoring in Pelvic Floor Surgery  
  Guillermo Martin Palomaque, MD
- 9:45AM Questions

**COFFEE BREAK – Visit Exhibits & Posters**
10:00 – 10:20AM  
Location: Liberty Hall, 1st floor

**GENERAL SESSION: Presidential Address, Gloor Award Presentation & Lecture**
10:20 – 11:45AM  
Location: Salon B, 2nd floor  
Co-Chairs: William O. Tatum, DO, FACNS and Jaime R. López, MD, FACNS

- 10:20AM Presidential Lecture  
  “Clinical Neurophysiology in Treatment of Disease”  
  Aatif M. Husain, MD, FACNS
- 11:00AM Gloor Award Presentation & Lecture  
  “Cortical Activities Associated with Voluntary and Involuntary Movements”  
  Hiroshi Shibasaki, MD, PhD, FACNS
### Program Agenda | Annual Meeting

**Friday, February 6, 2015, continued**

**LUNCH — Visit Exhibits & Posters**
11:45AM — 1:00PM
Location: Liberty Hall, 1st floor

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**CONCURRENT SESSIONS**

1:00 - 3:00PM

**Four Ways of Looking at Seizure Networks: A Critical Discussion**
Location: Salon A, 2nd floor
Chair: Charles Epstein, MD, FACNS

**Objectives:**
At the conclusion of this session, participants should be able to:

1. Describe possible advantages of causal/directed methods in analyzing seizure propagation;
2. Describe the features that may help distinguish between the seizure focus and the penumbra;
3. Describe the nature of the resting-state network and its possible interactions with seizure propagation;
4. Describe some of the near and remote anatomic changes associated with focal epilepsy.

**Agenda:**

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<th>Time</th>
<th>Topic</th>
<th>Speaker(s)</th>
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<tbody>
<tr>
<td>1:00PM</td>
<td>High-Frequency Granger Causality in the Analysis of Preictal Networks</td>
<td>Charles M. Epstein, MD, FACNS</td>
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<tr>
<td>1:30PM</td>
<td>Implications of Long-Range Effects of Seizures for Functional Network Theory</td>
<td>Ronald Emerson, MD, FACNS</td>
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<tr>
<td>2:00PM</td>
<td>Resting State Connectivity and Focal Epilepsy</td>
<td>Sarah K. Bandt, MD</td>
</tr>
<tr>
<td>2:30PM</td>
<td>Neural Network Architecture and the Clinical Course of Temporal Lobe Epilepsy</td>
<td>Leonardo Bonilha, MD, PhD</td>
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**Myoclonic Status Following Cardiac Arrest**
Location: Salon B, 2nd floor
Co-Chairs: Stephan U. Schuele, MD, MPH, FACNS and Jong Woo Lee, MD, PhD

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

1. Discuss the available literature on myoclonic status definition and prognosis and its limitations;
2. Understand the arguments for and against aggressive treatment of myoclonic status;
3. Apply this information and expert opinion to clinical cases.

**Agenda:**

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<th>Time</th>
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<tr>
<td>1:00PM</td>
<td>Introduction</td>
<td>Stephan U. Schuele, MD, MPH, FACNS</td>
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<tr>
<td>1:05PM</td>
<td>Definitions and Questions</td>
<td>Elizabeth Gerard, MD</td>
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**Advanced Electrodagnostic Techniques**
Location: Salon C, 2nd floor
Chair: Ioannis Karakis, MD, MSc

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

1. Understand the use of advanced electrodagnostic techniques and their potential role for the diagnosis, prognosis and monitoring of treatment in neuromuscular medicine.

**Agenda:**

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<tr>
<th>Time</th>
<th>Topic</th>
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<tr>
<td>1:00PM</td>
<td>Quantitative Electromyography</td>
<td>Paul E. Barkhaus, MD</td>
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<td>1:30PM</td>
<td>Macro Electromyography</td>
<td>Joe Jabre, MD</td>
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<td>2:00PM</td>
<td>Motor Unit Number Estimation</td>
<td>Ioannis Karakis, MD, MSc</td>
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<td>2:30PM</td>
<td>Electrical Impedance Myography</td>
<td>Seward Rutkove, MD</td>
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**COFFEE BREAK — Visit Exhibits & Poster Tours**
3:00 – 4:00PM
Location: Liberty Hall, 1st floor
## SPECIAL INTEREST GROUPS

### 3:00 - 4:00PM

**IOM**
Location: Salon A, 2nd floor  
Chair: Jaime R. López, MD, FACNS

**Objectives:**
At the conclusion of this activity, participants will be able to:
1. Describe the use of an infrared camera as an adjunct in facial nerve monitoring.
2. Understand the importance of an NIOM article review group in a medical society.
3. Promote discussion of important issues in NIOM.

**Agenda:**
- 3:00PM  Introduction  
  Jaime R. López, MD, FACNS  
- 3:05PM  Use of an Infrared Camera in Facial Nerve Monitoring  
  Emily K. Murphy, BA  
- 3:30PM  Establishing an NIOM Article Review Group  
  Matthew Eccher, MD, MS  
- 3:45PM  Open Forum Discussion

**ICU EEG: Periodic and Rhythmic Patterns**
Location: Salon B, 2nd floor  
Co-Chairs: Elizabeth Gerhard, MD and Nicholas Abend, MD

**Objectives:**
At the conclusion of this activity, 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:**
- 3:00PM  GPDs  
  Brandon Foreman, MD  
- 3:00PM  LPDs  
  Indranil Sen-Gupta, MD  
- 3:00PM  LRDA  
  Nicolas Gaspard, MD, PhD  
- 3:00PM  Periodic and Rhythmic Patterns in Pediatric Patients  
  Josh Goldstein, MD  
- 3:40PM  Discussion

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**Challenging EMG Cases**
Location: Salon C, 2nd floor  
Chair: Mark Ross, MD, FACNS

**Objectives:**
At the conclusion of this activity, 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:**
- 3:00PM  Utility of Neuromuscular Ultrasound to Improve Diagnostic Testing in Neuromuscular Disorders  
  Mark Ross MD, FACNS  
- 3:20PM  Clinical Presentation of Unusual Neuromuscular Disorders  
  Francis O. Walker, MD, FACNS  
- 3:40PM  Application of Advanced Electrodiagnostic Testing in Diagnoses of Neuromuscular Disorders  
  Erik Ortega, MD

**GENERAL SESSION: “Brain on Fire”**
4:00 – 5:30PM  
Location: Salon B, 2nd floor  
Chair: William B. Gallentine, DO, FACNS

- 4:00PM  Young Investigator Travel Award Ceremony
- 4:30PM  Brain on Fire: My Month of Madness  
  Susannah Cahalan
- 5:00PM  Clinical Features and Pathophysiologic Mechanisms of Autoimmune-Mediated Epilepsies  
  Souhel Najjar, MD

**NEUROPHYS BOWL**

5:30 – 7:00PM  
Location: Salon B, 2nd floor  
Co-Chairs: Susan T. Herman MD, FACNS and Sarah Schmitt, MD

**WELCOME RECEPTION**
7:00 – 8:00PM  
Location: Liberty Hall, 1st floor
CONCURRENT SESSIONS
8:00 - 9:30 AM

Innovative Electronic Devices in Clinical Neurophysiology
Location: Salon A, 2nd floor
Chair: Madeleine Grigg-Damberger, MD, FACNS

Objectives:
At the conclusion of this activity, participants should be able to:
1. Name the latest gizmos, gadgets, advanced and/or cutting edge electronic devices and technologies proposed and used by clinical neurophysiologists;
2. Identify the indications, patient populations, limitations, and/or flaws of these devices;
3. Decide whether to incorporate some of these new technologies into your clinical practice to improve the value and quality of care provided to your patients.

Agenda:
8:00 AM  Cutting Edge Technologies and Electronic Devices for Diagnosing and Managing People with Epilepsy
Steven Schachter, MD, FACNS
8:30 AM  Latest Developments in Non-epileptic Brain Stimulation and Quantitative EEG
Charles M. Epstein, MD, FACNS
9:00 AM  Gizmos, Gadgets and Electronic Devices for Diagnosing and/or Treating Patients with Stroke, Dementia, and Brain/Spinal Cord Injury
Madeleine M. Grigg-Damberger, MD, FACNS

Electroclinical Features of Autoimmune-Mediated Epilepsies: Case Discussions
Location: Salon B, 2nd floor
Chair: William B. Gallentine, DO, FACNS

Objectives:
At the conclusion of this activity, participants should be able to:
1. Recognize the clinical features of autoimmune-mediated epilepsy syndromes and related disorders;
2. Describe continuous video EEG features typical of autoimmune-mediated epilepsies, as well as recognize nonepileptic spells often associated with these syndromes;
3. Perform an appropriate diagnostic evaluation for a suspected autoimmune-mediated epilepsy;
4. Discuss potential immunomodulatory treatment options.

Agenda:
8:00 AM  NMDA Receptor Antibody Encephalitis
Sarah Schmitt, MD
8:30 AM  LGI1 Antibody Related Epilepsy, Faciobrachial Dystonic Seizures
Ji Yeoun Yoo, MD
9:00 AM  Anti-GAD Antibody and Other Autoimmune-Mediated Epilepsies
William B. Gallentine, DO, FACNS

Pedicle Screw Stimulation - A Discussion by Some Experts
Location: Salon C, 2nd floor
Chair: David Gloss, MD

Objectives:
At the end of the activity, the learners will be able to:
1. Identify common situations where pedicle screw stimulation might be used, and the range of its use.

Agenda:
8:00 AM  Pedicle Screw Stimulation: A Discussion with Some Experts
David Gloss, MD
8:30 AM  Technical Considerations for Pedicle Screw Testing
Alan D. Legatt, MD, PhD, FACNS
8:50 AM  Pedicle Screw Cases
Cormac O’Donovan, MD, FACNS
9:20 AM  Pedicle Screw Cases
Christine Hung, MD
9:20 AM  Questions/Panel

GENERAL SESSION: Jasper Award Presentation & Lecture
9:30 – 10:15 AM
Location: Salon B, 2nd floor
Chair: William O. Tatum, DO, FACNS
“A Personal Perspective on 50 Years of Clinical Neurophysiology”
John Ebersole, MD, FACNS

COFFEE BREAK – Visit Exhibits & Posters
10:15 – 10:30 AM
Location: Liberty Hall, 1st floor

GENERAL SESSION: Schwab Award Presentation & Lecture
10:30 – 11:15 AM
Location: Salon B, 2nd floor
Chair: William O. Tatum, DO, FACNS
“Fasciculation Potentials: The Enigma”
Mamede de Carvalho, MD
Saturday, February 7, 2015, continued

**Peripheral Nerve Injury and Evaluation**
Location: Salon A, 2nd floor  
Chair: Mark Hallett, MD, FACNS

**Objectives:**
At the conclusion of this activity, participants will be able to:
1. Improve participant’s ability to identify and verify peripheral nerve injury using state of the art methods.

**Agenda:**
11:15AM  Nerve Injuries in Sports  
   Mark Hallett, MD, FACNS  
11:45AM  Electrodiagnosis of Peripheral Nerve Injury  
   Jun Kimura, MD  
12:15PM  Use of Ultrasound to Evaluate Peripheral Nerve Injuries  
   Francis O. Walker, MD, FACNS

**Probing Cortical Physiology: The Use of TMS-EEG in Epilepsy and Psychiatry**
Location: Salon B, 2nd floor  
Chair: Bernard Chang, MD

**Objectives:**
At the conclusion of this activity, participants should be able to:
1. Understand the utility of transcranial magnetic stimulation (TMS) with simultaneous EEG recording as a tool that allows for the determination of cortical inhibition/excitation and plasticity;
2. Describe how TMS-EEG has been used, in both humans and rodent model examples, to identify focal cortical hyperexcitability in the brains of those with epilepsy from various etiologies;
3. Explain the key alterations in cortical physiology from studies of TMS-EEG in psychiatric disorders such as schizophrenia.

**Agenda:**
11:15AM  Brain Stimulation and Cortical Physiology in Patients with Epilepsy  
   Mouhsin Shafi, MD, PhD  
11:45AM  Translational Applications of TMS in Animal Models of Epilepsy and Brain Injury  
   Alexander Rotenberg, MD, PhD  
12:15PM  TMS-EEG Studies in Patients with Schizophrenia and Other Psychiatric Disorders  
   Zafiris J. Daskalakis, MD, PhD

**Epilepsy Case Study in Video-EEG Monitoring**
Joint ACNS/Canadian Society of Clinical Neurophysiologists Symposium
Location: Salon C, 2nd floor  
Chair: Seyed Mirsattari, MD

**Objectives:**
At the conclusion of this activity, participants will be able to:
1. Identify semiology of some epileptic seizures;  
2. Make a correlation between clinical features and anatomical localizations of epileptic seizures;  
3. Identify interictal and ictal EEG patterns in the presented cases;  
4. Make an appropriate differential diagnosis for each case;  
5. Provide a treatment plan.

**Agenda:**
11:15AM  Case Discussion 1  
   Richard McLachlan, MD  
11:45AM  Case Discussion 2  
   Dang Nguyen, MD  
12:15PM  Case Discussion 3  
   Seyed Mirsattari, MD

**LUNCH — Visit Exhibits & Posters Tours**
1:00 — 2:00PM  
Location: Liberty Hall, 1st floor

**Concurrent Sessions**
2:00PM - 3:30PM

**Advancing Artifactology**
Location: Salon A, 2nd floor  
Chair: William O. Tatum, DO, FACNS

**Objectives:**
At the conclusion of this activity, participants should be able to:
1. Describe common artifacts arising during EEG, EMG, Sleep and NIOM procedures;  
2. Explain the clinical treatment implications involved in failed recognition in both central and peripheral clinical neurophysiology;  
3. Evaluate the presence of specific artifacts based on the concepts of waveform temporal and spatial characteristics that are procedure-specific.
## Evidence, Ethics, and Epiphany in NIOM

**Location:** Salon C, 2nd floor  
**Chair:** Stanley S. Skinner, MD, FACNS

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

1. Understand the possibility and utility of led trials in NIOM;  
2. The use of cost/benefit analysis in NIOM;  
3. How the link between reversed signal change and causation suggests prevention;  
4. How mechanistic analysis, the dramatic effect (epiphany), and serendipity provide evidence in medicine and NIOM;  
5. Articulate and defend diverse means to secure evidence proofs beyond the so-called evidence hierarchy.

**Agenda:**

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<th>Time</th>
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<tr>
<td>2:00PM</td>
<td>Evaluating Costs and Outcomes for IOM Using Decision Modeling</td>
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<td>John P. Ney, MD, MPH</td>
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<td>2:25PM</td>
<td>Somatosensory and Motor Evoked Potential Biomarkers as Surrogate</td>
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<td>Endpoints During Surgery</td>
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<td>Robert N. Holdefer, MD</td>
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<td>2:55PM</td>
<td>Controlled (and Ethically Permissible) Outcome Studies in IOM</td>
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<td>Stanley S. Skinner, MD, FACNS</td>
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<td>3:20PM</td>
<td>Panel Q&amp;A</td>
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## SPECIAL INTEREST GROUPS

### Economics of MEG: How to Keep a MEG Center Afloat?

**Location:** Salon A, 2nd floor  
**Chair:** Anto Bagic, MD, PhD, FACNS

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

1. Recognize the key practical aspects of MEG economics;  
2. Explain the specific role of TeleMEG as an opportunity for buying expertise where available and using it where needed;  
3. Understand the potential of MEG research for improving economic viability of MEG centers in the complex and worsening healthcare reality.

**Agenda:**

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<th>Time</th>
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<tr>
<td>3:30PM</td>
<td>A Perfect Real World Scenario</td>
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<td>Michael Funke, MD, PhD</td>
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<td>3:50PM</td>
<td>Buying Expertise Where Available to Use Where Needed: TeleMEG</td>
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<td>Robert Knowlton, MD</td>
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<td>4:10PM</td>
<td>Can Research be a MEG’s Life Jacket?</td>
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<td>Jeffrey Lewine, MD</td>
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Saturday, February 7, 2015, continued

Intracranial EEG
Location: Salon B, 2nd floor
Co-Chairs: Gregory Worrell, MD & Rafeed Alkawadri, MD

Objectives:
1. Evaluate patients with TLE and normal MRI;
2. Understand the relative benefits & limitations of subdural, depth, and SEEG approach.

Agenda:
3:30PM Overview of Nonlesional TLE Outcomes
   Gregory Worrell, MD
3:40PM Invasive Monitoring Approaches
   Nitin Tandon, MD
4:00PM Analysis Techniques for Localizations
   Rafeed Alkawadri, MD
4:20PM Wrap Up & Recommendations for Nonlesional TLE

Neonatal and Pediatric EEG Patterns that may be Clues to the Specific Diagnosis: Case Examples and Discussion
Location: Salon C, 2nd floor
Co-Chairs: Tobias Loddenkemper, MD, FACNS & Heather Olson, MD

Faculty:
KCNQ2 Neonatal Encephalopathy
Eli M. Mizrahi, MD

Case: Prominent Fast Activity and Non-epileptic Events — a Zebra and a Tiger?
Heather Olson, MD

Case: Rhythmic High Amplitude Delta with Superimposed Spikes (RHADS)
Christelle Achkar, MD

Case Presentation
Takijah Heard, MD

CONCURRENT SESSIONS
4:30 - 6:00PM

Reflex Epilepsy: How to Define and Determine It
Location: Salon A, 2nd floor
Chair: Dorothee G. Kasteleijn-Nolst Trenite, MD, PhD, MPH

Objectives:
1. Present interesting clinical cases with EEG-video material; and
2. Discuss the concept of reflex epilepsy.

Agenda:
4:30PM Introduction
4:30PM Concepts of Reflex Epilepsy
   Howard Ring, MD
5:00PM Clinical Practice and EEG Diagnostic Tools
   Dorothee G. Kasteleijn-Nolst Trenite, MD, PhD, MPH
5:40PM Diagnosing Cognition-Induced Epilepsies
   Anthony L. Ritaccio, MD

Electrocorticography: Overview and Future Directions
Location: Salon B, 2nd floor
Chair: Daniela Minecan, MD

Objectives:
1. Describe the principles of electrocorticography;
2. Identify and interpret patterns, learn to differentiate between ictal, interictal, normal, abnormal;
3. Evaluate the benefits and understand its limitation; and
4. Describe how to create the framework to conduct clinical research and incorporate recent advances.

Agenda:
4:30PM Electrocorticography: How to Improve Signal Acquisition, Signal Analysis and Interpretation
   Brian Litt, MD
5:00PM Electrocorticography and Functional Mapping: Current and Future
   Josef Parvizi, MD, PhD
5:30PM Electrocorticography: Physiology, Methodologies and Anesthetic Considerations
   Rafeed Alkawadri, MD
Autonomic Function
Joint ACNS/International Federation of Clinical Neurophysiology (IFCN) – Latin American Chapter Symposium
Location: Salon C, 2nd floor
Chair: Marcondes Franca, MD, PhD

Objectives:
At the conclusion of this activity, participants should be able to:
1. Understand the anatomical and physiological bases of autonomic tests;
2. Learn how to interpret the results of the autonomic tests;
3. Identify and evaluate autonomic manifestations related to peripheral neuropathies;
4. Identify and evaluate autonomic manifestations related to neurodegenerative diseases.

Agenda:
4:30PM  Neurophysiological Investigation of the Autonomic Nervous System
        Renato J. Verdugo, MD
5:00PM  Autonomic Dysfunction in Disorders of the Peripheral Nervous System
        Arturo A. Leis, MD
5:30PM  Autonomic Dysfunction in Neurodegenerative Diseases
        Marcondes Franca, MD, PhD

RESEARCH HIGHLIGHTS
6:00 – 7:00PM
Location: Salon B, 2nd floor
Co-Chairs: Lawrence J. Hirsch, MD, FACNS and Jonathan Halford, MD

This session features four of the highest-scoring poster abstract submissions, selected by the Clinical Research Committee for podium presentation. The poster numbers are noted below and the full abstract of each presentation can be found on pages##-##.

Agenda:
6:00PM  Diffuse Optical Monitoring of Spinal Cord Blood Flow and Oxygenation (S48)
         Thomas Floyd, MD
6:15PM  Low and High Frequency Oscillations Reveal Distinct Absence Seizure Networks (F40)
         Jeffery Tenney, MD
6:30PM  The Time Course and Prognostic Values of Electroencephalographic Patterns after Anoxic Brain Injury (S8)
         Adithya Sivaraju, MD
6:45PM  Clinical Performance of a Prospective Continuous Electroencephalography (cEEG) Ischemia Monitoring Service for Predicting Neurologic Decline after Aneurysmal Subarachnoid Hemorrhage (SAH) (S7)
         Eric S. Rosenthal, MD

Business Meeting
7:00 – 7:30PM
Location: Salon B, 2nd floor

This meeting is open to all attendees, but only ACNS members may vote.
Professional Development Mentoring Breakfast
Location: Harris, 2nd floor

ACNS PROFESSIONAL DEVELOPMENT MENTORING PROGRAM
Sunday, February 8, 2015  (subject to change)
7:00 – 8:00AM Harris; 2nd Floor

Once again, the Annual Meeting will include a professional development session for clinical neurophysiologist. If you have signed up to be a mentor or mentee, please join us on Sunday, February 8 from 7:00 – 8:00AM in Harris on the 2nd floor!

CONCURRENT SESSIONS
8:00 - 9:30AM

Intraoperative Communication Strategies: Case Workshop
Location: Salon A, 2nd floor
Co-Chairs: Viet Nguyen, MD and Jaime R. López, MD, FACNS

Objectives:
At the conclusion of this activity, participants will be able to:
1. Identify strategies for effective communication for different types of surgical procedures and surgical styles;
2. List common pitfalls in operating room communication;
3. Explain the technologist’s perspective on proper supervision, reporting of critical changes, and proper documentation;
4. Discuss the surgeon’s perspective on clinically relevant findings and implications for surgical management.

Agenda:
8:00AM Overview & Peripheral Nerve Cases
Viet Nguyen, MD
8:20AM Spinal Cases
Elayna Rubens, MD
8:40AM Technologist’s Perspective & Intracranial Cases
Eric Jones, BS, CNIM, REEGT, RTLM
9:00AM Surgeon’s Perspective
Laurence McKinley, MD
9:20AM Q&A Discussion

Skills Workshop: How to Record and Analyze Wide-Band EEG in Clinical Epilepsy: Slow Shifts and HFO
Location: Salon B, 2nd floor
Chair: Akio Ikeda, MD, PhD

Objectives:
At the conclusion of this activity, participants should be able to:
1. Understand clinical application and practical procedures of how to record and analyze Wide-Band EEG that will be presented as the skills workshop.

Agenda:
8:00AM Epileptic Slow Shifts: Neocortical Epilepsy
Akio Ikeda, MD, PhD
8:20AM Epileptic Slow Shifts: Limbic Epilepsy
Pradeep Modur, MD
8:40AM High Frequency Oscillation (HF) in Childhood Epilepsy
Hiroshi Otsubo, MD
9:00AM High Frequency Oscillation (HFO) in Adult Epilepsy
Jean Gotman, PhD
9:20AM Discussion

Minimally Invasive Epilepsy Surgery: Case Based Discussion on the Role of Laser Ablation
Location: Salon C, 2nd floor
Chair: Heidi Munger Clary, MD, MPH

Objectives:
At the conclusion of this session, participants should be able to:
1. Describe 3 categories of refractory epilepsy patients who are potential candidates for laser ablation therapy;
2. Understand the basic surgical technique of laser ablation for treatment of refractory epilepsy;
3. Describe early outcomes of the laser ablation epilepsy surgery technique from the two largest, single-center case series available to date.

Agenda:
8:00AM Laser Ablation Treatment of Refractory Epilepsy: Surgical Technique and the Emory Experience
Robert Gross, MD, PhD
8:30AM Laser Ablation Treatment of Refractory Epilepsy: Role in Pediatric Epilepsy and the Wake Forest Experience
Gautam Popli, MBBS, MD
9:00AM Interactive Case Discussion

COFFEE BREAK
9:30 – 10:00AM
Location: Salon B Foyer, 2nd Floor
CONCURRENT SESSIONS
10:00 - 11:30AM

Clinical Neurophysiology Trials and Tribulations in the ICU
Location: Salon A, 2nd floor
Chair: Jong Woong Lee, MD, PhD, FACNS and Cecil Hahn, MD, MPH, FACNS

Objectives:
At the conclusion of this activity, participants will be able to:
1. Understand the steps required to successfully design, implement, and complete a clinical trial involving a clinical neurophysiology endpoint in critically ill patients;
2. Formulate the most important clinical questions that can be answered using a trial;
3. Explain the current state of clinical neurophysiology trials in critically newborns, children and adults.

Agenda:
10:00AM  Introduction
10:05AM  From Conception to Implementation: TRENDS
        Aatif M. Husain, MD, FACNS
10:25AM  TELSTAR: Treatment of Electroencephalographic Status Epilepticus after Cardiopulmonary Resuscitation
        Michel J. van Putten, MD MSc PhD
10:45AM  The Boston Bumetanide Trial for Neonatal Seizures
        Janet Soul, MD
11:05AM  Allopregnanolone as Adjunctive Therapy in Super-Refractory Status Epilepticus
        Henrikas Vaitkevicius, MD
11:25AM  What’s Next in Clinical Neurophysiology Trials?
        Cecil H. Hahn, MD, MPH, FACNS

A Practical Approach to Stereo EEG for Different Types of Focal Epilepsy
Location: Salon B, 2nd floor
Chair: Stephan U. Schuele, MD, MPH, FACNS

Objectives:
At the conclusion of the activity, participants should be able to:
1. Understand the principles of Stereo EEG;
2. Discuss implantation strategies with SEEG for various types of focal epilepsies;
3. Actively participate in the selection of electrode placement, discussion of safety aspects and surgical challenges, and the reading and resection planning of patients who are candidates for SEEG.

Agenda:
10:00AM  Introduction
10:00AM  Temporal and Temporal Lobe Plus Epilepsy
        Sam Lhatoo, MD
10:30AM  Frontal Lobe Epilepsy
        Juan Bulacio, MD
11:00AM  Case Presentation and Discussion
        Stephan U. Schuele, MD, MPH, FACNS

When Surgery is Not an Option: Neurostimulation and Beyond
Location: Salon C, 2nd floor
Chair: Dawn Eliashiv, MD, FACNS

Objectives:
At the conclusion of this activity, participants will be able to:
1. Identify patients with medically refractory epilepsy that may be candidates for Neurostimulation or MRI guided laser ablation;
2. Determine which technique is most appropriate for patients with medically refractory focal epilepsy that are not candidates for standard respective epilepsy surgery; and
3. Develop clinical skills to program RNS.

Agenda:
10:00AM  Eloquent Cortex, Bilateral Seizures, Difficult Approach; Evolving Approaches for Refractory Epilepsy
        Dawn Eliashiv, MD, FACNS
10:30AM  Future Directions in Neurostimulation
        Chris Digiorgio, MD
11:00AM  RNS: An Update
        Martha Morell, MD

ADJOURN
11:30AM
**SEP Monitoring**  
*Andres A. Gonzalez, MD, MMM*

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 or traction on the eighth nerve.

**BAEP Monitoring**  
*Alan D. Legatt, MD, PhD, FACNS*

BAEP Monitoring

**MEP Monitoring**  
*Ronald Emerson, MD, FACS*

This presentation will review the physiology of transcranial electrical motor evoked potentials, the effects of anesthetic agents and the methodology for intraoperative monitoring. Interpretative criteria will be discussed, and example cases will be presented.

**EEG and Doppler Ultrasound Monitoring**  
*Michael McGarvey, MD, FACS*

Intraoperative EEG and Transcranial Doppler Ultrasound Monitoring. This lecture details the use of intraoperative EEG and Transcranial Doppler in intraoperative monitoring. The methods are clearly distinct but are both used to monitor vascular procedures that place the brain at risk. The lecture details the specific techniques, why they are useful, and in what procedures they are best employed in for intraoperative monitoring.

**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.

**Monitoring of Spinal Nerve Roots**  
*Monica Islam, MD*

Peripheral nerve injuries are common though the degree of injury varies widely. Diagnosis of the spectrum of injuries to peripheral nerve is difficult to achieve acutely. Routine outpatient EMGs provide some information though it is very limited. During exploratory surgery of a peripheral nerve injury, it is easy to perform stimulation and recording that provides additional information to the surgical team concerning the status of the injured nerve at the time of surgery. This information facilitates difficult decisions that the surgeon must make as to how best to repair an injury. The session on Operative Nerve Recordings relates the methodology and the underlying rationale that provides this information. Both technical aspects and EMG principles relating to nerve recordings will be considered in this presentation.
SPEAKER ABSTRACTS

Tuesday, February 3, 2015, continued

Anesthetic Management in IOM
David Ferson, MD

In the 21st century, the monitoring of neurological functions during operations involving the nervous system represents a standard of practice. Several new neurological monitoring modalities have been introduced to improve the safety and accuracy of neurosurgery. These monitoring modalities require a deep understanding of neuroanatomy and neurophysiology. Also, neuromonitoring is best achieved through team work between all services involved in the operating room or in the intensive care unit. This is because of the potential effects of some physiological parameters (e.g., blood pressure, oxygenation, and temperature) and how different pharmacological agents (e.g., anesthetics, sedatives, and hypnotics) may affect the recording and interpretation of signals during neuromonitoring. New developments in neuroimaging techniques, such as functional magnetic resonance imaging (fMRI), tractography, and spectroscopy, offer some exciting opportunities. Modern neurosurgical suites also contain intraoperative MRIs that allow the surgeons to determine the accuracy and degree of resection in real time. Unfortunately, the MRI environment poses some challenges with regards to equipment and techniques for neuromonitoring. These challenges, however, frequently turn into opportunities for research and clinical care with regards to the correlation of surgical navigation systems with intraoperative mapping and monitoring of neurological functions that has not been available before.

Wednesday, February 4, 2015

EP Reading Session
(7:00 – 8:30AM)

Brainstem Auditory Evoked Potentials (BAEPs)
Alan D. Legatt, MD, PhD, FACNS

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 interpeak interval measurements derived from the absolute latencies, and on right-left differences of these absolute latencies and of the interpeak intervals. Examination of absolute component amplitudes is not useful because of marked amplitude variability across subjects, but examination of the IV-V:1 amplitude ratio may detect some abnormalities that are not detected by examination of latencies alone.

Introduction to Stereo-EEG
(7:00 – 8:30AM)

Patient and Electrode Selection
Stephan U. Schuele, MD, MPH, FACNS

Stereo EEG experiences a renaissance in North America related to advancements in stereotactic technology. Patients with deep lesions, s/p prior operation, or at risk for not being suitable for resection depending on result of invasive evaluation may be better suited for Stereo EEG than Subdural evaluations. Electrode selection is challenging, involves a close dialogue with the surgeon and requires stereotactic planning software and image co-registration technology. Depending on the case, a traditional orthogonal approach exploring the epileptogenic network or an oblique, 3D grid approach to delineate the epileptogenic lesion may be considered.

Image Processing and Surgical Planning
Giridhar Kalamangalam, MD, DPhil

Strategic sampling of the hypothesized epileptogenic zone will be discussed. Practical tips for optimizing diagnostic yield will be outlined, with illustrative cases. Post-implant electrode visualization using standard off-the-shelf software (Curry) will be discussed.

Nuts and Bolts of Surgical Implantation
Nitin Tandon, MD

The process of designing an implant and then proceeding to actual implantation will be discussed in detail. Frame-based, frameless and robotic SEEG techniques will all be discussed. Accuracy and complications of these various approaches will be reviewed. The process of planning resections after SEEG will also be analyzed.

Somatosensory Evoked Potentials (SEPs)
Ronald Emerson, MD, FACNS

This presentation will review the physiology of somatosensory evoked potentials, and the methodology and interpretative criteria of clinical somatosensory evoked potential testing. Representative examples will be presented.

Neurophysiologic Intraoperative Monitoring (NIOM): Part II
(9:00AM – 5:00PM)

Monitoring Cerebral and Spinal Endovascular Procedures
Viet Nguyen, MD

Aneurysms, arteriovenous malformations (AVMs), stenoses, fisulas, 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 neurophysiological 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.
EMG Monitoring of Central Motor Pathways During Spine Surgery

Stan Skinner, MD, FACS

Peripheral axons will depolarize if struck with sufficient energy. End velocity of the injurious impact to a nerve fascicle determines 1) whether individual axons will fire and 2) how many axons will fire within the fascicle, i.e., the amplitude of the recorded CMAP. Acutely (or chronically) injured nerves may lose the ability to accommodate; that is, they keep firing repetitively after the stimulus. Train neurotonics (“A trains” per Romstock) are likely explained by the “breakdown” of accommodation. We have seen similar phenomena upon spinal cord impact. 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. 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 D-Waves

Eva K. Ritzl, MD

The presentation will take the audience through the indications for and neurophysiology of D-wave monitoring.

Monitoring of Motor Cranial Nerves and Cranial Nerve Nuclei

Jaime R. López, MD, FACS

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 injury of a cranial nerve but can also involve the nucleus and its neural elements within the brainstem. 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 neuropsychiologic techniques, consisting primarily of EMG, used to monitor motor cranial nerves and cranial nerve nuclei.

Troubleshooting During IOM

Brett Netherton, MS, FASNM, CNIM

A thorough understanding of the fascinating worlds of electromagnetics, electronics, and physiologic signal volume conduction in tissue is most helpful for rapid IOM troubleshooting. This presentation will pull from these three worlds to attempt answers to the following questions: What is the best way to layout leadwire spaghetti to avoid noise?

Where is the ideal placement for the iso-ground electrode? And should you use multiple grounds? Why do some electrodes pick up more noise than others? How can stimulus artifact be minimized? How do electrode burns occur? How can they be avoided? What are electrode safety considerations in the MRI environment?

Electrocorticography and Intracranial EEG

(9:00AM – 5:00PM)

Phase 1 Evaluations That Lead to Phase 2 Testing

Giridhar Kalamangalam, MD, DPhil

The value-addedness of intracranial EEG in the workup of surgical epilepsy will be outlined in general. Archetypal situations for an invasive evaluation with SEEG will be discussed, with several case illustrations. Pitfalls and caveats to intracranial evaluation will be highlighted.

Choosing Phase 2 Electrodes

Stephan U. Schuele, MD, MPH, FACNS

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 predictable 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.

Patterns of Seizure Onsets & Spread, Underlying Pathological Substrates, Surgical Outcomes in Adults

Lawrence J. Hirsch, MD, FACNS

Intracranial EEG recordings are indicated for surgical treatment of refractory epilepsy when other tests to identify the seizure focus are discordant or inconclusive, when there is no MRI abnormality (except select medial temporal cases), when the seizure onset zone abuts eloquent cortex, and with dual pathology (e.g. hippocampal sclerosis plus a lesion). Complete removal of the seizure onset zone is associated with a greater chance of seizure freedom, even after accounting for lesion resection. Although outcomes in cases requiring intracranial electrodes are worse than those that do not, many patients still become seizure free, including those with negative imaging.
Complications of implanted intracranial electrodes occur in about 9% of patients and are mostly transient, with permanent deficits in <2% and rare mortality. Risks are higher with greater numbers of implanted electrodes, larger subdural grids, and peri-Rolandic location. The relative utility of subdural strips/grids, depth electrodes, stereo-EEG and combinations of these are unknown. Mesial temporal onset seizures on depth electrode recordings often begin with rhythmic spiking at <2 Hz (fairly specific for MTS, but can be seen with onset or spread into the hippocampus) or low voltage 10-16 Hz activity. Well localized neocortical onsets often start with low amplitude fast activity. Unfortunately, all onset patterns can also be seen as spread patterns. Many seizure onsets involve wider epileptogenic networks, such as the limbic network, the occipito-temporal network, and the parietal-frontal network.

High frequency oscillations (HFOs; ripples: 80-250 Hz, and fast ripples: 250-600 Hz) may help localize epileptogenic tissue, although there are many caveats. Fast ripples seem to be more specific than ripples for seizure onset zones, especially when associated with interictal spikes.

Discussions and Demonstrations of Patterns of Seizure Onsets & Spread, Underlying Pathological Substrates, Surgical Outcomes in Children
Tobias Loddenkemper, MD, FACSNS

Patients with refractory focal epilepsy are thoroughly evaluated to identify an area of cortex that, if removed or disconnected, will lead to seizure freedom. Clinical semiology, neuroimaging, and scalp electroencephalogram provide an approximation of this area, whereas intracranial recording may permit a more precise localization and investigation of a selected cortical area. Intraoperative electrocorticography delineates the irritative zone, and subdural electrode implantation also permits cortical stimulation of eloquent areas. Intraoperative electrocorticography rarely captures spontaneous seizures and may be influenced by the effect of anesthetic drugs, and the correlation between complete resection of the irritative zone and postsurgical seizure outcome is unclear. Exteroceptive monitoring is often superior to intraoperative electrocorticography, may present with unique patterns of seizure spread, but may also be associated with more risk of adverse events. Further development of ultrahigh-density electrode arrays is providing novel insights into the role of microseizures and high-frequency oscillations on ictogenesis and epileptogenesis, and may improve outcomes in the future.

The Use on Invasive Electrodes to Map “Epileptic Zones”
William C. Stacey, MD, PhD

Intracranial EEG has been the most sophisticated and precise means of diagnosing focal epilepsy for over 50 years, but for most of that time the technology remained unchanged. The resolution of IEEG was based upon identifying regional seizure foci, which led to epilepsy surgery and improved outcomes in many patients. However, traditional IEEG has resolution far below that necessary to understand the underlying mechanisms of seizures, and is much less effective in nonlesional extratemporal epilepsy. Recent technological advances with improved resolution may be able to help additional patients and improve our understanding of epileptic networks.
Neonatal EEG is often intimidating even to experienced neurophysiologists, with the EEG of premature newborns posing particular challenges. At the same time, with more preterm newborns surviving, there is a shifting emphasis toward providing neonatal intensive care that includes neurologic care, including EEG monitoring. This year’s course will review the evolution of normal findings in the EEG of preterm newborns across gestational ages. Common abnormal findings will be illustrated, with clinical correlations emphasized. This course will also provide an update regarding emerging evidence for seizures among preterm newborns, and suggest practical considerations for EEG monitoring in this unique patient population.

**New Directions in Sleep Medicine**  
(7:00 – 8:30AM)

**Chronobiology and Chronopharmacology Applied to Epilepsy**  
Tobias Loddenkemper, MD, FACNS

Approximately one-third of patients with epilepsy continue to have seizures despite antiepileptic therapy. Many seizures occur in diurnal, sleep/wake, circadian, or even monthly patterns. The relationship between biomarkers and state changes is still being investigated, but early results suggest that some of these patterns may be related to endogenous circadian patterns whereas others may be related to wakefulness and sleep or both. Chronotherapy, the application of treatment at times of greatest seizure susceptibility, is a technique that may optimize seizure control in selected patients. It may be used in the form of differential dosing, as preparations designed to deliver sustained or pulsatile drug delivery or in the form of ‘zeitgebers’ that shift endogenous rhythms. Early trials in epilepsy suggest that chronopharmacology may provide improved seizure control compared with conventional treatment in some patients.

**Case Discussion**  
Courtney J. Wusthoff, MD, MS

Neonatal EEG is often intimidating even to experienced neurophysiologists, with the EEG of premature newborns posing particular challenges. At the same time, with more preterm newborns surviving, there is a shifting emphasis toward providing neonatal intensive care that includes neurologic care, including EEG monitoring. This year’s course will review the evolution of normal findings in the EEG of preterm newborns across gestational ages. Common abnormal findings will be illustrated, with clinical correlations emphasized. This course will also provide an update regarding emerging evidence for seizures among preterm newborns, and suggest practical considerations for EEG monitoring in this unique patient population.

**Sleep and Stroke**  
Hrayr Attarian, MD

This presentation will introduce the audience to the bidirectional relationship between stroke and sleep disorders. The most common of these disorders is Obstructive Sleep Apnea followed by Central Sleep Apnea and Cheyne Stokes respirations collectively known as sleep disordered breathing (SDB). I will discuss the impact of SDB on stroke and related conditions such as atrial fibrillation. I will also discuss the impact of poor sleep in general as a risk factor for stroke and its association with longer hospital stays and poorer rehab outcomes. Lastly I will discuss the challenges associated with treating SDB in the setting of acute stroke and afterwards.

**Sleep as a Robust Biomarker of Neurodegenerative Diseases**  
Madeline M., Grigg-Damberger, MD, FACNS

This talk will summarize recent studies which show: 1) sleep is a robust biomarker for neurodegenerative diseases; 2) sleep fragmentation, short sleep duration and excessive daytime sleepiness are early biomarkers for minimal cognitive impairment (MCI), incipient Alzheimer’s Disease (AD), and future cognitive decline; 3) sleep loss in AD alters CSF beta-amyloid (Aβ) dynamics; 4) decrements in NREM 3 sleep are associated with decreased clearance of Aβ from brain; and 5) intermittent nocturnal hypoxemia (often associated with OSA) increases Aβ production, predisposing to activation of neurodegenerative processes contributing to AD.

**EMG**  
(9:00AM – 12:00PM)

**Motor Nerve Conductions and F-waves**  
Paul Barkhaus, MD

Motor nerve conductions are one of the core studies that comprise electoneuromyography. They are deceptively simple to perform- but are they? In order to record high quality, reproducible studies, the clinical neurophysiologist must have a thorough understanding of the physiology of the compound motor muscle action potential (CMAP), recording instrumentation, anatomic artifacts, and how recordings may be altered in neuromuscular disorders.

F waves are an extension of motor nerve conduction studies. There are different ways of analyzing F waves (e.g. minimum latency versus mean latency). Again, these may be easily recorded but are often misinterpreted and the data under-utilized.

The lecture will cover basic principles in performing motor conduction and F wave studies including their physiologic basis, pitfalls in recording, and examples in pathological states utilizing illustrative recordings and schematics.

**Muscle Disease and EMG**  
Elliot L. Dimberg, MD

The presentation will focus on the electrodiagnostic approach to various muscle diseases, including inflammatory myopathy, myotonic disorders, and inherited and metabolic disorders. Many will be reviewed via a case-based approach with audience participation. The primary goal will be to assist the attendee with muscle disease categorization clinically and electrophysiologically, show how electrophysiology can help narrow the differential diagnosis and investigative plan, and provide specific examples for clarification.
The goal of long term video EEG monitoring is to record the interictal and ictal EEG and the clinical manifestations of the patient’s typical seizures. Long term monitoring requires access to synchronized EEG, patient video/audio, and annotations. Flexible data access methods are needed to retrieve desired data quickly and permit sequential viewing of EEG, video, and annotations in forward and reverse mode, as well as “fast forward” and “fast reverse.” Other capabilities include jumping to next or previous annotation, jumping to an annotation selected from a list, and jumping to a specified date and time, repositioning the video as well as the EEG. Multiple modifiable display parameters, filters, and montages must be available. Digital filtering allows selecting the filter.

Epilepsy Surgery Evaluation in Pediatrics with VEM
Phillip L. Pearl, MD, FACNS

Presurgical evaluations for epilepsy surgery candidates aim to resolve the epileptogenic zone and relevant eloquent cortex, although the former concept is elusive and requires diagnostic modalities addressing at least five contributory zones: symptomatogenic, irritative, ictal onset, epileptogenic, and functional deficit. Modalities utilized range from electrophysiologic (scalp EEG, HD EEG, MEG, EEG, sEEG) to imaging (including MRI, DTI, PET, SPECT), and functional (fMRI, WADA, MEG EP, TMS, cortical stimulation, neurological and neuropsychological examinations). Criteria for utilization of intracranial EEG generally include cases that are nonlesional, near eloquent cortex, or have discordant results, ictal onset within a large lesion not amenable to complete resection, multiple potential ictal onset zones, or unclear margins. Intracranial EEG is generally not indicated in cases of unilateral MTS with concordant results, a well-defined ictal onset zone in noneloquent areas, or in cases appropriate for a hemispherectomy or hemispherotomy. The surgical procedure of choice also depends on etiology, e.g. hemispherotomy is indicated for Rasmussen encephalitis, hemihemangioencephaly, remote ischemic lesions, Sturge-Weber syndrome, or large cortical malformations. In contrast, focal resection is utilized for focal cortical dysplasia, tuberous sclerosis, tumors, or small cystic lesions. Cases utilizing video EEG monitoring concomitantly with intracranial EEG will be reviewed.

ICU EEG
(9:00AM — 5:00PM)

Overview of ICU EEG Monitoring in Neonates, Children and Adults
Nicholas S. Abend, MD

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

Guidelines and Nomenclature for ICU EEG Monitoring
Susan T. Herman, MD, FACNS

Continuous EEG monitoring (CEEG) in the intensive care unit (ICU) is an emerging technology for detection of secondary insults in patients with acute neurologic...
cEEG Interpretation: Seizures and Periodic Patterns
Suzette, M, LaRoche, MD, FACNS

Electrographic seizures can manifest as a variety of EEG signatures in the critically ill patients. Rhythmic, clearly evolving fast frequencies which are typical of seizures seen in the Epilepsy Monitoring Unit are uncommon in the ICU population and clinical signs are often absent. Seizures often present as slowly evolving, low frequency activity with ill-defined onset which makes identification more difficult. Extremely focal ictal activity, confined to one or two channels is also encountered emphasizing the importance of maintaining artifact free electrodes and utilizing full 10-20 montage electrode placement whenever possible. In addition, physiological and electrical artifacts are abundant in the ICU environment which can mimic seizures. Finally, there are a host of periodic and rhythmic patterns, both generalized and lateralized that can carry implications for treatment and outcome. This discussion will review electrographic seizures as well as periodic and rhythmic patterns often seen in the critically ill and include patient case discussions to illustrate some of the challenges of interpretation and management.

cEEG Interpretation: Neonates
Mark Scher, MD

Electroencephalographic interpretations are integral to neonatal neurocritical care. Recognition of EEG pattern abnormalities in the preterm and full-term infant provides essential diagnostic and prognostic information. Bedside EEG monitoring in the neonatal intensive care unit constitutes the current available technology for the visual recognition of pattern abnormalities associated with a suspected neonatal encephalopathy. Major abnormalities will be discussed relative to gestational maturity and trimester-specific etiologies including the peripartum and neonatal periods. Clinical neurophysiologists must consider maternal, placental, fetal and neonatal conditions to more accurately reflect both the severity and time course of fetal/neonatal brain disorders. Future comparisons of pattern abnormalities with normative EEG/critical databases will advance diagnostic, prognostic and therapeutic assessments for improved clinical service and research collaboration.

Quantitative EEG for Seizure and Ischemia Detection
M. Brandon Westover, MD, PhD
This talk will cover the fundamentals of interpreting spectrograms derived from continuous EEG data in ICU patients for detecting seizures and ischemia.

Logistics of ICU EEG Monitoring
Cecil D. Hahn, MD, MPH, FACNS
This presentation will provide an overview of strategies for organizing a successful ICU EEG monitoring program. I will review data on current EEG technologist and physician staffing practices for electrode application, troubleshooting and EEG interpretation across North America, including various solutions for after-hours coverage. I will illustrate the benefits of developing a team approach with educational outreach to ICU nurses and physicians in order to facilitate collaborative multidisciplinary care.

Finances, Billing and Coding
Mark R. Nuwer, MD, PhD, FACNS
CPT coding, billing, and adhering to regulations are necessary for Continuous ICU EEG practice. Coding CPT for procedures depends on whether the monitoring was continuously supervised, whether video was used, and for how long monitoring continued. The reading physician can be at a distance, but needs to be available to interpret during the recording so as to recommend changes in medical care during the monitoring and to determine when the recording can end. The main codes themselves inherently include digital spike and seizure detection, so those automated features cannot be separately coded. Diagnostic ICD coding can determine whether a service is paid, by setting the medical justification for the service. ICD also determine the Hierarchical Condition Categories (HCC), which affect the patient’s acuity level for payment purposes. In each case, chart documentation should justify the CPT and ICD codes chosen. Careful coding facilitates correct regulatory and payment processes. They are important parts of system-based practice.
Case Studies in Peripheral Neurophysiology

Case Studies in Peripheral Neurophysiology
Raghub Govindarajan, MD
The rapid expansion of clinical neuromuscular genetics, serological testing and greater understanding of pathological basis of these diseases have opened up new and exciting avenues for diagnosing once unknown neuromuscular disorders. While the role of nerve conduction studies/electromyography has been diminishing, it is still the first line of investigation for many neuromuscular disorders and provides diagnostic clues that in addition to clinical examination helps to direct appropriate tests including gene testing. In this session we will present real clinical vignettes and highlight the role of electro diagnostic studies in directing appropriate testing with active audience participation. This will be supplemented by imaging studies and pathology slides where appropriate.

Case Studies in Peripheral Neurophysiology
Elliot Dimberg, 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.

Business in Clinical Neurophysiology

(1:00 – 5:00 PM)

Criteria for Performance Evaluation — Concepts for Professional & Group Practice in an ACO
Elizabeth L. Mullikin, FACHE
Professional and Group Practice providers who generate the most value in an ACO healthcare delivery system will succeed and prosper. This presentation will take physicians through the overarching concepts of The Department of Commerce, National Institute of Standards & Technology, Baldrige Healthcare Criteria for Performance Excellence. This program offers a solid framework to achieve better outcomes, consistent high quality care, and cost savings to a populations of patients. Adopting a few of the key concepts from the Baldrige Performance Excellence Framework into practice can transform your clinical services into a system that is high performing, competitive, and sustainable.

ICU EEG Reading Session: Pediatric Cases

Eric T. Payne, MD, MPH
I will present two cases that highlight diagnostic and management dilemmas in paediatric patients undergoing ICU EEG monitoring. This will be an interactive presentation with ample opportunity for audience participation.

Applied Autonomic Neurophysiology

(3:00 – 5:00PM)

Neurological Disorders with Central Autonomic Failure
Alexandra Hovaguimian, MD
This talk will be an overview of common causes of central autonomic failure. This will include alpha-synucleinopathies, autonomic seizures and autonomic dysreflexia.

SE continuous IV midazolam or propofol and IV valproate are options and continuous IV pentobarbital is used for the most refractory cases.

Status epilepticus (SE) is a life threatening emergency requiring prompt recognition and treatment. SE may be classified into convulsive and nonconvulsive, based on the presence of rhythmic jerking of the extremities. Refractory status epilepticus (RES) is defined as ongoing seizures failing to respond to first- and second-line anticonvulsant drug therapy and carries a high morbidity and mortality. Treatment efficacy, morbidity and mortality are related to delays in starting treatment. Benzodiazepines are first line therapy usually followed by phenytoin/fosphenytoin. A low threshold should exist for obtaining an urgent electroencephalogram(EEG) since electrographic seizures are frequent after control of convulsions. For refractory SE continuous IV midazolam or propofol and IV valproate are options and continuous IV pentobarbital is used for the most refractory cases.

Case Studies in Peripheral Neurophysiology

(3:00 – 5:00PM)

Case Studies in Peripheral Neurophysiology
Patrick Chauvel, MD
Stimulation for Auras and Seizures
Patrick Chauvel, MD
Defining the epileptogenic zone with SEEG requires not only recording of spontaneous seizures, but also analyzing electrical and clinical effects of direct electrical stimulation. The use of electrical stimulation allows to confirm ictal onset topography and epileptogenic zone organization thereby guiding surgical planning. Methods. Two types of stimuli (single shocks or high-frequency trains) are used, depending on the cortical structure and on the goal of stimulation. No threshold determination is practiced, because seizure triggering is a all-or-none phenomenon, and repeated unsuccessful stimuli can decrease local excitability. Minimal current required to trigger an after-discharge or a seizure varies from patient to patient, from time to time in the same patient as a function of seizure occurrence, of interictal activity, and of anti-epileptic drug levels. Stimulation sessions are generally undertaken at the end of SEEG. Effects of stimulations are evaluated by analyzing electrical effects (none, after-discharge, seizure) and clinical manifestations (subjective or objective, functional or ictal) they have induced, and their correlations. There are actually divergent conceptions about the role of stimulation, leading to different practices according to epilepsy surgery centers. Discrepancies hold to the presurgical method used (SEEG or ECoG), which conditions strikingly different
**POSPONED**

**SPEAKER ABSTRACTS**

**Friday, February 6, 2015, continued**

**Cortico-Cortical Evoked Potentials**

**Dileep Nair, MD**

Clinically, understanding the connectivity between sites within an epileptic network may help clarify which patients are good candidates for surgical therapies as well as fine-tuning the amount of tissue which must be resected to achieve a good surgical outcome. Further, novel methods of characterizing an epileptic network offer the promise of allowing epileptologists to gather sufficient data to make confident hypotheses about surgical strategies. Towards this goal, cortico-cortical evoked potentials (CCEPs) offer a promising mechanism to investigate connectivity patterns in epileptic networks and in normal brain. CCEPs have been employed previously to illustrate the preferential excitability of seizure onset zones, describe normal connectivity patterns in language cortex and the motor cortex and to examine connectivity within contiguous areas of seizure propagation. The evaluation of epileptic cortex and seizure networks using CCEP will be discussed.

**Beyond Seizure Detection in Critical Care EEG Monitoring: Background Matters**

**(8:00-10:00AM)**

**EEG Reactivity and Variability: Standardization, Quantification and Significance**

**Nicolas Gaspard, MD, PhD**

Between ictal and interictal events, the EEG background also provides capital, but usually underappreciated and underused, information about brain function during critical illness. There is thus an opportunity for CEEG to be used as a cerebral function monitor and to help guide appropriate treatment. One obstacle to a more widespread application of CEEG in this indication is the relative lack of validated quantitative methods to translate conceptually important but loosely defined EEG terms, such as variability and reactivity, into clinically meaningful numbers that can be used by non-neurophysiologists at the bedside. At the conclusion of this presentation, participants will be able to discuss the physiological mechanisms of EEG reactivity and variability, how they can be quantified and how this quantification may assist in the assessment and prognostication of brain dysfunction and/or injury.

**Pelvic Floor Neurophysiology**

**(8:00 – 10:00AM)**

**Introduction & Anatomy; Autonomic and Somatic Sacral Reflexes**

**Jaime Ramos Peek, MD**

Pelvic floor dysfunction includes a wide range of symptoms; urinary or fecal incontinence, emptying abnormalities of the gastrointestinal or urinary tract, sexual dysfunction, and regional pain syndromes are common problems that can occur in diverse clinical conditions such as nerve compression syndromes, CNS disease, or systemic ailments like diabetes mellitus. To better understand pelvic floor dysfunction as well as the usefulness of neurophysiological tests in the many settings affecting this region, the knowledge of functional anatomy, physiology and neurological innervation of intrapelvic organs, muscles and support structures is necessary. The use of sacral reflex tests can evaluate normal autonomic and somatic neurological conduction in their afferent, efferent and central pathways as well as their voluntary and reflex regulation. Electromyography studies can be used to test the neuromuscular integrity of the urethral and anal striated external sphincters, puborectalis, and pubococcygeus muscles, sacral evoked potentials can help distinguish between peripheral and central dysfunction, and their use for monitoring these structures during surgical procedures can help prevent neurological deficits. They are an important addition to urodynamic and ano-rectal manometric tests in understanding the origin of these varied clinical symptoms.

**MEPs of the Pelvic Floor Muscles, Pudendal SEPs, EMG of Anal and Urethral Sphincters, Pudendal Nerve Terminal Motor Latencies**

**Armando Valdes-Tello, MD**

Pelvic floor disorders affect one in four adult women of all ages. The clinical have different presentations, such as pelvic organ prolapse, fecal or urinary incontinence and sexual dysfunction. With a neurological history and physical examination, the clinician might suggest as to where or which of the control mechanism is affected. However, most times, it is difficult to draw a conclusion on clinical grounds only. Given the complexity of the system, it is wise to add to the anatomic studies, neurophysiological evaluation. Neurophysiological studies currently available are quantitative electromyography (qEMG) of anal and urethral sphincters, pudendal nerve terminal motor latencies (PNTMLs), motor evoked potentials (MEPs) of the anal sphincter, pudendal somatosensory evoked potentials (SEPs), sacral reflexes, and perineal recording of sympathetic skin responses (SSRs). In lesions of lower motor, qEMG of anal and urethral sphincters is the most appropriate test, given the information of denervation/reinnervation this method give. If the lesion is at the level of the sacral level, sacral reflexes give important information. Pudendal nerve terminal motor latencies (PNTMLs), examines conduction of nerve fibers rather than levels of innervation, and examine large myelinated fibers conduction. In cases of upper motor neuron dysfunction, the utility of SEPs and MEPs is less clear. This symposium will review clinical usefulness, strengths and pitfalls of each technique, in establishing the integrity of the peripheral innervation of the pelvic floor and how the combination of different methods help to determine the level of the lesion.
SPEAKER ABSTRACTS

Friday, February 6, 2015, continued

Introoperative Monitoring of Pelvic Surgery
Guillermo Martin Palmomeque, MD

Introoperative neurophysiological monitoring (IONM) is widely used in spine, vascular and brain surgery. However, there are a variety of other surgeries where the risk of nerve injury is high but IONM is not used, such as in surgeries involving the pelvic floor. In pelvic floor surgery the complex intricate nervous system accompanying pelvic viscera, such as the inferior hypogastric plexus, pelvic splanchnic nerves parasympathetic, sacral splanchnic nerves, sympathetic pudendal nerve plexus, bladder plexus, rectal plexus, and in males the prostate plexus, dorsal penile and cavernous nerves, are at risk of injury. Injury to these structures can cause neurologic dysfunction and lead to serious functional impairment of activities of daily living, including urinary incontinence, fecal incontinence and impotence. The aim of this presentation is to demonstrate the utility of intraoperative electrical stimulation protocols that can be used to identify these structures in order to avoid iatrogenic surgical injuries. The technical stimulation and recording protocols are similar to those used in other surgeries. However, there are fundamental differences, primarily in the recording methods, which require the assessment of bladder pressure, penile tumescence, and the use of IAS needles. Published data indicate that these techniques are sensitive in identifying neural structures and that IONM is effective in helping preserve postoperative urinary, fecal and sexual function in a variety of gynecological, prostate and rectal surgeries. Thus, IONM, which is currently underutilized in pelvic floor surgery, should be used more frequently in these cases.

General Session
(10:20 – 11:45AM)

Gloor Award Lecture: “Cortical Activities Associated with Voluntary and Involuntary Movements”
Hiroshi Shibasaki, MD, PhD, FACNS

It was Kornhuber and Deecke of Germany who first recorded the pre-movement EEG activities by using a back-averaging technique in 1965. A symposium “Bereitschaftspotential (BP): 50 years after its discovery” was held during the ICCN2014 in Berlin. Since 1970’s, the author’s group has investigated the generating mechanism of BP by using various non-invasive techniques (multi-channel EEG, MEG, ERD, cortico-muscular coherence, PET and functional MRI), in combination with epidural recording of BP in patients with focal epilepsy as a part of pre-surgical evaluation. As the results, it is postulated that, before self-initiated simple movement, activation occurs first in the pre-supplementary motor area (pre-SMA) and the SMA proper bilaterally with some somatotopic organization, and then the lateral premotor cortex and the primary motor cortex, mainly contralaterally, with precise somatotopic organization. In praxis movements, BP starts from the parietal cortex of the dominant hemisphere. While the author was recording BP in a patient with progressive myoclonus epilepsy in 1975, a technique of jerk-locked back averaging was discovered with serendipity. Generating mechanisms of involuntary movements, especially myoclonus, can be studied by EEG-EMG polygraphic recording, jerk-locked back averaging and evoked potentials. Application of transcranial magnetic stimulation provides further information as to the cortical excitability. The sensori-motor cortex (SMC) was shown to play an important role in generation of cortical myoclonus, essential tremor, Parkinson tremor, and focal dystonia. Cortical myoclonus is actively driven by SMC, while essential tremor and Parkinson tremor are mediated by SMC which might serve as a relay center of the oscillations arising from subcortical structures.

Four Ways of Looking at Seizure Networks: A Critical Discussion
(1:00 – 3:00PM)

High-Frequency Granger Causality in the Analysis of Preictal Networks
Charles M. Epstein, MD

In recent decades, intracranial EEG (iEEG) recordings using increasing numbers of electrodes have indicated that seizure networks can be more extensive in space, time, and frequency than previously recognized. Often this complicates rather than simplifying the task of identifying areas for surgical resection, especially in cases of apparent multifocal onset. Granger causality (GC) is a form of mathematical regression that may help to identify which electrode sites are actually dominant in driving other locations. We have been using GC analysis at HFO frequencies to analyze features of preictal seizure networks and to aid in surgical decision making. All patients studied have had significant, widespread preictal GC network activity at peak frequencies from 80 to 250 Hz, beginning 2-30 seconds before visible seizure onset. In several prospective patients, GC source/sink comparisons supported the exclusion of early ictal regions that were not the dominant causal sources, and contributed to planning of more limited surgical resections. The first 2 cases, each had a class 1 outcome at one year. GC analysis of iEEG has the potential to increase understanding of preictal network activity, and help improve surgical outcomes in cases of otherwise ambiguous ictal onset.

Neural Network Architecture and the Clinical Course of Temporal Lobe Epilepsy
Leonardo Bonilha, MD, PhD

Even though modern neurophysiological and neuroimaging tools enable a better characterization of the type and location of seizure onset, patients with epilepsy with relatively similar findings may experience vastly different treatment outcomes. The shared similarities in clinical semiology and results from ancillary test are probably underscored by different pathophysiological mechanisms, which lead to different outcomes. Epilepsy is believed to be a disease supported by abnormal structure and function of brain networks. Nonetheless, until now, limitations in network mapping have prevented the assessment of individual patterns of network aberrations and their relationship with epilepsy treatment phenotype. With new developments in mapping whole brain connectivity, i.e., the connectome, this hypothesis can now be more directly tested. In this presentation, we will review methods employed to assess the connectome, findings from connectome research in epilepsy and the future translation of these findings to the bedside assessment and treatment of epilepsy.

Myoclonic Status Following Cardiac Arrest
(1:00 – 3:00PM)
Definitions and Questions
Elizabeth Gerard, MD

What is myoclonic status? The prognostic implications of post-anoxic myoclonic status have been studied and debated for years but surprisingly few studies have
**Pathophysiology of Anoxic Myoclonus**

*Mark Hallett, MD, FACS*

Two different types of myoclonus can arise after anoxia, acute and chronic. Chronic post-anoxic myoclonus has been more extensively studied. Here the myoclonus arises days or weeks after the anoxia and is associated with seizures and cerebellar signs. There can be spontaneous myoclonus, action myoclonus, both positive and negative, and reflex myoclonus. The pathophysiology is most commonly due to hyperexcitability of the sensorimotor cortex, and called cortical myoclonus. Some cases may also have hyperexcitability of the brainstem, apparently in the reticular formation, called reticular reflex myoclonus. The pathological substrate is less clear, but there is some evidence for cerebellar injury to be etiologic. Acute anoxic myoclonus is typically periodic and seen in the setting of deep coma with little cortical function. EEG may well show burst suppression. This myoclonus very likely arises from the brainstem, as cortical functioning is minimal or absent. Some patients who have more complex EEGs or cortical SEPs, might have a (partial) cortical origin of the myoclonus. Studies in animal models suggest that the relevant characteristics can be observed, that correlate with clinical outcome. Of importance is the moment of time with respect to the moment of the insult when particular clinical signs or EEG patterns occur. For instance, in the first few hours after cardiac arrest, the EEG may be iso-electric, but this is not invariably associated with a poor outcome; clearly, if this pattern persists, outcome is poor. This importance of timing also holds for spontaneous movements, including myoclonic jerks. Postanoxic myoclonus can be defined as spontaneous myoclonic jerks in a patient with a postanoxic encephalopathy. In most patients, this period is prolonged (>30 minutes), and can then be defined as postanoxic myoclonic status. Myoclonic status epilepticus is defined as frequent myoclonus of presumed or certain cortical origin, regardless of etiology, although one may argue that this needs an explicit EEG correlate (showing epileptiform discharges, proving cortical involvement). For completeness: myoclonic status epilepticus is observed in various clinical conditions, ranging from idiopathic epilepsy syndromes to inflammation, toxic-metabolic derangements and anoxic brain injury. In this talk, I will shortly review some key pathophysiological processes that occur after the primary insult in patients with postanoxic encephalopathy, and correlate these with the potential to generate myoclonic jerks. Recent literature will be reviewed. I will also argue that EEG monitoring can assist in the interpretation of the postanoxic myoclonus, including the prognostic significance.

**Prognosis After Cardiac Arrest in Era of Therapeutic Hypothermia: In Children with Postanoxic Myoclonus**

*Courtney J. Wusthoff, MD, MS*

Following the widespread use of therapeutic hypothermia following cardiac arrest in adults, hypothermia was soon applied for neuroprotection in pediatric cardiac arrest patients. With clinical trials still underway to demonstrate a benefit of hypothermia in pediatric arrest patients, there remains much to be learned regarding seizures and EEG during hypothermia. Conflicting data exists regarding the incidence of seizures in children receiving post-arrest hypothermia; short-term outcome data is limited. When considering prognosis in the pediatric patient following pediatric arrest, some principles from the adult populations are likely applicable, though with adjustment needed for the unique causes of arrest in the pediatric population, for divergent pathophysiology of brain injury in arrest, and attention to overall different expectations for children following cardiac arrest as compared to adults.

**Myoclonic Status After Cardiac Arrest: To Treat or Not to Treat**

*Jay Gavwale, MD*

Two cases of post-anoxic myoclonic status will be presented including discussion of the patient’s co-morbidities and presenting history. The cases will be presented in chronological order with detailed discussion of the presenting myoclonus including video EEG findings. Discussion of the two cases between the two panelists will focus on whether the presentation meets criteria for myoclonic status, necessity of EEG correlate to the clinical signs and if that changes management and what further diagnostic and therapeutic steps would be recommended. The two contrasting opinions will be compared to the actual hospital course and the pros and cons of each approach will be weighed.

**Prognosis After Cardiac Arrest in Era of Therapeutic Hypothermia: In Adult Patients with Postanoxic Myoclonus**

*M. J. van Putten, MD, MSc, PhD*

In patients with postanoxic encephalopathy, various clinical signs and EEG characteristics can be observed, that correlate with clinical outcome. Of importance is the moment of time with respect to the moment of the insult when particular clinical signs or EEG patterns occur. For instance, in the first few hours after cardiac arrest, the EEG may be iso-electric, but this is not invariably associated with a poor outcome; clearly, if this pattern persists, outcome is poor. This importance of timing also holds for spontaneous movements, including myoclonic jerks. Postanoxic myoclonus can be defined as spontaneous myoclonic jerks in a patient with a postanoxic encephalopathy. In most patients, this period is prolonged (>30 minutes), and can then be defined as postanoxic myoclonic status. Myoclonic status epilepticus is defined as frequent myoclonus of presumed or certain cortical origin, regardless of etiology, although one may argue that this needs an explicit EEG correlate (showing epileptiform discharges, proving cortical involvement). For completeness: myoclonic status epilepticus is observed in various clinical conditions, ranging from idiopathic epilepsy syndromes to inflammation, toxic-metabolic derangements and anoxic brain injury. In this talk, I will shortly review some key pathophysiological processes that occur after the primary insult in patients with postanoxic encephalopathy, and correlate these with the potential to generate myoclonic jerks. Recent literature will be reviewed. I will also argue that EEG monitoring can assist in the interpretation of the postanoxic myoclonus, including the prognostic significance.

**Pediatric Case Presentation (Debate)**

*Courtney J. Wusthoff, MD, MS & Cecil D. Hahn, MD, MPH*

This debate will discuss the pros and cons of aggressive treatment of myoclonic status epilepticus after cardiac arrest in a child.

**Advanced Electrodiagnostic Techniques**

**Quantitative Electromyography**

*Paul E. Barkhaus, MD*

Quantitative EMG (Q-EMG) or “objective EMG” is a disciplined approach to acquiring and analyzing concentric or monopolar needle electrode recordings of motor unit potentials (MUPs) using parametric statistics. With contemporary electromyograph systems, some consider Q-EMG obsolete; but is it? Q-EMG means much more than “mean MUP amplitudes”, etc. First, it is our foundation for understanding clinical EMG, the features of the MUP, and how the motor unit (MU) is altered in neuromuscular disorders (NMD). Second, it encompasses other techniques such as single fiber EMG, macro-EMG, and interference pattern analysis that add more information on the MUs. Muscle biopsy remains the “gold standard” for specific
diagnoses but sampling is typically limited to a single site. Conversely, Q-EMG is a sensitive technique that allows sampling of multiple muscles to determine an optimal site for potential biopsy (author’s term, the “electrophysiologic biopsy”). With improved training, the electromyographer can obtain more data from the myogenic signals (i.e., “interactive EMG”) with little added testing time. Future challenges in Q-EMG include how to extract even more information from signals by varying the selectivity of a disposable recording electrode. Improved diagnostic sensitivity will depend on greater electrode selectivity, whereas monitoring NMD will require less selective electrodes.

Macro Electromyography
Joe Jabre, MD

Starting in the late seventies, Electroencephalography, once the premier way of monitoring the central nervous system, began to get eclipsed by the advent of new brain imaging techniques. But EEG all of a sudden showed a new resurgence with the advent of video EEG monitoring, sleep studies, and novel algorithms for the prediction of seizures etc. Just as progress in Electromyography started to plateau. In this presentation I will describe four novel EMG techniques that, with other ongoing changes in the field, may lead the resurgence of the field of electromyography. The four techniques I will review are: A Volitional Unit that consists of functional cortico-motoneuronal connections between affector cortical motoneurons and effector spinal motoneurons for the generation of a purposeful movement. The Oxyneurography technique, a Near Infra Red Spectroscopy technique (NIRS) that allows the monitoring of nerve tissue oxygenation. The Concentric Macro EMG electrode that permits the recording from the majority of muscle fibers in a motor unit. And, the extrapolated norms or e-norms technique that allows for the extraction of a population reference values from a laboratory patient cohort.

Motor Unit Number Estimation
Ioannis Karakis, MD, MSc

Determining the number of functioning motor units is an integral part of the assessment of diseases that afflict them, not only to evaluate for disease progression, but also response to treatment. Clinical and histological methods cannot easily and reliably provide the actual number of functioning motor units. Hence, we rely on their estimation through electrophysiologic means. Motor unit number estimation (MUNE) can be obtained by a number of different techniques such as manual incremental stimulation, multiple point stimulation, spike-triggered averaging, statistical method, F-wave technique as well as with combinations of the aforementioned methods or mathematical models of them (e.g. motor unit number index-MUNIX). Each method has different advantages and disadvantages. Therefore, the selection of a method depends on the muscle being tested, the equipment available and the physician’s experience. This talk will focus on the basic physiology behind the motor unit estimation techniques, describe the various methods along with their strengths and weaknesses, and explore their research and clinical applications in neuromuscular disorders.

Electrical Impedence Myography
Seward Rutkove, MD

Electrical impedance myography (EIM) is a non-invasive technique for the evaluation of neuromuscular disease. The technique is finding application predominantly as a biomarker in the evaluation of a variety of neuromuscular conditions ranging from amyotrophic lateral sclerosis to muscle dystrophy. It may also have diagnostic value. This lecture will introduce the underlying theory of the technique, review EIM data in different conditions, both in humans and in animal disease models, and present some of the ongoing technological developments. By the conclusion of the presentation, attendees should have a firm grasp of the science underlying the technique, methods for its application, and its potential uses in the field of neuromuscular disease.

ICU EEG SIG: Periodic and Rhythmic Patterns
(3:00 – 4:00PM)

GPDs
Brandon Foreman, MD

Generalized periodic discharges (GPDs) have been described for over half a century. Clinical associations vary from post-cardiac arrest hypoxic-ischemic encephalopathy to Creutzfeldt-Jacob disease to drug toxicity to liver failure. GPDs are highly associated with the development of nonconvulsive seizures, although debate exists about certain subtypes, triphasic waves, which are not typically considered epileptiform. The underlying pathophysiology of GPDs remains to be clearly defined, although most evidence suggests that different pathology may lead to a common manifestation on EEG. Evidence is mounting that all GPDs exhibit an increased risk for seizures regardless of morphology, and that our impressions of these patterns as EEGers may be inaccurate without individual clinical details. Further, even traditionally ‘benign’ appearing triphasic GPDs respond frequently to trials of anticonvulsants. Cases are discussed along with management options.

LPDs
Indranil Sen-Gupta, MD

Lateralized periodic discharges (LPDs) remain a controversial EEG pattern. Are they “ictal” or “interictal” phenomena? How aggressively should they be treated? This presentation will explore some of these controversies and highlight potential insights from one institution’s experiences with LPDs. We found that LPDs having time-locked somatosensory manifestations had significantly higher odds of being associated with central head regions than those that did not; otherwise, no significant differences in multiple patient characteristics were noted between these two groups of LPDs. In addition, the prevalence of LPDs appeared to be much higher in our inpatient population undergoing EEG monitoring than that historically recognized based on prior mixed inpatient/outpatient populations obtaining routine EEGs. The majority of patients with LPDs had seizures: most seizures and LPDs were detected during the first day of EEG monitoring, and >90% of both were detected by the second monitoring day. These findings may have implications regarding conceptualizations of LPDs as “ictal” versus “interictal”, the prevalence of LPDs in the inpatient setting, and the duration of EEG monitoring in patients with LPDs.

Friday, February 6, 2015, continued
**Speaker Abstracts**

**Friday, February 6, 2015, continued**

**LRDA**

Nicolas Gaspard, MD, PhD

Lateralized rhythmic delta activity (LRDA) is a recently described EEG pattern occurring in critically patients. It resembles lateralized periodic discharges (LPDs) in several ways as it is most often associated with an acute brain injury and a high risk of acute seizures. In this presentation, we will provide an update on the clinical significance of LRDA and discuss its relation to similar EEG patterns, such as LPDs and other types of rhythmic delta activity.

**Periodic and Rhythmic Patterns in Pediatric Patients**

Josh Goldstein, MD

Named coma patterns including alpha coma, spindle coma, suppression burst coma are regularly encountered in critically ill adults undergoing continuous EEG monitoring (cEEG). A variety of data demonstrate that named coma patterns are not uncommonly seen in critically ill children. I will review the relevant studies and suggest some specific outcomes based on specific patterns.

**Saturday, February 7, 2015**

**Innovative Electronic Devices in Clinical Neurophysiology**

(8:00 – 9:30AM)

**Cutting Edge Technologies and Electronic Devices for Diagnosing and Managing People with Epilepsy**

Steven Schachter, MD, FACNS

Ambulatory and home monitoring of patients with epilepsy using simultaneous EEG and video provides valuable diagnostic information in the evaluation of episodic behaviors. 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 sudden death, track response to changes in therapy, and increase our understanding of epileptogenesis. Challenges associated with regulatory pathways and proof of clinical value are important for clinicians to consider in adapting these technologies into their practices or recommended them to patients.

**Latest Developments in Non-epileptic Brain Stimulation and Quantitative EEG**

Charles M. Epstein, MD, FACNS

Transcranial magnetic stimulation (TMS) is now widely used in neuroscience research. Three systems with highly dissimilar features have been approved in the US for the specific clinical applications of depression and migraine. Other potential neuropsychiatric applications are being extensively studied. The modern reincarnation of transcranial direct current stimulation (TDCS) requires not much more than a handful of parts from Radio Shack. At typical currents it’s considered fundamentally safe, but the nature and magnitude of its effects are weak and widely debated. A surprising number of other electrical stimulation techniques are being studied or are already prevalent in the community. Recently, transcranial ultrasound has been proposed for brain stimulation as well. Decades ago the early excitement over quantitative EEG (QEEG) was swamped by abuse; as a consequence its research potential has been under-appreciated by the neurological community. One QEEG system has been approved for ADHD. Although abuse persists, and many promising applications have been displaced by modern genetics and neuroimaging, QEEG continues to produce noteworthy research findings under other names.

**Gizmos, Gadgets and Electronic Devices for Diagnosing and/or Treating Patients with Stroke, Dementia, and Brain/Spinal Cord Injury**

Madeleine M. Grigg-Damberger, MD, FACNS

Our symposium and this lecture aims to review new, advanced and/or cutting edge medical gizmos, gadget electronic devices and smartphone apps being tried or used to diagnose and/or treat patients with particular neurological disorders. Medical gizmos are mechanical devices or procedures for which the clinical benefit is not clearly established. Medical gizmo idolatry is the conviction that a more technological approach is intrinsically better than one less technological unless there is strong evidence to the contrary. This presentation will focus on discussing innovative devices to diagnose and/or treat patients with dementia, stroke and spinal cord/brain injury. Some of the more fetching devices reviewed: 1) monitor wandering patients with dementia and autism; 2) powered exoskeletons to provide standing and walking functions to individuals with paraplegia; and 3) virtual reality rehabilitation systems for patients with stroke. The scientific basis (or lack of) for these devices, as well as the advantages, limitations, flaws, availability, and cost of these will be reviewed. You can decide whether to incorporate some of these into your clinical practice to improve the value and quality of care provided to your patients.

**Electroclinical Features of Autoimmune–Mediated Epilepsies: Case Discussions**

(8:00 – 9:30AM)

**NMDA Receptor Antibody Encephalitis**

Sarah Schmitt, MD

Anti-NMDA receptor (anti-NMDA-R) encephalitis is a clinical condition characterized by dyskinesia, psychosis, cognitive impairment, seizures and autonomic instability in individuals with antibodies to the NMDA receptor. Some patients with anti-NMDA-R encephalitis develop a unique EEG pattern, known as extreme delta brush. This presentation will discuss the case of a 19 year old man who presented with clinical features of anti-NMDA-R encephalitis, and the EEG features associated with his illness.

**LGI1 Antibody Related Epilepsy, Faciobrachial Dystonic Seizures**

Ji Yeoun Yoo, MD

A case of a 58 year old man who had a rapid onset of progressive confusion, twitching of the face and hand, and abnormal basal ganglia on brain MRI, initially diagnosed to have a CJD is presented. Faciobrachial dystonic seizures, possibly pathognomonic for the limbic encephalitis associated with anti-voltage-gated potassium channel complex (VGKCC) antibodies (VGKCC syndrome), have been misdiagnosed as myoclonus. His symptoms completely resolved with treatment. Given the similarities yet very different prognosis, recognizing the distinctive features is emphasized.
SPEAKER ABSTRACTS

Saturday, February 7, 2015, continued

Anti-GAD Antibody and Other Autoimmune-Mediated Epilepsies
William B. Gallentine, DO, FACNS
Glutamic acid decarboxylase (GAD) antibodies have been associated with several neurologic conditions including limbic encephalitis and epilepsy. This video EEG case discussion will emphasize the electroclinical features of GAD antibody encephalitis / epilepsy which are suggestive of an underlying autoimmune etiology. Diagnostic and therapeutic approaches will also be discussed.

Pedicle Screw Cases
Christine Hung, MD
Neurophysiologic intraoperative monitoring data recorded during several simulated pedicle screw testing cases will be presented in an interactive session. The audience will be asked to analyze the NIOM data, to discuss possible causes of the observed changes in the evoked potential data, and to address the clinical significance of the NIOM findings. At the conclusion of this activity, the participant should understand the related neurophysiology, technical aspects, interpretation, and communication of pedicle screw testing results.

Pedicle Screw Stimulation - A Discussion by Some Experts
(8:00 – 9:30AM)

Pedicle Screw Stimulation- A Discussion with Some Experts
David Gloss, MD
Objective: To determine the efficacy of pedicle screw stimulation as an intraoperative monitoring modality.

Methods: A systematic review was performed of the following question: In patients with spinal disorders, does pedicle screw stimulation compared with either other forms of pedicle screw stimulation or routine care reduce the risk of poor outcome. I used the 2004 AAN methodology with the 2013 AAN risk of bias to answer the question.

Results: There were 160 unique articles identified by the search. Three studies met inclusion criteria; they were all rated as Class III.

Conclusions: There were no studies which compared pedicle screw stimulation to routine care, so no definitive conclusions about efficacy can be made. Both standard stimulation and trains of stimulation are possibly effective interventions to reduce the risk of poor outcome in spinal surgery.

Technical Considerations for Pedicle Screw Testing
Alan D. Legatt, MD, PhD, FACNS
When a pedicle screw is properly placed, completely encased by bone, a high stimulating current is needed to stimulate nearby nerve roots and elicit EMG responses; a low threshold indicates a high probability of pedicle screw breakthrough. Technical factors can make an improperly placed screw appear to have a high threshold, however. Stimulation of the movable head of a screw rather than the shank, or the use of hydroxyapatite-coated screws, can introduce a high resistance into the stimulation circuit. Fluid puddled around the screw head can shunt much of the stimulating current away, artifactually elevating the apparent threshold. Chronically compressed nerve roots may be more difficult to stimulate, prompting revision of the threshold criterion. Neurovascular blockade would eliminate EMG responses; recording of an EMG response to stimulation of at least one site is useful as a positive control.

Pedicle Screw Cases
Cormac O’Donovan, MD
Neurophysiologic intraoperative monitoring data recorded during several simulated pedicle screw testing cases will be presented in an interactive session. The audience will be asked to analyze the NIOM data, to discuss possible causes of the observed changes in the evoked potential data, and to address the clinical significance of the NIOM findings. At the conclusion of this activity, the participant should understand the related neurophysiology, technical aspects, interpretation, and communication of pedicle screw testing results.

General Session
(9:30 – 11:15AM)

Jasper Award Lecture: “A Personal Perspective on 50 Years of Clinical Neurophysiology”
John Ebersole, MD, FACNS
Using my own career as the temporal template, I will review the evolution of clinical (and some basic) neurophysiology over the last 50 years. There have been significant improvements in equipment and technology, but many of our procedures and attitudes toward data analysis have essentially remained unchanged. The digital era should have brought vast changes in the way we approach interpretation, but alas in many instances it has not. Despite enormous analysis flexibility, the majority of clinical EEG is interpreted from just 10-20 electrodes, in a longitudinal bipolar montage, with 1-70 Hz filtering, and a 10 second page. So what has really changed? Hopefully its sister technology, MEG, with its emphasis on field analysis and source modeling, will encourage or perhaps shame practitioners of EEG to exploit more fully all the information contained in these signals.

Peripheral Nerve Injury and Evaluation
(11:15 – 12:45PM)

Nerve Injuries in Sports
Mark Hallett, MD, FACNS
Injury to peripheral nerves in sports can be due to several mechanisms. There can be direct, acute trauma and entrapment with microtrauma over time due to repetitive stress. Each sport has its characteristic injuries depending on the nature of the activities in that sport. In baseball, the most common problems have to do with the throwing motion, that is also seem with throwing a football or javelin and serving in volleyball and tennis. Due to valgus strain of the elbow, there can be ulnar nerve injury. Problems arise on the opposite side of the elbow with “tennis elbow” and might include entrapment of the posterior interosseous nerve. With bicycle riding the ulnar nerve is vulnerable in Guyon’s canal and the pudenal nerve can be compressed by the bicycle seat. With jogging, there can be heel pain due to the tarsal tunnel syndrome, medial calcaneal neuritis or entrapment of the first branch of the lateral planter nerve. Jogger’s foot is an entrapment of the medial plantar nerve.

FINAL PROGRAM
SPEAKER ABSTRACTS

Saturday, February 7, 2015, continued

Electrodiagnosis of Peripheral Nerve Injury
Jun Kimura, MD

Seddon defined three degrees of nerve injury: neuapraxia, axonotmesis, and neurotmesis. In neuapraxia, or conduction loss without structural change of the axon, recovery takes place within days or weeks. In axonotmesis, the axon loses continuity with subsequent wallerian degeneration along the distal segment. Recovery depends on regeneration of subsequent nerve fibers, a process that takes place slowly over months or years at a rate of 1 to 3 mm per day. In neurotmesis, an injury also involves the supporting connective tissue. Sunderland (1978) has proposed three subdivisions of neurotmesis. In the first type, the injury damages the axon and surrounding connective tissue, preserving the architecture of the nerve sheath. The nerve regenerates effectively after this type of injury though less completely than in axonotmesis. Misdirected nerve fibers may reach the muscle previously not supplied by the injured nerve, causing the clinical phenomenon of synkinesis. In the second type, the nerve barely maintains the continuity although it may look grossly intact on inspection, usually necessitating surgical intervention. The third type represents a complete separation of the nerve with loss of continuity. Surgical repair consists of suturing the stumps, usually with a nerve graft to bridge the gap. This classification, originally proposed for traumatic injury, also applies to entrapment and compression neuropathies such as the carpal tunnel syndrome and tardy ulnar palsy.

Use of Ultrasound to Evaluate Peripheral Nerve Injuries
Francis O. Walker, MD, FACNS

Neuromuscular ultrasound is a unique tool for evaluating peripheral nerve and nerve injuries. Because it evaluates the morphology of nerves, it can evaluate nerve continuity at any point in a nerve injury, and can provide information regarding neuroma formation, hemorrhage, or damage to nearby structures. Furthermore, nerve injuries, even when incomplete, are often associated with focal nerve swelling distal or proximal to the site of injury. Ultrasound can also provide physiologic information about nerve mobility and blood flow, but the extent to which these parameters are informative in nerve injury is unknown. Prior case series have shown that ultrasound is particularly informative in the evaluation of suspected nerve trauma and this presentation will highlight a number of examples, which include a discussion of evaluating failed or complicated operative repairs. It is recommended that those who perform electrodiagnosis develop familiarity with neuromuscular ultrasound as it is likely to help in the evaluation of affected patients. That it is non-invasive, portable, and of low cost further enhances its value.

Probing Cortical Physiology: The Use of TMS-EEG in Epilepsy and Psychiatry

(11:15AM – 12:45PM)

Brain Stimulation and Cortical Physiology in Patients with Epilepsy
Mouhsin Shafi, MD, PhD

Many forms of epilepsy are associated with aberrant neuronal connectivity, but the relationship between such pathological connectivity and the associated epilepsy is unclear. We employed transcranial magnetic stimulation (TMS) with simultaneous EEG recording in eight patients with epilepsy from periventricular nodular heterotopia (PNH) and matched healthy controls. We used resting-state fMRI connectivity imaging findings to guide TMS targeting, and compared the evoked responses to single-pulse stimulation from different cortical regions. Heterotopia patients with active epilepsy demonstrated a relatively augmented late evoked response that was greater than that of matched controls, and was specific to cortical regions with connectivity to subcortical heterotopic gray matter. Topographic mapping of the late response differences showed distributed cortical networks that were not limited to the stimulation site, and source analysis in one subject revealed that the generator of abnormal TMS-evoked activity overlapped with the spike and seizure onset zones. Our findings indicate that patients with epilepsy from gray matter heterotopia have altered cortical physiology consistent with hyperexcitability, and that this abnormality is specifically linked to the presence of aberrant connectivity. These results support the idea that TMS-EEG could be a useful biomarker in epilepsy, expand our understanding of circuit mechanisms of epileptogenesis, and have potential implications for therapeutic neuromodulation in conditions associated with deep lesions.

Translational Applications of TMS in Animal Models of Epilepsy and Brain Injury
Alexander Rotenberg, MD, PhD

Transcranial magnetic stimulation (TMS) is emerging as a practical tool in clinical epilepsy, and has recently been adapted to rodent epilepsy models. In translational experiments, the capacities for repetitive TMS (rTMS) to depress cortical excitability, and for paired-pulse TMS (ppTMS) to measure cortical excitability can be developed as means for suppressing seizures or for estimating seizure vulnerability. This presentation will provide an overview of translational TMS methods and findings in acute seizure models, and will focus on recent recent insights into the pathophysiology of post-traumatic epileptogenesis that have been obtained with novel rat TMS methods.

TMS-EEG Studies in Patients with Schizophrenia and Other Psychiatric Disorders
Zafiris J. Daskalakis, MD, PhD

Objectives: Transcranial magnetic stimulation (TMS) has been used as both a probe to measure neurophysiological processes including connectivity, inhibition and plasticity in the human cortex. Methods: An overview of the methods involved in demonstrating connectivity, inhibition and plasticity measures as indexed through TMS electromyography (EMG) and electroencephalography (EEG) studies will be provided. Results: TMS combined with EMG and EEG can be used to identify key neurophysiological processes in both the motor and the dorsolateral prefrontal cortex. These neurophysiological processes have been shown to be altered in patients with severe psychiatric disorders (e.g., depression, schizophrenia) and such findings will be reviewed. Conclusions: TMS can be used as an effective tool to help identify the pathophysiology of severe psychiatric disorders. I will conclude by discussing such findings in the context of their potential as biological makers of illness and of treatment response.

Epilepsy Case Study in Video- EEG

(11:15AM – 12:45PM)

Case Discussion 2
Dang Nguyen, MD

The speaker will present an epileptic patient. After showing the history, the video of her seizures, EEG and MRI findings, the audience will have the opportunity to discuss
Recognition of Artifacts During Nerve Conduction Studies and Needle EMG
Devon Rubin, MD

Artifactual responses are common during nerve conduction studies and needle EMG in the electrophysiologic evaluation of neuromuscular diseases. Many artifactual responses recorded during nerve conduction studies are the result of technical factors (such as inappropriate electrode placement or overstimulation), physiologic factors (such as low limb temperature), or result from anomalous anatomy. During needle EMG, the sensitivity of the recording electrode and the very low amplitude waveforms allows for externally generated artifacts to be recorded. Many of these artifactual EMG waveforms have features that could mimic and be misinterpreted as abnormal waveforms. Recognition of these different artifacts and identification of methods to minimize them is critical for adequate interpretation of electrophysiologic studies.

EEG and the Trouble with Artifact
William O. Tatum, DO, FAAN, FACS

EEG is a critical means of evaluating neurological disorders used in increasing frequency in “hostile” environments. Some physiologic artifact is necessary for interpretation, though frequently recordings are contaminated by non-essential artifacts. Potentials that originate from sources other than the brain can create pattern interference of the true sources generated by cerebral electrical fields. Artifacts that are produced by external “noise” may be produced by deterioration of the scalp electrode interface, 60 Hz (or 50 Hz outside the US), electronic devices, and machines that are extrinsic to the patient-EEG circuit.

Artifacts are crucial for the clinical neurophysiologist to understand (artifactology) and reflect a common pitfall in EEG often leading to misinterpretation and mistreatment of patients. In addition little consensus exists as to when artifacts make EEGs unable to be analyzed and interpretable. With the advent of digital EEG and implementation of software-based post-hoc filtering and montage selection, artifact reduction may be achieved. However reduction based upon frequency filtering relies on cerebral-artifact frequency separation often limited by similar overlapping waveforms that are independently indistinguishable. Similarly, post-hoc changes of referential montages may help but have selective application. On the horizon of development are new methods employing sophisticated computational methods that are important to recognize.

Common and Uncommon Artifacts in Polysomnography
Madeleine M. Grigg-Damberger, MD, FACNS

Expected and unexpected artifacts are common in polysomnography (PSG). Some are easily recognized, others less so. Having seen the artifact before makes it easier to recognize. Not all artifacts are undesirable; some represent paroxysmal events which if missed, miss a particular diagnosis. Most recording artifacts are recognized by their exaggerated appearance, breaking the background and catching the eye.

Some artifacts need to be recognized by the technologist, and then corrected; others identified and noted for later clinical correlation and interpretation. This brief presentation will review some common and uncommon physiological artifacts or mistaken electrode placements encountered in PSG which demonstrate the value of time-locked video to confirm their origins.

To Alert or Not - Artifacts in NIOM
Aatif M. Husain, MD, FACNS

Artifacts are common in all neurophysiologic tests. When they appear in neurophysiologic intraoperative monitoring (NIOM), they often present as a change in baseline data. The neurophysiologist must determine whether this represents potential neurologic compromise or is simply artifact. In this presentation, various examples will be presented in which NIOM changes that appear to be significant are simply artifacts. Suggestions on how to distinguish between real changes and artifacts will be presented.

EEG - fMRI in Epilepsy
(2:00 — 3:30PM)

Translational EEG-fMRI: Narrowing the Gap Between Animal and Human Studies
Seyed Mirmirani, MD

This is a review of the current state of simultaneous EEG-fMRI technology and its application to clinical epileptology. By the end of this course, participants will be able to understand the basics of simultaneous EEG-fMRI technology, the rationale for its use and become familiar with animal models and human studies using simultaneous EEG-fMRI. The evolving role of this technology as a pre-surgical tool in epilepsy surgery with its pitfalls and shortcomings will be discussed.

Brain Hemodynamic Responses Related to Classical EEG Rhythms
Ana Carolina Coan, MD

In the last two decades, studies using EEG-fMRI have improved our knowledge of the functional significance of different physiological and pathological EEG patterns. New understanding about the generation of the posterior dominant rhythm has emerged. Sleep physiology has advanced through studies identifying the brain response to stimuli during different wake-sleep stages and the cortical and subcortical circuits associated with the transition from wakefulness to different sleep stages. In addition, the brain hemodynamic response associated with specific sleep structures have been described. In the epilepsy field, EEG-fMRI have contributed to the identification of neural network associated with different characteristic epileptiform discharges. Worth mentioning, the studies with patients with idiopathic generalized epilepsies brought notable advance in the pathophysiology of generalized spike-and-wave complexes. Specific hemodynamic responses have also been recognized for other specific epileptiform patterns as continuous spike and wave during slow sleep and paroxysmal fast activity in patients with Lennox Gastaut syndrome. Being a non-invasive and accessible tool, EEG-fMRI has a vast potential to bring new advances to the neurophysiology field.
Evaluating Costs and Outcomes for IOM Using Decision Modeling
John P. Ney, MD, MPH

Intraoperative Neurophysiological Monitoring (IONM) for surgeries of the spine has been around for several decades, but recent alterations in reimbursement schemes by third party payers have raised the issue of the value of these procedures. Decision modeling using comparative effectiveness techniques holds the promise of evidence-based assessment of both cost and meaningful outcomes. In this presentation, we will review the elements of comparative effectiveness analyses followed by a detailed examination of existing decision models for IOM in spinal procedures.

Perspectives for the Clinical Use of EEG-fMRI
Fernando Cendes, MD

Neuroimaging techniques are widely used for definition of the epileptogenic lesion and surgical decision in patients with epilepsy. However, its application extends to the knowledge of epileptic mechanisms and includes the identification of prognostic features on an individual basis. Neuroimaging may be able to identify patients more likely to respond to anti-epileptic drug (AED) treatment and better candidates for earlier surgical treatment. In the last decade, multiple studies have shown that changes in the brain hemodynamic network related to epilepsy. Previous reports of EEG-fMRI in patients with temporal lobe epilepsy have demonstrated a consistent pattern of BOLD responses related to IEDs in areas including bilateral mesial and neocortical temporal structures but also extra-temporal regions such as insula and anterior cingulate. Advances in functional EEG-fMRI will add further knowledge in the field of epilepsy.

Reflex Epilepsy: How to Define and Determine It
Howard Ring, MD

Seizure precipitation is a defining characteristic of reflex seizures and epilepsies, but seizure precipitants are also commonly reported for patients with epilepsies not considered to be reflex in nature. This raises the questions of exactly how reflex and nonreflex epilepsies with seizure precipitants are defined, and how these concepts are differentiated from one another in current practice.

In this presentation I will discuss how definitions of reflex seizures, reflex epilepsies, and precipitation in a nonreflex context have been employed in published primary research papers. I will describe how content analysis has been applied to these definitions to identify their main features, allowing comparisons to be made between definitions of the different concepts. I will also consider how different classifications of potential precipitants have been considered in terms of their nature.
Clinical Practice and EEG Diagnostic Tools
Dorothée G. Kastelein-Nolst Trenite, MD, PhD, MPH

Besides a detailed clinical history about specific stimuli that might have provoked seizures, diagnosing reflex epilepsy is made especially through EEG recording. A routine EEG with intermittent photic stimulation might already be sufficient. However, in many cases and dependent on complexity of the reflex stimulus, it may take longer stimulation and extensive evaluation before a relation is detected, like in pattern sensitivity, reading or musicogenic epilepsy. In addition, it may require extensive evaluation by standardized procedures, such as those that have been developed for photosensitivity. The variety in reflex epilepsies and the crucial role of EEG in diagnosing will be shown and discussed.

Diagnosing Cognition-Induced Epilepsies
Anthony L. Ritacci, MD

Activating specific cognitive networks can affect seizure frequency or directly precipitate seizures. Cognition-related epilepsies comprise a group of loosely tied syndromes characterized by seizures regularly precipitated by cognitive tasks. Linguistic operations (e.g., reading, writing) and decision making associated with visuospatial manipulation are the most frequent and best-characterized triggers. Syndromes linked to language and thinking as principal activators suggest that “higher-order” seizure triggers are more common than currently identified. As pure reading epilepsy exists, so too are well-documented examples of individuals whose seizures are activated by multiple modalities, e.g., reading, speaking, or writing. Recorded cases document a spectrum between a narrow and a more broad range of cognitive activations suggesting a continuum within these artificially segregated syndromes. Evidence from fMRI-EEG coregistration, electrocorticography, and magnetoencephalography suggests that alteration of gamma frequencies within the ictal zone during function or afferent modification may be causal. Cognitive induction, as well as other established provocations are imbedded in systems/topographies (linguistic, visuo-spatial), whereby alteration in discrete sites or global function of the complex system may be effective. This explains the overlap between reported lesional and genetic cases. Treatment options include typical pharmacological and surgical interventions as well as stimulus alteration, threshold alteration, and avoidance conditioning. Commonly encountered epilepsy syndromes may also have complex triggers that are difficult to discern.

Electrocorticography: Overview and Future Directions
(4:30 – 6:00PM)

Electrocorticography: How to Improve Signal Acquisition, Signal Analysis and Interpretation
Brian Litt, MD

Techniques to record and analyze ECoG are a mainstay of pre-surgical evaluation for epilepsy surgery. While good technique and visual interpretation of these signals are vital patient management, newer methods for data acquisition and analysis hold great promise for improving localization of epileptic networks and therapy. Examples of these techniques include new high density transparent arrays for simultaneous optical and electrical recording, network analysis for localization of regions that drive seizure initiation and propagation, and cloud-based methods for analyzing and comparing seizure patterns between new and known cases. New technologies, analytical methods and data sharing/comparison methods hold great promise to improve and standardize high quality epilepsy care.

Electrocorticography: Physiology, Methodologies and Anesthetic Considerations
Rafeed Alkawadi, MD

Recent advances in EEG data acquisition have opened exciting opportunities and new levels of investigation for clinical practice and research. This continues to advance our understanding of the function and pathology of human brain. This presentation will provide an overview of the basics, methods, and indication of intra-operative and extra-operative electrocorticography (ECoG). We will discuss the clinical significance of common ECoG findings, contribution to the decision-making and correlation with surgical outcomes. We will provide a quick overview of current and future applications and recent research findings.

Electrocorticography and Functional Mapping: Current and Future
Josef Parvizi, MD PhD

Localization of functions in the human brain is at the core of clinical neurology practice. Recent advances in electrocorticography (ECoG) and electrical brain stimulation (EBS) in epilepsy patients implanted with intracranial electrodes have given rise to unique findings about the functional and causal role of specific regions of the brain. These new observations have yielded unprecedented data about the mode of responses in localized patches of cortical neurons during not only controlled experimental paradigms but also in daily-life natural settings when conscious patients interact with their natural environment while their brain is being monitored with intracranial electrodes. In this presentation, a historical overview will be followed by some of the most recent empirical data.

Autonomic Function
(4:30 – 6:00 PM)

Autonomic Dysfunction in Disorders of the Peripheral Nervous System
Arturo A. Leis, MD

This presentation will review some of the peripheral nervous system (PNS) disorders that are commonly associated with autonomic dysfunction. Case presentations will further aid attendees to recognize and formulate a differential diagnosis of the PNS disorders that may present with dysautonomia, including systemic diseases, toxic causes, immune-mediated causes, and infectious etiologies. Appropriate diagnostic evaluation and treatment of these clinical disorders will be briefly reviewed. Attendees will also improve their ability to communicate with and educate patients, families, and other healthcare colleagues about the clinical features of autonomic nervous system dysfunction.
EPILEPTIC SLOW SHIFTS: NEOCORtical Epilepsy

Akio Ikeda MD, PhD

In the current era of wide-band, clinical digital EEG, epileptic slow shifts, or ictal slow shifts, are one of the successful examples of translational research from animal studies in 1960s to clinical application in epilepsy surgery since 1990s. It is now regarded as a surrogate marker of epileptogenicity, similar to high frequency oscillation (HFO). However, since both deal with extremely slow or fast activity, careful recording, observation, analysis and interpretation are needed to extract real signals. This skills workshop was aimed to standardize and to share the minimum knowledge and skills in slow shifts and HFO.

(1) Recording of intracranial slow shifts: AC amplifier with long time constant (TC) of 10sec enables us to record. High input impedance >50Mohm, and platinum electrode are essential. A single system recording reference is usually selected.

(2) Data analysis: ECoG is recorded and also displayed with long TC. Initially, by visual inspection, slow shifts are observed at the time of seizure occurrence mainly of negative polarity (>3sec in duration, at least >200microV in amplitude, Ikeda et al, 1999). Slow artifact drift mimicking ictal shifts should not be misinterpreted very
Epileptic Slow Shifts: Limbic Epilepsy
Pradeep Modur MD

It is well known that slow baseline fluctuations of EEG signals around 0 Hz, called DC shifts, occur during epileptic seizures. Although these slow potentials are most clearly recorded with special DC-coupled amplifiers, it has been convincingly shown that they can also be recorded using the routine AC amplifiers, giving rise to the terms epileptic slow shifts or ictal baseline shifts to refer to the DC shifts. In clinical practice, the slow shifts can be successfully visualized in the relatively artifact-free intracranial recordings using an AC amplifier with a long time constant (≥ 10 seconds), high input impedance (>50 megohms), and platinum electrode contacts. The recordings are usually analyzed using referential montages, 0.016 Hz (~0.02 Hz) low frequency filter, and 30–60-second time window. The slow shifts have gained increasing importance recently, and are felt to be useful in the localization of the seizure onset zone (SOZ) in both temporal and extratemporal epilepsies. Specifically, the slow shifts studies have shown to have a more restricted spatial distribution than the conventional frequency-defined SOZ, and to occur in close temporal relation to the high frequency oscillations. Methods of recording, analysis, and interpretation of slow EEG potentials are discussed in this workshop.

High Frequency Oscillation (HFO) in Adult Epilepsy
Jean Gotman, PhD

EEG has traditionally included frequencies up to 80 or 100 Hz. In the last decade, EEG activity between 100 and 500Hz (High Frequency Oscillations or HFOs) consisting primarily of low amplitude short oscillations, has been recorded first in human intracranial microelectrodes and then in the intracranial EEG of epileptic patients. HFOs often but not always coincide with epileptic EEG spikes. They appear to be a good surrogate marker of epileptic activity for several reasons: (1) they are most frequent in the seizure onset zone; (2) they seem to reflect disease activity as they follow the fluctuations of seizure occurrence when medication levels are changed (unlike spikes); (3) they appear to be a good indicator of the epileptogenic region as they are a better predictor of post-surgical outcome than spikes or the seizure onset zone, in adults and children. Surprisingly, it has recently been demonstrated that some HFOs can be recorded on scalp EEG in focal epilepsy as well as in some forms of generalized epileptic discharges. This has raised the possibility that it is possible to record from the scalp EEG activity generated over a small region of cortex.

The above topics will be reviewed briefly, and we will discuss the issues encountered in marking HFOs from intracerebral as well as from scalp EEG.
When GABAergic drugs open chloride channels, chloride flows down its concentration gradient out of the cell, causing excitation. The diuretic bumetanide (BUM) was shown to block NKCC1, thereby reversing the chloride gradient and reducing seizure activity. These findings prompted interest in testing BUM to treat neonatal seizures. We initiated the Boston Bumetanide Trial to test BUM as add-on therapy to treat newborns with seizures refractory to an initial loading dose of phenobarbital in a randomized, double blind Phase I/II multicenter trial. We are using EEG monitoring to determine the pharmacodynamics of different doses of BUM, in addition to determining pharmacokinetics and safety of BUM in this population of newborns. I will review the challenges and successes of implementing the study design with regard to using continuous video-EEG monitoring to detect and quantitate seizure activity in newborns.

**Clinical Neurophysiology Trials and Tribulations in the ICU**

(10:00 – 11:30AM)

**From Conception to Implementation: TRENDS**

Aatif M. Husain, MD, FACNS

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.

**TELSTAR: Treatment of Electroencephalographic Status Epilepticus after Cardiopulmonary Resuscitation**

Michel J. van Putten, MD, MSc, PhD

In patients with postanoxic encephalopathy, various clinical signs and EEG characteristics can be observed, that correlate with clinical outcome. Of importance is the moment of time with respect to the moment of the insult when particular clinical signs or EEG patterns occur. For instance, in the first few hours after cardiac arrest, the EEG may be iso-electric, but this is not invariably associated with a poor outcome; clearly, if this pattern persists, outcome is poor. This importance of timing also holds for spontaneous movements, including myoclonic jerks.

Postanoxic myoclonus can be defined as spontaneous myoclonic jerks in a patient with a postanoxic encephalopathy. In most patients, this period is prolonged (>30 minutes), and can then be defined as postanoxic myoclonic status. Myoclonic status epilepticus is defined as frequent myoclonus of presumed or certain cortical origin, regardless of etiology, although one may argue that this needs an explicit EEG correlate (showing epileptiform discharges, proving cortical involvement). For completeness: myoclonic status epilepticus is observed in various clinical conditions, ranging from idiopathic epilepsy syndromes to inflammation, toxic-metabolic derangements and anoxic brain injury.

In this talk, I will shortly review some key pathophysiological processes that occur after the primary insult in patients with postanoxic encephalopathy, and correlate these with the potential to generate myoclonic jerks. Recent literature will be reviewed. I will also argue that EEG monitoring can assist in the interpretation of the postanoxic myoclonus, including the prognostic significance.

**The Boston Bumetanide Trial for Neonatal Seizures**

Janet Soul, MD

Seizures occur more frequently in newborns than at any other age, are difficult to control and are associated with poor long-term neurologic outcome, yet there have been very few clinical trials or prospective studies of antiepileptic drugs (AEDs) to treat neonatal seizures. NKCC1 is a cotransporter found to be highly expressed in neonatal neurons that results in a high intracellular chloride concentration, such that when GABAergic drugs open chloride channels, chloride flows down its concentration gradient out of the cell, causing excitation. The diuretic bumetanide (BUM) was shown to block NKCC1, thereby reversing the chloride gradient and reducing seizure activity. These findings prompted interest in testing BUM to treat neonatal seizures. We initiated the Boston Bumetanide Trial to test BUM as add-on therapy to treat newborns with seizures refractory to an initial loading dose of phenobarbital in a randomized, double blind Phase I/II multicenter trial. We are using EEG monitoring to determine the pharmacodynamics of different doses of BUM, in addition to determining pharmacokinetics and safety of BUM in this population of newborns. I will review the challenges and successes of implementing the study design with regard to using continuous video-EEG monitoring to detect and quantitate seizure activity in newborns.

**What’s Next in Clinical Neurophysiology Trials?**

Cecil D. Hahn, MD, MPH, FACNS

This brief talk will summarize the important questions that remain to be addressed by clinical neurophysiology trials in the intensive care unit and highlight trials currently in progress or in the planning stages.

**When Surgery is Not an Option, Neurostimulation and Beyond**

(10:00 – 11:30AM)

**Eloquent Cortex, Bilateral seizures, Difficult Approach; Evolving Approaches for Refractory Epilepsy**

Dawn Eliashiv, MD

Patients with medically refractory focal seizures are often evaluated for resective epilepsy surgery through imaging and neurophysiological evaluation. Unfortunately the epileptogenic region at times colocalizes with eloquent cortex or is bilateral. In these patients alternate approaches such as laser guided ablation close to fMRI or direct cortical stimulation defined regions, RNS which requires neurophysiological evaluation and management of seizure detection threshold require additional neurophysiological knowledge and decision making. The first presentation will provide an overview of these principles.
Friday, February 6, 2015
Display Time: 7:00 AM — 5:00 PM
Poster Tours: 3:00 — 4:00 PM
Location: Liberty Hall, 2nd floor

Categories: F1 Autonomic Function
F2 – F3 Basic Neurophysiology
F4 – F10 EEG
F11 – F13 EMG/NCV TESTING
F14 – F22 Epilepsy: Clinical
F23 – F38 Intraoperative Monitoring
F39 – F40 Magnetoencephalography
F41 – F44 Peripheral Neuropathy
F45 – F51 Video EEG for Epilepsy Monitoring

F1
Central Neural Substrates of Cardiorespiratory Control During Slow Breathing and Hypoxic Challenge
Alessia Nicotra, MD; Hugo Critchley, MD; Patrizia Chiesa, MD; Yoko Nagai, MD; Marcus Gray, MD; Ludovico Minati, MD; Luciano Bernardi, MD

Controlled slow breathing (at 6/min) can have beneficial effects on cardiovascular function, including responses to hypoxia. We tested neural substrates of cardiorespiratory control in 20 healthy subjects (F=8; age = 35 ± 10 yrs) during paced (slow and normal rate) and unpaced spontaneous breathing of normoxic and hypoxic (13% inspired O2) air using functional magnetic resonance imaging. Cardiovascular and respiratory measures were acquired synchronously. Peripherally, slow breathing was associated with increased tidal ventilatory volume; hypoxia suppressed heart rate variability and increased heart rate. Centrally, slow breathing activated dorsal pons, cerebellum, periaqueductal grey matter, hypothalamus, thalamus, lateral and anterior-insular cortices. Blocks of hypoxia induced activation of mid pons, bilateral amygdalae, anterior insular and occipitotemporal cortices. Interaction between slow breathing and hypoxia was shown in ventral striatal and frontal polar activity. Across conditions, within brainstem, dorsal medullary and pontine activity correlated with tidal volume and inversely with heart rate. Activity in rostroventral medulla correlated with beat-to-beat blood pressure and heart rate variability. Our study outlines slow breathing effects on central and cardiovascular responses to hypoxic challenge. The findings show involvement of discrete brainstem nuclei to cardiorespiratory coupling and corticostriatal circuitry during physiological responses accompanying breathing regulation in hypoxic challenge.

F2
The Effects of Hypoxia on TRPC4 Down Regulation in an Invitro BBB Cell Model
Nida Pasha, MD

Many studies have confirmed an increase in paracellular permeability after a hypoxic or ischemic event. In particular a new superfamily of cation-permeable channels TRP are of particular interest as they are expressed on BBB endothelial cells, previous studies indicate TRPC4 regulates vascular permeability in lung during hypoxia. However the role of TRPC4 in BBB permeability is unknown.

ECV304 cells were used to set up in an invitro BBB model, after which they were used to measure monolayer permeability after 3, 6, 24, 48 hours of hypoxic/ anoxic exposure. The monolayer permeability was quantified by calculating the flux of Na-F across the monolayer. Knock down of TRPC4 gene expression using siRNA was used to investigate the role of this channel, for which a new siRNA delivery method was optimized. Western blot analysis was carried out on these knock down cells in order to quantify the efficiency of TRPC4 knockdown. Monolayer permeability and cell viability was assessed after 24 & 48hours of hypoxic exposure. Results suggested that 48 hours of hypoxia caused a significant increase in monolayer permeability compared to control. Although there was no difference in permeability during 3-6 hours of hypoxic exposure, there was a fall in cell viability and protein concentration at 24 hours. The transfection of siRNA-TRPC4 resulted in cells that were more resistant to 48 hours of hypoxia compared to wild type cells. The transfected cells exhibited a lower permeability and higher cell viability compared to wild type cells. This study indicates the importance of TRPC4 in mediating the disruption of BBB permeability following prolonged hypoxia and the channel may be a useful therapeutic target for stroke.

F3
A Computational Analysis of Potential Mechanism of Action in High Frequency Spinal Cord Stimulation
Jay L. Shils, MD; Lonzhi Mei, MD; Kris Carlson, MD; Jeff Arle, MD

Introduction: High frequency (>1000 Hz) spinal cord stimulation (SCS) is a new technique for treating neuropathic pain (NP) without the standard side effects of parasthesias typical of lower frequency (F) SCS. The specific neurophysiologic mechanisms that allow for reduction of NP with generating the parasthesia side effect are not known. Using computational modeling techniques we have determined the specific ionic gate dynamics combined with the geometry based activating function (AF) for different axonal fibers diameters (D) are potentially the primary mechanism behind this therapy.

Methodology: A 50 node active model of an axon was created that allowed for variations in D, intermodal length, (IL) and multiple ionic gate sub-component parameters. Using spatially correct electric field data from a model of the spinal cord the dynamics of multiple axons D were studied. From the spatial geometry, the AF, and the temporal resoiunse of the axon, down to the level of the H, M, and N gate the system dynamics were calculated at various stimulation F, pulse widths (PW), and amplitudes (A) as well as axon diameters.

Results: By varying the stimulation F, A and fiber D, axons can be excited or blocked including the blocking of large D fibers while still allowing for transmission through smaller D fibers. The data demonstrates that the specific F where the HF stimulation starts to cause a complete conduction in the 14um fibers is around 4200 Hz which coincides with the time constant of the m-gate and its ability to reset within the stimulation period.

Conclusion: Present single pulse trained neuromodulation techniques act by either generating trains of action potentials in axons following a large to small D recruitment order or by caused anodal blocking. Our model proposes that for HF stimulation the blocking mechanism is a function of the gate dynamics and the AF side bands.

F4
Generalized Periodic Discharges in Antibiotic-Induced Neurotoxicity
Justin Cheongsitdamy, MD; Chutima Saipetch, MD; Dawn Eliashev, MD, FACNS; John Stern, MD; Marc R. Nower, MD, FACNS

Generalized Periodic Discharges (GPDs) can occur in anoxic injury, metabolic encephalopathy, drug toxicity, CNS infections, and neurodegenerative disorders. We describe a 60-year-old woman with end stage renal disease on peritoneal dialysis who was admitted for induction chemotherapy for acute myeloid leukemia.
The hospitalization was complicated by bacteremia and rapid clinical decline after initiation of culture-sensitive vancomycin and cefepime. Although her neurological exam was intact prior to receiving antibiotics, she quickly worsened into a state of minimal responsiveness with myoclonus over the course of four days. Brain MRI and lumbar puncture did not reveal structural lesions or CNS infection. Repeated blood cultures were negative and renal function remained at baseline. An initial EEG demonstrated intermittent, sharply contoured waves with triphasic morphology. Subsequent EEG monitoring showed electrographic progression into unremitting and synchronous 2.5 Hz GPDs without evolution. GPDs persisted despite empiric treatment with IV lorazepam, levetiracetam, and valproic acid. Due to family wishes against intubation, burst suppression was not pursued and the family placed the patient in hospice. In retrospect, we postulate that cefepime toxicity may be a contributing factor to this patient’s course. Encephalopathy and myoclonus are known manifestations of cefepime-induced neurotoxicity. EEG findings include triphasic-like waves as well as continuous, generalized, sharply contoured complexes with associated slow waves. In this patient, there was a progression from the former to the latter which correlated with her clinical decline. Physicians should be aware of the potential for pharmacological neurotoxicity and remain vigilant when observing abnormal electrographic features in this setting. Clinical outcomes may improve if offending agents are promptly identified and withdrawn.

F5
**Small Fast Rhythmic Eye Movements (SFREM) Misdiagnosed as Frontal Seizures**

Ana Tercero, MD; Carles Goig, MD; Alex Iranzo, MD; Juan Bernardo Gómez, MD; Joan Santamaria, MD

AMPAR encephalitis is an infrequent disease and its clinical presentation, course and prognosis are not well known. Electroencephalographic changes reported are nonspecific. We report a 23-year-old patient with AMPA-receptor encephalitis who developed prominent small fast rhythmic eye or eyelid movements (SFREM) in the EEG that were misdiagnosed as frontal seizures. The artifact was seen in the EEG channels as intermittent runs of low voltage theta activity at 6 Hz that did not change in frequency, and initially occurred associated with jaw rhythmic movements, yawns, and drowsiness (image 1). The background EEG of the patient was low amplitude and this fact probably allowed seeing this eye movement artifact better. Recording the eye movements with a piezoelectric transducer showed that the low voltage theta activity in the frontal channels was synchronized with similar frequency motions as seen by the transducer. This misdiagnosis caused changes in the therapeutic attitude. Our case emphasizes the need to recognize SFREM and to differentiate them from frontal seizures.

F6
**EEG Findings May Predict Outcomes of Herpes Simplex Encephalitis**

Daiki Fujii, MD; Hitoshi Mori, MD; Katura Shindo, MD;

Introduction: Herpes simplex encephalitis (HSE) is a severe life threatening disease. Although acyclovir has significantly improved the outcome, mortality rate and risk of neurological sequelae remain high. Quick diagnosis and treatment are inevitable. HSE shows various types of electroencephalogram (EEG) findings, but the relationship between EEG findings and prognosis is obscure.

Methods: We retrospectively identified HSE patients admitted in our hospital between December 2004 and September 2014. We assessed clinical courses, modified Rankin scale at discharge, initial EEG findings, brain imagings, and cerebrospinal fluid (CSF) findings. EEG findings were classified into four categories as follows, 1) burst suppression, 2) bilateral periodic lateralized epileptiform discharges (biPLEDs), 3) PLEDs, and 4) intermittent slow waves (IS).

Results: 9 patients were enrolled. 5 were men, and mean age was 53.7 years old. 4 patients were diagnosed by positive CSF HSV PCR, and 5 by rising titer of CSF HSV antibody. All patients showed abnormalities on brain MRI. One patient showed burst suppression, and mRS at discharge was 5. 2 showed biPLEDs, and mRS was 4 and 4. 3 showed PLEDs, and mRS was 3, 3, and 1. 3 showed IS, and mRS was 1, 0, and 0.

Conclusions: Abnormal EEG findings tend to indicate bad outcomes. Initial EEG findings may be an independent prognostic factor of HSE.

F7
**Paroxysmal Generalized Electrographic Delta Activity with Confusion During Natalizumab Infusion**

Hamid Kadivola, MD; Ryan S. Hays, MD; Paul C. Van Ness, MD; Mark Agostini, MD; Benjamin Greenberg, MD; Kan Ding, MD

Introduction: Natalizumab infusion is a FDA approved treatment for relapsing-remitting multiple sclerosis (RRMS). This is the first reported case of a suspected natalizumab infusion related encephalopathy.

Case: A 35-year-old woman with a history of RRMS was referred to the Epilepsy Monitoring Unit (EMU) for characterization of her spells. Episodes of confusion and shaking would exclusively occur within two hours after natalizumab infusion; but she would subsequently return to baseline within 24 hours. The most recent episode occurred three days prior to her EMU admission. 48-hour video-EEG monitoring (vEEG) was non-diagnostic, since no events were captured and the interictal EEG was normal. On the date of the next scheduled infusion, she was at neurologic baseline with clear mentation; she was treated with ibuprofen, acetaminophen, and cetirizine prior to natalizumab per infusion protocol. An EEG obtained during infusion demonstrated generalized 2-3 Hz polymorphic delta activity; these EEG changes were accompanied by her typical spell within 15 minutes of infusion completion. During this time she became confused, agitated, and subsequently pulled off her electrodes eight minutes later. Her mental status returned to baseline after 24 hours.

Discussion: This case illustrates transient non-epileptic encephalopathy as a potential reversible complication of natalizumab infusion.

F8
**Clinical and EEG Features in Autoimmune Encephalitis**

Michael Mendoza, MD; Robert Beach, MD; Kiran Aravapalli, MD

Prognosis of autoimmune encephalitis depends on early clinical recognition. Electroencephalography and long term monitoring are important to guide treatment in cases with equivocal imaging and CSF results. Retrospective chart review of 7 patients admitted at SUNY Upstate Medical Center admitted between 2011-August 2014 with a (+) serum antibody for P/Q VGCC, VGKC or NMDA receptor. The age range was 2-61 years old with 4 males / 3 females. The most common presenting symptom was seizure preceded by behavioral or movement disorders. MRI imaging and spinal fluid were mostly normal. Serum P/Q-type VGCC (2/7), VGKC (3/7) and NMDA (2/7) were detected. Normal EEG, generalized slowing, focal slowing,
and epileptiform abnormalities mostly involving the temporal head region were seen on EEG. Long term video EEG monitoring showed electrographic seizures (3/7) and non-epileptic events (4/7). Metastatic screening revealed malignancy or suspected mass in 2/7 cases. MRS on discharge range was 2-5.

Brain Imaging was normal in most cases implying that these antibodies cause a functional or micro-structural change. Long term Monitoring is important to delineate seizures from non-epileptic events which prevents inadvertent use of AEDs. Patients with abnormal EEG had poorer functional outcome on discharge.

F10
Emergent EEG- Utility During Business Versus Non-business Hours
Gowri Lakshminarayan, MD; Thandar Aung, MD; Laura L. Lehnhoff, MD

Introduction: Emergent EEGs are ordered for rapid diagnostic and treatment decisions. Not all hospitals have the capability to perform this highly valuable tool during non-business hours in their clinical setting due to lack of available man power. In order to determine the yield of this test, we reviewed the results of 3 months of emergent EEGs in our tertiary epilepsy facility.

Results: Emergent EEGs done over 3 months (N=168) were analyzed retrospectively. N=122 from business hours (7AM-7PM) and N=46 from non-business hours (7PM-7AM).

Discussion: Two-thirds of the emergent EEGs were ordered during regular business hours and one-third during non-business hours. The most common reason for the request in either group was altered sensorium. Electrographic seizures were seen in 4% of the studies in each group. There was higher incidence of normal studies (15% vs 8%) during non-business hours (perhaps to facilitate ER discharges overnight). One fourth of the recordings in each group showed epileptiform abnormalities, suggesting that emergent EEGs are a valuable tool in the appropriate clinical setting.

F11
EMG Findings of Radiation Induced Myopathy Mimicking ALS
Irisa Devine, MD; Devon Rubin, MD

Introduction: Myopathy is a rare effect of radiation therapy and the clinical and EMG features of radiation myopathy are not well described.

Objective: To describe a case of a patient with clinical features suggestive of ALS, in whom needle EMG findings helped confirm radiation induced myopathy.

Case Report: A 54-year-old woman with remote past medical history of breast cancer, Hodgkin's lymphoma, and radiation therapy to the entire spine 25 years prior to presentation, developed a 10 month history of right leg weakness, falls, and camptocormia. Neurologic examination demonstrated severe axial weakness, right leg weakness, hyperreflexia, and bilateral triple flexion responses. She was referred for EMG with a presumptive clinical diagnosis of ALS. Needle EMG revealed severely short duration motor unit potentials in the cervical and thoracic paraspinal muscles with a few fibrillation potentials and no myokymic discharges. The EMG was consistent with radiation induced axial myopathy. MRI of the spine revealed a normal spinal cord, with severe atrophy of the cervical and thoracic paraspinal muscles.

Conclusion: Radiation myopathy can mimic other neuromuscular diseases, such as ALS. Needle EMG is an important ancillary test that can help distinguish radiation induced myopathy from other mimicking disorders.

F12
Masseteric Contraction or Motor Imagery Enhances Ulnar Nerve F Wave
Motohiko Hara, MD; Miki Kawanmata, MD; Mizuki Kobayashi, MD; Rina Yoshida, MD; Jun Kimura, MD

Background: We tested the validity of instructing patients to clench the teeth to facilitate F-waves recorded from a limb muscle in clinical practice.

Objective: To study effect of motor imagery and voluntary contraction of the masseter muscles on F waves recorded from the first dorsal interosseous muscle.

Methods: In 7 healthy subjects, 50 F waves each were recorded at rest, during motor imagery and during voluntary contraction monitored by a force transducer to maintain the level at 10% of maximal effort.

Results: F-wave persistence increased significantly (p<0.05) from 33.7±21.4% (mean ± SD) at rest to 44.3±22.8% during motor imagery and 54.9±27.9% during voluntary contraction. The F-wave amplitude and latency revealed no significant changes.

Discussion: We have previously shown that motor imagery or slight voluntary contraction of the target muscle enhances F-wave persistence. The current study indicates the same process involving a non-target muscle can also cause a transient
POSTER ABSTRACTS

F13
Biopsy and EMG Findings in a Case of Late-Onset Dropped Head Syndrome after Mantle Field Radiotherapy
Amro Stino, MD; Mark Ross, MD, FACNS

We present the case of a 66 year old lady with a chronic dropped head syndrome initially misdiagnosed as dystonia. She had received mantle radiation therapy from her chest up for stage IIA Hodgkin's Lymphoma. Cancer recurrence in 2006 was treated with chemotherapy alone. Multiple treatments, including Botox, and physical therapy failed to improve the head drop. Bedside examination at our institution showed weak and atrophic sternocleidomastoid and trapezius muscles with paraspinal cervical muscle atrophy, with no associated anterocollis and a normal exam otherwise. EMG/NCS showed normal spinal accessory and supraspinal nerve responses with fibrillation potentials and myopathic units in the cervical paraspinals with occasional similar changes in the trapezius. No myokymia was seen. An infraspinatus muscle biopsy showed a mixed neuromyopathic picture with type I fiber grouping with slight myopathic features. This case is unique in that it provides biopsy and electrodiagnostic evidence to support the growing body of literature that suggests a mixed neuromyopathic (Leeuwen-Segarceanu, et al, 2012) picture in patients with late onset radiation therapy head drop. This is a diagnosis that needs to be entertained in the setting of head drop syndrome, as Botox therapy, used for presumed dystonia, may exacerbate patient complaints.

F14
Hemifacial Spasm or Epilepsia Partialis Continua
Tammam Dayyoub, MD; Sarita Maturu, MD; Andrew J. Zillgitt, MD

Introduction: Epilepsia partialis continua (EPC) is a form of focal motor status epilepticus that may be seen in the adult population following stroke. Hemifacial spasm (HFS) is a movement disorder characterized by recurrent contractions of muscles innervated by the facial nerve. Although these two conditions are distinctly different, when facial muscles are affected, EPC may be mistaken for HFS.

Case Presentation: A 79 y.o. woman with a history of left HFS presented with a right MCA acute ischemic stroke and facial twitching. An initial EEG revealed 5 seizures and she was loaded with phenytoin. Her spells continued and levetiracetam was added to phenytoin. She was placed on cEEG monitoring and a total 187 spells of left facial contractions were recorded over 2 days. Her spells did not improve despite aggressive treatment with levetiracetam, phenytoin, and lamotrigine.

Discussion: HFS is a form of segmental myoclonus of muscles innervated by the facial nerve, while EPC is a form of focal motor status epilepticus that is most commonly seen in adults following stroke. Usually EPC consists of continuous focal jerking of a limb, but when the facial muscles are affected, EPC may be mistaken for HFS. Only 1 case describing chronic isolated HFS as a possible manifestation of EPC was identified after a literature review.

Conclusion: This case involved a woman with chronic HFS who presented following an ischemic stroke with stereotyped spells of left hemifacial contractions. Although her presenting history was suggestive of EPC, it was difficult to ascertain a definitive diagnosis in light of her history of left HFS. Her course did not improve with aggressive seizure medication trials and after further review of her neuroimaging, there was an extensive right MCA stroke with little preservation of cortical tissue. In light of these findings it was felt that these spells were less likely seizures and more consistent with HFS.

F15
Adult Onset Electrographic Status Epilepticus of Sleep
Ursula Thome MD; Paula Klima MD; Ahsan Moosa MD; Ajay Gupta MD; Elia M. Pestana Knight MD

Continuous Spike and Wave during Sleep is a childhood epileptic encephalopathy defined by the presence of Electrographic Status Epilepticus of Sleep (ESES) in the EEG, seizures and neurocognitive/behavioral changes. Reports of adult patients with ESES are rare in the literature. We report a 27 year-old woman with history of childhood onset epilepsy who developed cognitive decline and worsening of seizures at age 21 years. Seizures increased progressively to daily. In the last 2 years, she had neurological regression in language, motor skills and cognition. She also developed excessive daytime sleepiness. Video-EEG monitoring showed rare spikes when awake and ESES pattern during sleep (Figure 1A and B). Continuous spike and wave discharges were present in more than 85% of slow waves sleep. EEG improved after treatment with oral diazepam 20mg at night. She became more alert and her speech improved.

Our case and the ones reported in the literature indicate that ESES can be a cause of neurocognitive, behavioral and motor decline in adults. Recognition of the syndrome in adults needs to be encouraged to provide adequate and effective treatment.

F16
A case of Tuberous Sclerosis Complex with First Clinical Presentation in her 40s.
Thandar Aung, MD

Intro: TSC is initially recognized as childhood disease. We present a 45-year-old woman, with normal intelligence and no personal or family history of TSC, presented with this neurocutaneous disorder.

Case: A 45-year-old woman presented after experiencing first-time unprovoked complex partial seizures. In ER, she had another seizure. Benzodiazepine and phenytoin were given intravenously. She returned to her baseline. She had two small nodules on the left iris, a nodule on the nose and several ash leaf spots. Brain imaging showed scattered areas of dystrophic coarse parenchymal calcification in the subependymal region and in the cerebellar hemisphere. Innumerable bilateral subcortical and cortical T2/Flair hyper intensities (cortical tubers) were found which consistent with TSC according to the diagnostic criteria of Roach et al., 1998.

Seizure was controlled with one AED. Nothing was found on her liver, kidney and heart. TSC2 gene was found to be mutated.

Diss: TSC is a neurocutaneous autosomal dominant genetic disorder with an incidence of approximately 1 in 5000 to 10,000 live births. Diagnosis of TSC in adult can be difficult because of variable expressivity and incomplete penetrance. Two thirds of TSC cases result from sporadic genetic mutations in TSC1 or TSC2 but their offspring may inherit it from them. Neurological presentation of tuberous sclerosis occurs typically in children with seizures and intellectual impairment. However approximately 50% of patients who fulfill the diagnostic criteria have normal intellect and 15% remain free from seizures. The exceptionally mild disease
in our patient may simply be an example of variation in expression characteristic of
tuberous sclerosis observed between affected members of the same family or due
to somatic mosaicism where a postzygotic new dominant mutation only affects a
proportion of cells and to a varying extent in different tissues.

Results: 10 patients met the criteria. Age range was 5-18 years, seizure onset
ranged from 1 to 17 years of age. 2 had seizures originating from the frontal
lobe, 4 from the temporal region, 2 from the occipital region and 2 from the
parietal area. 7 patients had seizures originating from the left hemisphere, while 3
originated from the right.

We did not find a statistically significant difference between the symptomatic
hemisphere and lobe and the control hemisphere and lobe when hemispheric volume,
lobar volume, volume of the thalami, volume of gray or white matter were compared.

Conclusion: We will perform further data analysis and enroll more patients in this
study to find out whether volumetric analysis could be useful as a marker of focal
epilepsy and correlate this with clinical presentation.

F19
A Case of Status Epilepticus in Adult from Post-viral Influenza A H1N1 Vasculitis
Thandar Aung, MD

Introduction: Influenza-related neurologic complications are rare, especially in
immunocompetent adults.

Case: A 57-year-old female, with past medical history of atrial fibrillation and
hypertension, was admitted to hospital with H1N1 influenza pneumonia. Hospital
course was complicated by secondary bacterial pneumonia, ARDS, and bilateral
DVTs with pulmonary embolism for which she required tracheostomy with full
ventilation support. She was transferred to long term care facility after receiving
intensive treatment for six weeks. Two weeks later, she experienced first new
onset seizure which progressed into SE. Brain imaging showed diffuse subcortical
edema throughout the brain descending of 4mm cerebellar tonsil through foramen
magnum. The diffuse effacement was seen along the perivascular spaces which
followed the pattern of vasculitis. CSF analysis showed unremarkable including viral
studies except from nucleated cell count of 3/ul with elevated protein of 97.6 mg/
dl. Electroencephalogram showed multi-Focal spike wave discharges. IV high dose
steroid was started. Her mental status clearly improved on two epileptic medications
and repeated MRI showed complete resolution of edema.

Discussion: This is a first case report on H1N1 post viral related CNS vasculitis in
adult. Febrile seizure was reported as most common influenza-related neurologic
complication in pediatric population. In adult, there are case reports regarding
influenza associated with encephalopathy, meningoencephalitis, transverse myelitis,
and Guillian-Barre syndrome. However, the pathogenesis of the CNS illnesses
associated with influenza remains poorly understand. In our case, patient developed
post viral CNS vasculitis after eight weeks of first symptoms which resolved with
steroid. The presence of inflammation with negative influenza assay has led to the
view that inflammation is of post-viral autoimmune in origin.

F20
Usefulness of 3T MRI in Detecting Lesion in Medically Refractory Epilepsy
Patients
Deepal Shah, MD; Abuhuziefa Abubakr, MD

Rationale: To show that 3T MRI with thick slices and high interslice gap has a higher
failure rate in detecting lesions and emphasize the need to use thin slices (1-1.5
mm) and no interslice gap in every institution.
**POSTER ABSTRACTS**

Method: Retrospective review of medical records of medically refractory epilepsy patients who underwent surgery at UMC between 2012 and 2013. Review their MRI (3-4 mm thickness, 3-4 (3.5) mm slice spacing) and pathology findings. Primary objective to ascertain percentage of correlation between MRI and pathology findings.

Results: Out of 15 patients who underwent surgery, 14 patients had lesional MRI. In 8 out of 14 patients (57.14%), there was no correlation between their MRI and pathology. In 6/8 patients (75%), diagnosis was missed (2 FCD, 2 MTS, 2 FCD+MTS). 2/8 patients (25%) were misdiagnosed (HS instead of FCD, FCD when there was no FCD on pathology).

Conclusions: Despite small sample size, study shows that MRI (3-4 mm thickness, 3-4 (3.5) mm slice spacing) has a high failure rate > 50% in identifying lesions correctly, which indirectly affect identification of potential surgery candidates. Therefore, we recommend the universal use of standard 3T MRI epilepsy protocol with 1-1.5 mm thin slices with no interslice gap.

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**F21**

The Cessation of CSWS Following a Temporal Lobectomy

Brian D. Moseley, MD; Radhika Dhamiya, MD; Elaine Wirrell, MD

Introduction: The role of respective surgery in epilepsies with generalized EEG patterns such as continuous spike wave in slow wave sleep (CSWS) has not been robustly explored. We report a case of CSWS that was secondary to focal pathology and treated with surgery.

Methods: Case-report/literature review.

Results: An 11-year-old boy presented with medically refractory focal onset seizures since the age of 2 years. At age 10, he developed decreased attentiveness and increased aggressiveness. Based on his clinical picture and an EEG demonstrating electrical status epilepticus in slow wave sleep (ESES), he was diagnosed with CSWS. This failed to respond to high dose diazepam.

MRI revealed prior right basal ganglia and thalamic infarcts and right mesial temporal sclerosis. During repeat EEG monitoring, he had nearly continuous generalized spike and wave discharges during sleep. He had one seizure of right midtemporal onset. He underwent a right temporal lobectomy. Postoperatively, he was seizure free, with improved attention and behavior. Repeat EEG revealed no further ESES.

Conclusions: Our case provides evidence that epilepsy syndromes with generalized discharges can be secondary to focal pathology amenable to surgery. The postoperative normalization of his sleep EEG suggests temporal lobe structures are involved in the network generating CSWS.

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**F22**

The Correlation of Epileptiform Discharges in Sleep with Ictal Onset in Schizencephaly

Marjan Dolatshahi, MD; Alexei Yankovsky, MD; Marcus Ng, MD

Schizencephaly is a common developmental cause of epilepsy. In most cases, the cortical structural abnormalities may be diffuse or multifocal. When refractory to medication, delineating the zone of ictal onset for the purpose of epilepsy surgery may be challenging. Sleep has shown promise in localizing the zone of ictal onset in other diffuse and multifocal developmental epileptogenic conditions, such as tuberous sclerosis. We sought to determine the impact of different stages of sleep on the electrographic extent of epileptiform discharges in schizencephaly patients. Then we sought to correlate the zone of ictal onset with the extent of these epileptiform discharges in sleep. In a retrospective chart review, we found 9 patients with open-lip or closed-lip schizencephaly and associated polymicrogyria. We will present the fields of epileptiform discharges in various stages of sleep and wakefulness, zones of ictal onset, and radiographic abnormalities for each patient. We will interpret these findings in relation to the role of sleep in predicting the zone of ictal onset for these patients.

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**F23**

Comparison of Transcranial Electrical Motor Evoked Potentials (TcMEPs) from Different Hand Recording Montages

Ricardo Bravo, MD; Laurence McKinley, MD; Jaime R. López, MD, FACS

TcMEPs are widely used for direct monitoring of corticospinal motor pathways in a variety of surgical procedures. However, there is no published recording technique indicating the optimal electrode placement for recording myogenic responses from the hand. The purpose of our study was to compare three different hand recording montages recorded simultaneously. Data from 12 patients undergoing a variety of thoracic or lumbar spinal column surgeries were prospectively collected. TcMEPs from each hand were obtained using standard stimulation techniques. All patients underwent some anesthesia regimen. Simultaneous recordings were acquired from the following electrode sites, using subdermal needle electrodes: APB-1stDIO, APB-ADM, and APB-thumb. TcMEP peak-to-peak amplitudes were analyzed for each recording montage. APB-1stDIO had the largest average amplitude on either hand, left-1811.3 µV (69.1-9963.2 µV), right-1974.8 µV (40.4-7688.1 µV); followed by APB-ADM, left-1651.2 µV (63.5-8029.5 µV), right-1864.4 µV (54.2-7867.7 µV); and lastly APB-thumb, left-1341.2 µV (70.9-5230.2 µV), right-1799.9 µV (39.5-7334.6 µV). However, paired t-test between each montage showed no significant differences. Although the findings are not statistically significant, the results may be influenced by the wide range of amplitudes and the relatively low number of patients studied. Further study is warranted to clarify if a particular recording montage is superior.

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**F25**

Increased Spinal Cord Compression from Patient Positioning Detected by Intraoperative MEP and SEP Monitoring

Alan D. Legatt, MD, FACNS; Jonathan Nakhla, MD; Michael A. Weicker, MD; Reza Yassari, MD

A 42-year-old woman with persistent back pain had sudden-onset left leg weakness and numbness. MRI showed extensive osseous destruction at L2-L3 with a large epidural abscess causing severe thecal sac compression. The patient was positioned prone for laminectomy, decompression, and spinal instrumentation/fusion. Large tibialis anterior and abductor hallucis MEPS and posterior tibial nerve SEPs were present initially, but deteriorated and disappeared bilaterally during the soft-tissue dissection; upper-limb MEPS and SEPs were unchanged. The surgeons were notified and performed the laminectomy before placing the pedicle screws, the reverse of the usual sequence. When the laminectomy was done, the lower-limb MEPS and SEPs reappeared and recovered fully to baseline. MEPS and SEPs were then stable during washout of the abscess and the instrumentation/fusion. Postoperatively, the patient recovered full
strength in the left leg and her sensory exam was improved. The pathologic diagnosis was Poit's disease. Changes in spinal curvature with positioning on the operating table most likely increased the spinal cord compression in this patient, leading to the loss of lower-limb MEPs and SEPs. The neurophysiologic monitoring findings led to performance of the laminectomy about 90 minutes earlier than it would have otherwise been done, potentially contributing to the good neurologic outcome.

F26
Somatosensory and Transcranial Motor Evoked Potential Changes During Sitting Position Craniotomy
Ana Tercero, MD; Josep Gonzalez, MD; Eliseo Jorge Torales, MD; Ricard Valero, MD; Joan Santamaria, MD

Surgical procedures of the posterior cranial fossa are preferably performed with the patient in the sitting position and somatosensory evoked potentials ( SSEP) and transcranial motor evoked potentials (t-MEP) are usually recorded in these cases. Nonspecific SSEP and t-MEP changes have been reported. Posterior tibial nerve SSEP changes have never been reported before. We analyzed median and posterior tibial nerve SSEP and t-MEP changes of 7 patients who were operated in a sitting position. Median and posterior tibial nerve SSEP amplitudes decreased in 6 of them. The partial loss of posterior tibial nerve SSEP amplitude was always seen in the Cz-Fz channel but curiously it increased in the Cz-Fz channel. MEP changes were observed only in 4 of 7 patients. These changes were not related with neurological impairment and all of them appeared few minutes after opening the dura mater. We recommend to record SSEP at least in two cortical channels and emphasize the need to be cautious with the interpretation of certain SSEP or MEP changes during surgeries performed in sitting position because they could not be associated with postoperative neurological deficits.

F27
Intraoperative Monitoring of Simultaneous Cases: A Risk-Based Approach Utilizing Monte Carlo Simulation
Stephen Fried, MD

Although utilization of Intraoperative monitoring continues to grow, the number of reading physicians is limited, often necessitating the monitoring of simultaneous cases. While risk grows as the number of simultaneous cases increases, how can that risk be quantified? To evaluate the risks involved, our model asks: For N simultaneous cases, given the probability (P) of a significant change in any case at any given time, and amount of time (T) that increased attention needs to be paid to a case once a change occurs, what is the likelihood (L) that any two time periods will overlap with concurrent changes requiring increased attention? A Monte Carlo simulation (one million trials) was performed for P=0.1, 0.15, and 0.2, using T=60 minutes as well as T=Time until case ends (Tmax). A value of 1% of time monitored was set as the threshold for L. For T=60 minutes, L crosses threshold at N=8, 5, and 4 cases for the three values of P respectively. For T=Tmax, L crosses threshold at N=4, 3, and 2 cases respectively. This model may be useful to evaluate the risks involved with most commonly monitored procedures, which require straightforward evoked response evaluation, but would be limited in more complex monitoring cases.

F28
The Off-label Use of External Cardiac Pacemaker Electrode for D-wave Intraoperative Monitoring: A Common Practice in Economically Developing Countries
Paulo A. Kimaid, MD; Rafael De Castro, MD; Charles M. Nascimento, MD; Rodrigo N. Cardoso, MD; Rinaldo Claudino, MD; Marcondes Franca, MD

Background: D-wave intraoperative monitoring is accessed by an epidural electrode (EE) which costs 3 to 4 times more than an external cardiac pacemaker electrode (ECPE). Considering also the similarity of their technical properties, ECPE is being largely used instead of EE in economically developing countries.

Purpose: The aim of our study is to present D-wave data obtained with the off-label use of ECPE in cases of spinal tumor surgery.

Methods: Ten patients with spinal tumor were submitted to tumor resection under intraoperative monitoring with our standard protocol for spinal tumors: upper and lower limbs SSEP and tc-MEP with muscle and epidural registration. We used the external cardiac pacemaker electrode to register D-wave, positioned under direct vision in the midline, distal to the tumor, instead of the EE.

Results: In 8 cases we could register an easy to identify D-wave. In 2 cases, as we couldn’t obtain the traces we decided to exchange the ECPE for an EE in order to exclude any technical problem, but D-wave still could not be registered.

Conclusion: D-wave monitoring can easily be registered with ECPE reducing the costs of IONM without detriment to the technique. Despite the small number of cases, our data agreed with previous reports warning that D-wave can be absent in 20% of spinal tumor patients.

F29
Correlation Between Bispectral Index and the Quality of the Electroencephalography During Epilepsy Surgery.
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Introduction. The Electroencephalography (ECoG) is useful to identify the epileptic zone during epilepsy surgery and the Bispectral Index (BIS) allow the hypnotic anesthesia component monitoring, however, the correlation between the scores of BIS and the ECoG patterns to optimizing the quality and time of the ECoG recordings is unknown. Objective. Analyze the correlation between the BIS scores and the duration of suppressions periods (seconds) in the burst-suppression (BS), background frequency (Hz) and type of patterns (1 [normal] to 5 [ECoG seizure]; Bindro A et al., 2012) of ECoG recordings during epilepsy surgery under intravenous general anesthesia with propofol. Material and Methods. Prospective study that included consequently pharmacoresistant epileptic patients who underwent epilepsy surgery guided by ECoG and BIS (September 2008 to October 2012). Results. We included 28 epileptic patients, 15/28 (53.5%) female, age mean 30.5 (13-56) years old, weight mean 68.32 (42-100) kg who underwent 22/28 (79%) temporal and 6/28 (21%) extra temporal epilepsy surgeries with propofol mean plasmatic concentration 3.2 (0.75-4.4) µg/ml and ECoG duration mean 40 (5-178) min. We found on a non-linear relationship (e.g. polynomial cubic) between the mentioned variables by emphasizing that for a BIS range 40-60 the following characteristics follow: ECoG burst suppression periods below 5 s, background brain
F30
Intraoperative MEP Monitoring Beyond the Aortic Clamp Period of Open Thoracoabdominal Aneurysm (TAA) Repair Further Decreases the Risk of Postoperative Paraplegia
Oluwale Awosika, MD; Reiner B. See, MD; Richard P. Cambria, MD; Mark F. Conrad, MD; Virendra I. Patel, MD; Glenn M. LaMuraglia, MD; Rae Allain, MD; Mirela V. Simon, MD, FACS

Motor evoked potentials (MEP) monitoring promptly detects reversible spinal cord ischemia directly related to clamping of the aorta in open thoracoabdominal aneurysm (TAA) repair with Atri-femoral (A-fem) bypass. We hypothesized that extension of MEPS monitoring beyond aortic clamp period (ACP) further decreases the risk of postoperative paraplegia. Methods: We identified 120 patients who underwent open TAA with A-fem bypass and MEPS monitoring at MGH, between Jan 2008-Dec 2012. Using a multivariate logistic regression analysis, we studied the independent effect of MEPSs monitoring (ie predictor of interest) beyond ACP, the risk of developing postoperative paraplegia (ie outcome). We used a propensity score analysis to adjust for the potential impact on the outcome of other factors such as presence of acute Aortic dissection, urgent splenectomy, TAA Type, age >65 years, previous TAA surgery, history of poorly controlled hypertension, diabetes, smoking. Results: From 120 patients, 89 (74%) did not have monitoring extended beyond ACP, while 31 patient (26%) did. The proportion of patients who had acute splenectomy was significantly higher in the group which did not receive extended MEPSs monitoring (96.88% vs 3.13%, p < .0001). Other potential predictors of post-operative paraplegia were present in similar proportions within the two cohorts. MEPs monitoring beyond ACP independently decreased the odds of developing postoperative paraplegia, after adjusting for the other potential predictors (OR=.011, CI95 [0.001, 0.92] p=.04). Conclusions: By allowing titration of the systemic blood pressure to ensure appropriate spinal cord perfusion in the immediate postoperative period, extension of MEPSs monitoring beyond ACP during open TAA repair with A-fem bypass, further reduces the risk of postoperative paraplegia.

F31
Spinal Intradural Tumours: A Single Center Experience
Lidia Cabanes, MD; Ignacio Regidor, MD; Gema de Blas, MD; Federico Abreu, MD; Rodrigo Carrasco, MD; Marta del Alamo, MD; Luis Ley, MD

Question: Primary spinal cord tumours represent 2% to 4% of all central nervous system neoplasms and are anatomically separable into two broad categories: intradural intramedullary and intradural extramedullary. We present our experience treating this kind of tumours in the last 10 years, with an especial focus on the use of intraoperative neurophysiologic monitoring (IOM).

Methods: We have performed a retrospective study within our institution, from an epidemiologic, clinical, radiologic and surgical point of view, including the use of IOM.

Results: A total of 93 patients, ages between 22 and 81 years old. The most frequent clinical presentation was motor deficit and pain. There was great histological variation, but neurinomas, meningiomas and ependymomas were more frequent. Multimodal IOM was used in 41% of the cases. We observed that in the group of monitorized patients the rate of neurological sequelae was lower that in the non-monitorized group.

Conclusions: The use of IOM in primary intradural spinal cord tumours reduces the incidence of neurological complications. IOM can identify neurological injury with excellent sensitivity.

F32
Utility of Lumbosacral Pedicle Screw Electrical Stimulation in a Mexican Community Hospital
Alvaro A. Zavala, MD; Sergio Aguilar, MD; Samantha Pineda, MD

Neurological injury incidence during lumbosacral fusion with pedicle screws can range up to 15%. Injury results from a breach of the pedicle wall potentially injuring the lumbosacral roots. The aim of our study was analyze the usefulness of lumbosacral pedicle screw stimulation using 1.5 mA as “warning threshold” in a Mexican nongovernmental institution. We stimulated 608 titanium lumbosacral pedicle screws in 125 patients, 56 males, 69 females, ages 25-85 years, using monopolar, cathodal, constant voltage stimulation. Balanced or total intravenous anesthesia was used. Of the 608 pedicle screws, 71 had thresholds below 1.5 mA, each one was redirected, with re-stimulation levels above 15 mA in 67 and between 12 and 14 mA in 4. Postoperative CT scans were done in all patients showing: medial wall breach of two screws (both patients asymptomatic), 3 screws malpositioned laterally. The four screws between 12 and 14 mA thresholds remained in place. Four false positive and 5 false negative cases were seen, with a negative predictive value of 0.991. Our experience shows the importance of intraoperative testing of pedicle screw placement, the feasibility of performing this possibility of doing in middle income countries, with comparable results to those found in more experienced centers.

F33
EEG Patterns During Deep Hypothermic Circulatory Arrest: Utility of qEEG
Abeer J. Hani, MD; Aatif M. Husain, MD, FACNS

Objective: To determine the temperature and time course of EEG patterns during deep hypothermic circulatory arrest (DHCA) and to assess utility of quantitative EEG (qEEG) in determination of burst suppression (BS) and electrocerebral inactivity (ECI).

Methods: The charts of 10 patients undergoing DHCA during aortic surgery were reviewed. The onset of periodic discharges (PD), BS and ECI was determined. Using qEEG analysis, BS during cooling was defined as a suppression ratio of about 90% and ECI was defined as suppression ratio of 97-100%.

Results: The mean nasopharyngeal temperature when PD appeared was 25.9 ± 1.1oC, BS appeared at 23.0 ± 0.8oC, and ECI appeared at 17.1 ± 0.7oC. During rewarming BS was seen at 20.8 ± 0.6oC, and baseline EEG activity returned at 28.8 ± 1.2oC. Using qEEG, ECI could be determined about 11.4 ± 3.2 minutes earlier than when using raw EEG.

Conclusion: The temperatures at which the various EEG patterns were observed are similar to that of previous studies. Use of quantitative EEG may assist in standardizing the time of determination of ECI.
F34
Utilization of Free Running EMG and SSEP Recordings for Spinal Cord Stimulator Placement and Optimization Under General Anesthesia
Emily B. Kale, BS, CNIM; Aatif M. Husain, MD, FACNS; Lindsay Rambeaut, MD

Introduction: Spinal cord stimulator (SCS) placement under general anesthesia (GA) reduces the means by which SCS leads can be ideally placed and optimized since patient feedback is unavailable. Neurophysiologic recordings taken before, after, and during lead placement and activation are complementary and often a more sensitive means of SCS lead placement and optimization.

Methods: Ten thoracic SCS leads were placed using tibial and ulnar somatosensory evoked potential (SSEP) recordings and free running electromyography (EMG) recordings to aid in lead placement and optimization. Lead placement over the midline of both dorsal columns was determined by radiography and bilateral activation of EMG channels in the lower extremity. SSEP recordings were taken before, during and after lead activation to assess optimal interruption of sensory input during lead activation.

Results: EMG findings were complimentary to radiographic images, but indicated that small adjustments should be made to SCS lead placement to maximize activation of both dorsal columns. SSEP recordings taken before during and after lead activation demonstrated interruption of sensory pathways with decreased amplitude and increased latency or total interruption of sensory signals.

Conclusion: SSEP and EMG recordings taken before, after, and during lead placement and activation are complementary and often a more sensitive means of SCS lead placement and optimization.

F35
NIOM for Spinal Cord Stimulator Placement - Detection of an Unusual Complication
Jose Devesa, MD; Emily B. Kale, BS, CNIM; Aatif M. Husain, MD, FACNS

Introduction: Neurophysiologic intraoperative monitoring is used during spinal cord stimulator (SCS) implantation to optimize lead placement. The lead is placed epidurally, and injury to the spinal cord is unlikely. We present a case of an unusual complication during SCS placement.

Case Report: A 57-year-old male presented for revision of a thoracic SCS due to lead fracture. No motor or sensory deficits were noted prior to surgery. During the procedure lower extremity electromyography (EMG) and ulnar and tibial somatosensory evoked potentials (SEP) collision technique were used for optimal lead placement. After lead placement, SEP were monitored, and during closure a significant amplitude decrease of both cortical tibial SEP was noted. Significant motor deficits were noted in both lower extremities after the patient was awakened. The patient was immediately re-intubated SCS lead removed. Thereafter strength in both lower extremities improved.

Conclusion: In addition to assisting with optimal lead placement during SCS implantation procedures, NIOM may detect inadvertent injury to the spinal cord.

F36
Variability of Motor Evoked Potentials Under Steady-State Anesthesia During Scoliosis Surgery: Are All Muscles the Same?
Stephen Fried, MD; Alan D. Legatt, MD, FACNS; Diane Smith, MD

Intraoperative monitoring of corticospinal tract motor pathways with motor evoked potentials (MEPs) reduces the likelihood of neurological deficits following scoliosis surgery. The most commonly used warning criterion for MEP changes is a significant amplitude drop, though what constitutes “significant” is not universally agreed upon and may, in fact, be different in different muscles. We compared the run-to-run variability of MEPs across three muscle groups (thenar/hypothenar, tibialis anterior, and abductor hallucis), recorded under steady-state propofol infusion during 30 surgeries for idiopathic scoliosis in which MEPs remained present throughout and there were no post-operative neurological deficits. For each muscle group, there was no significant difference in the coefficient of variation (CV) between the left-sided and right-sided MEPs (Wilcoxon rank-sum). The thenar/hypothenar MEPs showed the greatest variability, with an average CV of 48%. This was significantly larger than the average CV within the abductor hallucis MEPs, 35% (p<0.001). This, in turn, was significantly larger than the average CV within the tibialis anterior MEPs, 27% (p<0.001). We conclude that, due to differences in run-to-run variability across muscle groups, MEP warning criteria might best be tailored to the particular muscle group being monitored.

F37
Success Rate of Obtaining Baseline SEP & MEP in Consecutive Cranial and Spinal Surgeries
Brian S. Droker, MD; Andres A. Gonzalez, MD, MMM, FACNS; Parastou Shilian, DO

Introduction: Intraoperative monitoring (IOM) has been documented as an adjunctive technique used to minimize neurologic deficit during brain and spinal surgeries. IOM is predicated on the patient acting as an internal control, and that obtaining reliable baseline potentials is essential for effective monitoring. Previous studies reported success rates in MEP as low as 66.6% in the lower extremities and greater than 93% in spinal surgeries for idiopathic scoliosis in which MEPs remained present throughout and there were no post-operative neurological deficits. For each muscle group, there was no significant difference in the coefficient of variation (CV) between the left-sided and right-sided MEPs (Wilcoxon rank-sum). The thenar/hypothenar MEPs showed the greatest variability, with an average CV of 48%. This was significantly larger than the average CV within the abductor hallucis MEPs, 35% (p<0.001). This, in turn, was significantly larger than the average CV within the tibialis anterior MEPs, 27% (p<0.001). We conclude that, due to differences in run-to-run variability across muscle groups, MEP warning criteria might best be tailored to the particular muscle group being monitored.

Methods: Chart review of consecutive cranial and spinal cases from January 2010-2011. SEP and MEP baselines were after initiation of general anesthesia and before skin incision. Anesthesia consisted of TIVA or TIVA with halogenated agents with less than 0.5 MAC. Outcome measures: Primary: Success rate in obtaining SEP and MEP in all subjects. Secondary: Rates by preoperative surgical diagnosis.

Results: 695 cranial and spinal cases that required IOM were reviewed. Greater than 90.1% success rate in both SEP and MEP. For cranial surgeries the success was more than 93%. In spinal surgeries, a similar success rate was seen in deformity and degenerative cases. However, a decrease in success rate was noted in patients with either trauma or infection.

Discussion: Our success rates were higher than previously reported. Success rates in spinal surgery cases were lower than previously reported; perhaps reflecting the disease process. Though preexisting deficits are associated with reduced success rate, preoperative diagnosis seems to serve as an indicator as well.

Conclusions: Success rate of obtaining baselines is higher than previously reported. The preoperative diagnosis may predict the success rate. With this information, the
**F38**

**Laryngeal TCMEP Technique: From the EMG Lab to the OR**

Paulo A. Kimaid, MD; Rafael de Castro, MD; Charles Nascimento, MD; Rodrigo N. Cardoso, MD; Rinaldo Claudino, MD; Marcondes Franco, MD

Background: Laryngeal electromyography (LEMG) is a routinely and valuable technique to evaluate diseases of laryngeal nerves and muscles in the EMG laboratory (EMG LAB). The technique of percutaneous approach of laryngeal intrinsic muscles is easy to apply and uses cheap needle electrodes.

Purpose: The aim of this study is to describe the technique we adapted from EMG LAB to register corticobulbar laryngeal motor evoked potentials (CoLMEP) in the vocalis muscle complex (VOX) after transcranial electric stimulation (TES).

Methods: A retrospective review of 30 cases of cerebellopontine angle or brainstem tumors undergoing tumor resection with our standard protocol of intraoperative monitoring: upper and lower limbs SSEP and TCMEP, free-running EMG of muscles innervated by cranial nerves V, VII, IX, X, XI e XII, updated with a protocol of cortical motor evoked potentials registered in oribucularis ori (ORI), vocalis muscle (VOX), trapezius muscle (TPZ) and tongue were studied.

Results: In all cases we used the adapted technique with VOX percutaneous approach and it was easy to identify CoLMEP after TES.

Conclusion: The proposed technique is trustful and can be easily reproducible. It is also cheaper than the previously described techniques. In our opinion it should also be tested in Direct Cortical Stimulation.

**F39**

**Utility of Beamformer Source Analysis in Clinical MEG**

Paul Ferrari, MD; Douglas Cheyne, MD; Mark Mcmanis, MD; Mark Lee, MD; Dave Clarke, MD; Fredrick F. Perkins, MD

MEG beamformer source localization provides a high temporal- and spatial-resolution source estimation of brain activity and has the special property of being robust in the presence of interfering noise. While the beamformer’s use in mapping epileptiform activity and eloquent cortex has been demonstrated the technique has yet to be widely validated for clinical applications. We first show, through simulation, that event-related beamformer reconstruction accurately localizes transient activation at various locations within the brain, even at low signal to noise. We then present specific case studies demonstrating the utility of the beamformer method in practice, applied to datasets where various sources of interfering noise distort or preclude the equivalent current dipole model. We subsequently show examples of how source level multivariate spatiotemporal decomposition can be applied to enhance our description of multifocal epileptiform discharges, reveal hyperactive epileptiform networks during quiescent resting state, and also provide an intuitive analysis of event-related language late-fields for assessing language laterality. We conclude that the MEG beamformer, used judiciously, has a unique and complimentary role in clinical MEG analysis.

Cheyne D, et. al., Clin Neurophysiol. 2007;118(8):1691-704

Rose DF. et. al., Front Neurol. 2013;4:56


**F40**

**Low and High Frequency Oscillations Reveal Distinct Absence Seizure Networks**

Jeffrey Tenney, MD; Hisako Fujiwara, MD; Paul Horn, MD; Jennifer Vannest, MD; Jing Xiang, MD; Tracy Glauser, MD; Douglas F. Rose, MD

Objective: The aim was to determine the frequency-dependent, spatiotemporal involvement of corticothalamic networks to absence seizure generation.

Methods: Magnetoencephalography was recorded in 12 subjects (44 seizures) with untreated childhood absence seizures. Time-frequency analysis of each seizure was performed to determine significant power at ictal onset. Source localization identified regions contributing to generalized spike and wave discharges (SWDs).

Results: Significant power was seen in 1-20 Hz, 20-70 Hz, and 70-150 Hz bandwidths. Sources localized to the frontal cortex similarly for the low and gamma frequency bandwidths, while at the low frequency bandwidth (3-20 Hz) significantly more sources localized to the parietal cortex (OR=16.7) (Fig 1). Cortical high frequency oscillations (HFO) (70-150 Hz) localized primarily to the frontal region compared to the parietal (OR=7.32) or temporal (OR=2.78) areas.

Interpretation: Neuromagnetic activity in frontal and parietal regions confirms hemodynamic changes reported using functional MRI. The frequency dependent nature of these networks has not previously been reported and the presence of HFOS during absence seizures is a novel finding. Co-occurring frontal and parietal corticothalamic networks may interact to produce a pathological state which contributes to SWDs.

**F41**

**Proximal Median Neuropathy a Complication of Reverse Shoulder Arthroplasty**

Reiner B. See, MD; Bashar Tahiri, MD; David C. Preston, MD; Barbara Shapiro, MD

Reverse shoulder arthroplasty (RSA) is indicated for rotator cuff-tear injury. It reverses the physiologic ball and socket, resulting in distal displacement of shoulder joint’s centre of rotation, increasing the lever arm of deltoid muscle for more muscle fiber recruitment for shoulder elevation & abduction. A 72 y/o lady, underwent left RSA surgery, 10 days after surgery developed left hand grip weakness, EMG study revealed left proximal median mononeuropathy at/above the pronator teres muscle. Mechanism likely from traction injury, demonstrated on a 3D computerized model that calculated strain after prosthesis placement (VanHoof et al). Results show median nerve’s medial (19%) and lateral (15%) root strain.

To the authors’ best knowledge, this case is the first report of proximal median mononeuropathy from RSA.

**F42**

**Hypoglossal Nerve Palsy is Associated with a Variety of Etiologies and Sometimes Results in Tongue Myokymia**

Amro Shino, MD; Benn Smith, MD

We present a case series of 7 patients seen at the Mayo Clinic in Arizona over a 27 year period diagnosed with hypoglossal nerve (CN XII) palsy. Three were radiation induced, with one patient having CN V + XII involvement, a second having CN XI + XII involvement, and a third showing isolated disease of CN XII. All three had myokymia on tongue EMG. Vascular causes ranked second, with one patient developing CN XI + XII palsy from an internal carotid artery dissection and a second developing hypoglossal nerve palsy from medulla compression by an
enlarged vertebral artery. One patient presented with a unique acute onset case attributable to a synovial cystic compression of the right hypoglossal canal coupled with congenitally narrowed hypoglossal canals in the setting of platybasia. Of note, no such case has been previously reported to our knowledge, including in a case series of 100 patients by Keane et al (1996). One case remained indeterminate with regards to etiology. Most patients presented either in the setting of a known cancer, prior external beam radiation or as chronic headache with imaging diagnostic of vascular etiologies.

F43
Idiopathic Peripheral Neuropathy and Subsequent Diabetes Mellitus
Ritika Mahajan, MD; Molly King, MD; Madeleine M. Grigg-Damberger, MD, FACS

Objectives: Determine how often and when patients with idiopathic sensorimotor axonal neuropathy (IPN) confirmed by nerve conduction studies (NCV) will subsequently develop diabetes mellitus type-2 (DM2).

Methods: Retrospectively searched electronic medical records of NM VA Health Care System from 1995-2013 for veterans diagnosed with IPN by history and NCV. Collected Hemoglobin A1c (HgbA1c) near time of initial diagnosis, and subsequent visits until 2013. Using 2010 American Diabetes Association guidelines, classified HgbA1C 5.7-6.4% as prediabetic, > 6.5% as diabetic. Excluded veterans whose initial HgbA1c was >6.5%, taking antidiabetic medications, or developed another IPN cause.

Results: 283 veterans initially diagnosed with IPN (median age 65 years, 95% men). When diagnosed, 156 (55.1%) were normoglycemic, 127 (44.9%) prediabetic. On followup 36.5% initially normoglycemic became prediabetic, 10.3% diabetic, 37.2% normoglycemic, and 16% no data. 18.9% of initially prediabetic became diabetic, 54.3% remained prediabetic, 7.9% normoglycemic, and 18.9% no data. Kaplan-Meier analysis showed 46.8% with IPN became prediabetic or diabetic in a median of 6.5 years.

Conclusion: Nearly half of military veterans with IPN will develop prediabetes or diabetes within a median of 6.5 years. Lifestyle modifications and surveillance are needed to reduce risk for subsequent DM2 in IPN.

F44
Sonography in the diagnosis of Neurolymphomatosis
Joy Vijayan, MD

Nerve ultrasoundography is a sensitive and useful investigative tool in the evaluation of neuropathic symptoms in patients with a diagnosis of Lymphoma. Neurolymphomatosis is an uncommon clinical complication of Non-Hodgkin’s Lymphoma and is characterized by infiltration of lymphomatous cells into the peri- and endoneurium of peripheral nerves. Confirmation of a clinical diagnosis is based on MRI and PET scan studies which are followed by invasive histopathological studies. We present three patients with a history of lymphoma who were seen in the Neurodiagnostic Laboratory for electrodagnostic evaluation of presumed radiculopathies. Electrodiagnostic studies proved involvement of the peripheral nerves. Ultrasonography showed nerve thickening with increased blood flow in those sites which were electrodagnostically abnormal. Further histopathological studies confirmed diagnosis of neurolymphomatosis. Nerve ultrasoundography is a useful tool in evaluating neuropathic symptoms in patients with Lymphoma.

F45
An Alternative to Collodion
Esperanza E. Wagner, MD

Purpose: Improve Electrode Application for Long-Term EEG/Video Monitoring without using Collodion.

Method: Prepare the skin as usual. Cut gauze into one inch by one inch squares. Fill a 10 mm electrode cup with a conductive paste (Ten20(R) conductive paste or Elifex (R) just enough to fill the cup. Squeeze a bit of cream (EC2(R)genuine Grass electrode cream) on a piece of gauze to hold the electrode down for about 10 seconds, which dries up fast. This method does not actually “mix” conductors, since there is almost no contact between the two. One conductor is inside the electrode cup and the other one is on the outside and not serving any conducting function. The electrode impedance should be less than 5,000 Ohms and balanced. After the impedances are found to be satisfactory, apply a piece of 3M(TM) Micropore(TM) Microporous Hypo-Allergenic Surgical Tape over the electrodes on forehead and the temples, e.g., F7, Fp1, Fp2, F8, T1 and T2. Now you are ready to wrap the head. Two 4 inch self-adhering, conforming bandages are used. Tape the head wrap for security and then place a net over the head, which is very convenient, especially for children. Eight patients per week were monitored and evaluation for diagnosis of Epileptic seizures vs. non epileptic spells.

Results: This method is fast, easy and convenient with no Collodion odor, no skin breakdown and easy electrode removal with just water. The electrodes remain secured on patients with severe epileptic seizures and autistic children. Electrodes continue with low impedance and practically no repairs on patients monitored for 3 to 4 days. This procedure is also for patients who are allergic to Collodion.

Conclusion: Most of the recordings are of high quality and the Epileptologists are able to see the beginning, evolution and end of the seizure.

F46
Brain Seizing Heart Ceasing- Revisiting Changes In Seizure Semiology
Sally V. Mathias, MD; Samy Claude Elayé, MD; Isabel Derera, MD; Meriem Bensalem-Owen MD, FACS

Rationale: Seizure induced asystole is rare and could be responsible for increased risk of morbidity and mortality associated with epilepsy.

Methods: Case report of two patients with established epilepsy who developed de novo “drop attacks” characterized by brief loss of consciousness prompting the need for repeat video-EEG monitoring and concomitant cardiac monitoring.

Results: The first patient is a 60 year old woman with an established diagnosis of focal onset epilepsy, during video-EEG monitoring, had one stereotypical event that was consistent with a focal seizure of left temporal origin associated with 16 seconds asystole on the single lead ECG. The second patient is a 48 year old woman with history of well controlled seizures of right temporal origin. Repeat video-EEG monitoring captured a subclinical seizure originating from the right temporal region that was associated with significant bradycardia detected by cardiac telemetry. A Holter monitor completed a day before monitoring recorded 3 syncopal episodes with asystole up to 20 seconds. Both patients had a pacemaker inserted with resolution of arrhythmias.
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Conclusions: Repeating video-EEG monitoring while paying close attention to concomitant ECG is critical in the evaluation of epilepsy patients who develop a sudden change in seizure semiology particularly drop attacks.

F47 Advocating Cardiac Telemetry in EMU Based on Case Reports
Ardia Fu, MD; Laura L. Lehnhoff, MD

INTRODUCTION: Cardiac arrhythmia and asystole are well recognized phenomena in patients undergoing long-term video-electroencephalography. However, the current monitoring protocols used in many epilepsy monitoring units (EMUs) nationwide do not require continuous cardiac telemetry (CCT).

CASE DESCRIPTION:
Case 1: An 85 year-old male with no known cardiac arrhythmia presented to EMU for characterization of spells. We captured an event where the patient was unresponsive and shocked in all four extremities. During the event, the electroencephalography (EEG) showed diffuse attenuation, and the one-lead electrocardiogram (ECG) showed atrioventricular block leading to 40-second asystole. The patient underwent pacemaker placement the next day.

Case 2: A 57 year-old female with no known cardiac history presented to EMU for characterization of seizure like episodes. On day two of admission, she felt dizzy, became unresponsive, and had a brief full body myoclonic jerk. During the event, the background EEG activity was completely suppressed, and the one-lead ECG showed bradycardia evolving to 37-second asystole. The patient had pacemaker placed the following day.

DISCUSSION: We strongly advocate for CCT as part of a standardized protocol for patients undergoing EMU admissions to mitigate the potential mortality from cardiac events associated with seizures or from previously unrecognized cardiac conditions.

F48 Myoclonic Jerks and Generalized Tonic-Clonic Seizures — The Diagnostic Dilemma.
Inna Keselman, MD; Rafael J. Lopez-Baquer, MD; Yana Bukovskaya, MD; Joaquin Barredo, MD; Christina B. Baca, MD

Juvenile myoclonic epilepsy is a genetic generalized epilepsy syndrome characterized by myoclonic jerks and generalized tonic-clonic seizures (GTC) with electroencephalogram (EEG) showing irregular 3–6 Hz generalized spike/poly-spike and wave (SW/PSW) discharges. Focal semiological and interictal EEG findings have been recognized; focal semiology, however, typically correlates with generalized ictal EEG activity. We describe a 27-year-old man with a presumptive diagnosis of generalized epilepsy who was admitted to our epilepsy monitoring unit for differential diagnosis of GTCs unresponsive to anti-epileptic drugs (AEDs). His GTCs started at age 23 and were preceded by myoclonic jerks. His brother also had unclassified epilepsy since his early twenties. Brain magnetic resonance imaging was non-lesional and position emission tomography was normal. Interictal EEG showed bursts of either bisynchronous or left sided 3-4 Hz PSW complexes. The patient had two symmetric myoclonic jerks, one correlating with a large amplitude bifrontal SW complex, and the other with a large amplitude left hemisphere SW discharge. The patient had one generalized tonic-clonic seizure characterized by a generalized myoclonic jerk followed by confusion, head version to the right and “figure 4” sign, followed by generalized tonic-clonic movements. Ictal EEG revealed left-sided semi-rhythmic 4-5 Hz slow waves over Fp1, F7, F3 at onset. Behavior and ictal EEG suggest focal epilepsy with secondary bilateral EEG synchrony. The patient was discharged home on levetiracetam and lamotrigine and has not had a GTC in one year. Video-EEG monitoring is essential for characterization of ictal behavior and EEG in patients with presumed generalized epilepsies that are unresponsive to AEDs to assess for focal epilepsy with rapid bilateral EEG synchrony or co-existent focal epilepsy.

F49 Invasive Monitoring and Surgery for Isolated Epileptic Auras
Vinita J. Acharya, MD; Michael Sather, MD; Krishnamoorthy Thamburaj, MD; Jayant Acharya, MD

Background: Invasive monitoring and surgery are rarely performed in patients with isolated epileptic auras. We report a patient with disabling psychic auras, who underwent surgery after stereo-EEG.

Case Report: A 22 year old woman developed staring spells at age 6. She was placed on antiepileptic drugs (AEDs) after EEG showed right frontotemporal spikes, but was resistant to multiple AEDs. MRI showed a right mesial temporal gangioglioma, which was resected. Staring spells stopped, but the disease developed, disturbing sensations of déjà vu, anxiety and fear. On noninvasive video-EEG monitoring, she had interictal sharp waves in the right temporal region but no ictal EEG changes during multiple events. MRI showed residual tumor in the right uncus. Stereo-EEG was performed with 5 depth electrodes in the right temporal and orbitofrontal regions. She had six typical events with ictal EEG onset in the right amygdala and anterior hippocampal depth electrodes. There were no changes on scalp EEG. She underwent right amygdalo-hippocampectomy with removal of tumor. Postoperatively, she remains aura-free for more than 2 years, with significant improvement in quality of life.

Conclusions: Invasive monitoring and epilepsy surgery can be performed in patients with isolated epileptic auras if they are frequent and disabling, there are no scalp ictal EEG changes, and there is an MRI lesion. Invasive monitoring is useful to confirm the epileptic nature of isolated auras, study the relationship between the epileptic focus and MRI lesion, and to plan surgery.

F50 A Complex Epileptogenic Network Involving Periventricular Nodular Heterotopia
Jessica Templer, MD; Jay Ganvalo, MD; Elizabeth Gerard, MD; Micheal Macken, MD; Aditi Narechania, MD; Stephen A. Vanhaerents, MD; Stephen S. Schuele, MD, MPH, FACNS

Periventricular nodular heterotopia (PNH) is a neuronal migration disorder often associated with medically refractory epilepsy. Prior studies have shown that patients with PNH can have several possible epileptogenic foci: the periventricular nodule itself, cortex overlying the nodule, or distant structures. We present the case of a 23-year-old right-handed woman with a three year history of focal seizures and a single generalized convulsion. Clinical semiology consisted of déjà vu followed by nausea and occasional vomiting with subsequent loss of awareness associated with automatisms. Frequently, she would have a post-ictal headache. MRI demonstrated two discrete nodular foci of heterotopic gray matter along the lateral aspect of
the right temporal horn. Initially, it was thought that the ictal onset zone was one or both of the periventricular nodules. Stereo EEG revealed that the epileptogenic network included not only the anterior focus of gray matter heterotopia, but independently involved the mesial hippocampal and basal temporal structures. Stereo EEG with depth electrodes allowed for adequate identification of the complex epileptogenic network without the morbidity associated with subdural grid placement. Epileptologists should be vigilant in their pursuit of a true ictal onset zone despite the presence of a probable lesion.

F51
A New Waveform: The Texting Rhythm
Benedetto S. DiCiaccio, MD; William O. Tatum, DO, FACNS; Kirsten Yelvington, MD; Valerie Davis, MD; Shannon Anderson, MD

Objective: To describe a previously unidentified “texting rhythm” (TR).

Methods: 66 patients (51 F; mean age 34.74 years) with paroxysmal events (38 with epileptic (ES) and 23 with non-epileptic seizures (NES)) were admitted for video-EEG monitoring. The TR is a generalized burst of 5-6 Hz theta maximal in the fronto-central head regions during active texting. Other methods of activation included finger tapping/swiping, mathematical computation, scanning eye movements, speech/cognitive testing, and audio telephone conversations. The presence of a TR was compared between patients with ES and NES and between methods of activation.

Results: The TR was highly specific to texting without the appearance of a similar waveform during mental activation or audio cellphone use (p<0.0001). A TR was present in 44.0% of patients lasting from 2 seconds to continuous runs during the waveform during mental activation or audio cellphone use (p<0.0001). A TR was present in 57.9% of patients with ES but only 17.4% of patients with NES (p=0.0019). Additionally, the average age for those with a TR was 30.45 yrs while those without a TR was 38.11 yrs (p=0.0117).

Conclusions: The TR is a new and specific waveform linked to using new technology associated with text messaging. It likely reflects a visual-cognitive network activation unique to cellphone use.

Saturday, February 7, 2015

Display Time: 7:00AM – 2:00PM
Poster Tours: 1:00 – 2:00PM
Location: Liberty Hall, 1st floor
Categories: S1-S8 Critical Care Monitoring
S9 – S11 Digital/Quantitative EEG & Topography
S12 – S17 EEG
S18 – S27 Epilepsy: Clinical
S28 – S34 Evoked Potentials
S35 – S36 Functional Imaging
S37 – S51 Intraoperative Monitoring

S1
Unilateral Independent DiPLEDs and SPECT Imaging
Whitney Griffith, MD; Bruce Fisch, MD, FACS; Joanna Fair, MD

Periodic lateralized epileptiform discharges (PLEDs) are associated with hypermetabolism on PET imaging, and increased cerebral perfusion on SPECT imaging, although not invariably. Different PLED patterns vary in significance within the ictal-interictal continuum according to certain electrographic features (e.g., PLEDs Plus). We present acute SPECT and EEG findings in a unique case in which 2 independent periodic epileptiform patterns occurred within the same hemisphere.

A 57 year old man with a history of left sided craniotomy for subdural hematoma developed a subdural empyema and presented with convulsive status epilepticus. Convulsive status resolved following the administration of lorazepam, levetiracetam and phenytoin. The patient subsequently appeared to be alert but was unable to follow commands. Continuous video and EEG monitoring revealed left hemispheric PLEDs in 2 independent locations associated with 2 discrete areas of increased perfusion on SPECT imaging (illustrations to be presented). Our case demonstrates that in a setting of acute neurological impairment following convulsive status, multiple PLEDs can be associated with multiple independent areas of hyperperfusion. We believe additional studies using nuclear imaging are indicated to determine the pathophysiological correlates of nonconvulsive epileptiform patterns encountered in the ICU setting and their possible implications for treatment.

S2
EEG Characteristics in Therapeutic Hypothermia — Cooled and Warmed
Kyung-Wha Kim, MD; Elaya Robens, MD; Douglas Lubar, MD

Electroencephalogram (EEG) monitors brain activity continuously in therapeutic hypothermia (TH) patients where obtaining neurological exam and brain imaging are difficult. To better understand the effects of temperature on the brain, we retrospectively characterized EEG patterns during the cooled and subsequent warmed phases of 12 TH patients in a blinded fashion. For each patient, we analyzed six 30-minute segments (three cooled and three warmed) of EEG. We characterized and systematically classified EEG on the following criteria: continuity, background frequency, inter-ictal epileptiform discharges (IEDs), and discrete seizures.

All EEGs were abnormal, and they did not necessarily improve upon warming. Group improvement was seen on continuity and seizures: number of at least “nearly continuous” records improved from seen in 6/12 to 9/12 patients; seizures declined from seen in 2/12 to 0/12 (but warmed group was on antiepileptics). Group worsening was seen in presence of IEDs: these increased from seen in 2/12 to 6/12 patients. There were no deaths during EEG, but only three patients survived to hospital discharge. Upon warming, only 1/3 survivors developed new IEDs. However, 5/9 non-survivors developed new or more active IEDs.

EEG is a dynamic functional test, whose characterization during temperature changes may elucidate better understanding of brain activity.

S3
EEE Periodicity Checker
Fumisuke Matsuo, MD; Michael Ball, MD

Recent examinations of interictal epileptiform transients suggested that PGCO (polygraphic channel overlay) offered advantages for some applications (J Clin Neurophysiol 2014; 31: 289). When configured to cover prolonged time period in one display, PGCO can make realistic inspection of entire EEG of prolonged duration for periodic transients, simple or complex in waveform. Figure illustrates transition between periodic and electrographic seizure discharges in 500-s PGCO in serial bipolar derivations (A). Hemispheric asymmetry can only be appreciated in conventional display (B). Underlined 10-s segments in A and B correspond. Applications of PGCO to be presented from intensive care EEG monitoring have
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revealed wide variations of phase relationship among diffuse periodic discharges, including triphasic transients. Change in phase relationship infers change in EEG generator configuration. PGCO may contribute to case-controlled study of acute epileptiform encephalopathy.

S4
Incidence and Risk Factors for Skin Breakdown During Continuous EEG Monitoring
Christine Scott, MD; Lidia Moura, MD; Junior Valdery Moura, MD; M Brandon Westover, MD

Purpose: “Skin breakdown” during continuous EEG monitoring (cEEG) in acutely hospitalized patients has recently become a topic of national concern. We measured the incidence and possible factors responsible for cEEG-related skin breakdown.

Methods: We tracked skin breakdown among 1024 patients who underwent cEEG over 9 months. Variables analyzed included cEEG duration, patient age, within-hospital location, electrode type, adhesive type, and technologist who applied the electrodes. After univariate analysis, we included significant variables in a Cox regression model to estimate the time-dependent risk of skin breakdown.

Key Findings: cEEG-related skin breakdown occurs in <10% of patients overall. Predictors of skin breakdown in univariate analysis include age (most cases occurred in elderly patients); location (most cases occurred in ICUs); technologist (more experienced technologists had fewer cases), and electrode type (most cases involved MRI-compatible electrodes). Average overall risk was stable from month to month around 8%. One cEEG day conveys an 8% average risk. Patients undergoing prolonged monitoring experience a cumulative risk over 10 days of 30%.

Significance: Technologist experience/technique, electrode type and monitoring duration appear to be important modifiable risk factors for cEEG-related skin breakdown. Other potential factors that we are currently investigating include pressors, nutritional status, fever, and allergies.

S5
EEG Monitoring in Critically Ill Patients
Ammar Kheder, MD

Objective: The objective of this study was to estimate the burden of electrographic seizures in consecutive adults who underwent EEG monitoring and were admitted to non-Neurological ICUs.

Setting: Medical, surgical and cardiac intensive care units at Massachusetts General Hospital

Patients: 166 consecutive patients in non-neurointensive care without known acute neurologic injury between January and September 2013.

Main Results:

The Mean age was 60.39 years old. 56% were male and 44% were females. There was a total of 240 days of monitoring and the mean duration of monitoring was 1.44 days. The main indication for performing studies was altered mental status (144 patients 87%). The majority of patients had an underlying infection or sepsis (39% 23%). Other common diagnoses included: structural brain abnormalities or prior stroke diagnosed by imaging 21% (35), metabolic disturbances 9% (15), and substance abuse or overdose 7%. Fifteen patients had seizures (9%). Seizures were recorded from patient with sepsis 6, brain metastases 3, prior stroke 3, hypotension 1, known epilepsy 1, and unknown cause 1. Seven of those had electroclinical seizures and eight had electrographic seizures (48%). Time to detect the first seizure was less than 6 hours in 12 of 15 (80%). Of the remaining two seizures; one was captured within 24 hours and the other within 48 hours. Epileptiform discharges detected included sharp waves in 53 patients (32%), lateralized periodic discharges (LPDs) in 20 patients (12%), and generalized periodic discharges in 26 (16%).

Conclusion: In an unselected cohort of critically ill adults; seizures were detected in 9%. Purely electrographic seizures without apparent clinical accompaniment were present in 4.8%. Further larger prospective studies are required to evaluate the prevalence and the burden of electrographic seizures in critically ill patients without obvious acute neurological disorder.

S6
Cardiac Complications in Convulsive and Nonconvulsive Status Epilepticus
Yara Nazzal, MD; Kanika Arora, MD; Mohammad R Haider, MD; A K M Arifuzzaman, MD; S Pati, MD; M. Brandon Westover, MD

Objectives: Cardiac complications in status epilepticus (SE), especially in convulsive SE(CSE) are well known. Our goal was to assess the patterns of cardiac complications in non-convulsive SE(NCSE) and compare them with CSE. We hypothesized that cardiac complications in NCSE differ in type and frequency from CSE.

Study methods: A single centre, retrospective study involving 47 consecutive patients (adults and children) admitted in the neuroscience intensive care unit following diagnosis of CSE and NCSE. Cardiac complications were assessed by EKG, echocardiogram, and troponin values.

Results: 70% (N=33) had CSE and the remaining 30%(N=14)had NCSE. The most common etiology of SE was intracranial bleed (42%). Average length of stay in the ICU was 9.7 days. Cardiac complications were seen in 43% (N=6) of NCSE and 55% (N=18) of CSE patients. Cardiac complications included: cardiomyopathy or left ventricle hypokinesia, elevated troponins, and EKG abnormalities. Cardiomyopathy was predominantly seen in CSE (33%) VS 0% in NCSE while malignant arrhythmias were commonly seen in NCSE (28%) VS 0.09% in CSE .

Conclusion: The frequency of cardiac complications in NCSE was similar to CSE. However patterns of cardiac complications in NCSE were clearly different from CSE. These findings suggest that the underlying mechanisms and optimal methods of monitoring to detect and prevent adverse cardiac outcomes may be different for CSE and NCSE. These preliminary findings should be confirmed in a larger cohort.

S7
Clinical Performance of a Prospective Continuous Electroencephalography (cEEG) Ischemia Monitoring Service for Predicting Neurologic Decline after Aneurysmal Subarachnoid Hemorrhage (SAH)
Eric S. Rosenthal, MD; Kathryn L. O’Connor, MD; Sahar F. Zafar, MD; Siddharth Biswal, MD; M. Brandon Westover, MD

Introduction: Retrospective analysis has identified various cEEG features associated with delayed ischemic neurologic decline (DIND) or delayed cerebral infarction (DCI)
after SAH. We evaluated the performance of clinical neurophysiologists in clinical practice prospectively reporting cEEG for SAH ischemia monitoring.

Methods: Nontraumatic Hunt-Hess Grade 4-5 or Fisher Group 3 SAH patients met inclusion criteria for clinical cEEG monitoring. cEEG was scored in the clinical neurophysiology report for prospective determination of: percent alpha variability decline from baseline; new alpha-delta ratio decrement or asymmetry; new focal slowing; new epileptiform discharges; or new subjective impression of focal electrographic worsening. Maximal daily TCD peak systolic velocity (PSV) was recorded. DIND/DCI events occurring after the initial day of cEEG monitoring and before cEEG discontinuation were prospectively recorded by daily clinician interviews and multi-rater adjudication. Events were classified as Global Neurologic Decline (DIND-G), Focal Neurologic Decline (DIND-F), or Radiologic Delayed Cerebral Infarction (DCI).

Results: 34/71 (48%) patients undergoing cEEG monitoring over 2 years developed DIND/DCI, which was more common when preceded by cEEG deterioration vs. not (71.4% vs. 25.0%). cEEG had good sensitivity (Se 76%) and positive predictive value (PPV 69%) in clinical practice for detecting DIND/DCI, improving pre-test probability by >40%. TCD PSV>200 cm/sec provided only 48% Se and 38% PPV.

Conclusion: cEEG findings documented in clinical practice increase DIND/DCI prediction over the baseline prevalence. The biologic mechanism mediating these findings may include vasospasm-related ischemia, cortical spreading depolarizations, and events on the ictal-interictal continuum.

**S9**

QEEG in Psychopath Women
Ana A. Calzada Reyes, MD

Objective: The aim of the present study is to find out whether there are differences in quantitative EEG spectral parameters and intracranial distribution of QEEG activity during the rest condition, between psychopath and non-psychopath female inmates. Methods: The resting EEG activity and LORETA for the EEG spectral-slow bands were evaluated in 35 violent female offenders, 12 will and 23 without psychopathy according to the Hare Psychopathy Checklist-Revised. All subjects were assessed using the DSM IV-R criteria. The EEG visual inspection characteristics and the use of frequency domain quantitative analysis techniques (Narrow band spectral parameters) are described. Results: QEEG analysis showed a pattern of excess of theta activity on the the right frontonal region. LORETA revealed an increase of alpha activity (3.906 Hz) in psychopath group relative to non-psychopath female group within franto-temporal regions. Conclusions: These findings indicate that QEEG analysis and techniques of source localization may reveal differences in brain electrical activity among female offenders with psychopathy, which was not obvious to visual inspection. Taken together, these results suggest that abnormalities in a franto-temporal network playa relevant role in the neurobiological basis of psychopathy.

**S10**

Automatic Detection of Burst-Suppression Patterns in Scalp EEG
Franz Fürbass, MD; Manfred Hartmann, MD; Hannes Perko, MD; Johannes Koren, MD; Johannes Herta, MD; Andreas Gruber, MD; Christoph Baumgartner, MD; Tilmann Kluge, MD

The occurrence of burst-suppression patterns in the EEG of ICU patients is usually associated with poor outcome if induced by pathological conditions. To avoid exhaustive manual evaluation of the EEG, we developed an automatic and parameter-free method to detect burst-suppression patterns. To assess the detection performance of the method, continuous scalp EEG of 64 consecutive patients was recorded at two ICUs sites resulting in 3962 hours of EEG in total (min 6h, max 192h). A clinical neurophysiologist was asked to manually review the first minute of each recording hour in the EEG and to mark these segments as burst-suppression or normal EEG. The results of our automatic detection method were compared to the manual annotations to define detection performance by means of sensitivity and specificity. During manual review burst suppression patterns were found in 21 patients, segments without burst suppression were found in 63 patients. The average detection sensitivity was 82% (95% confidence interval from 70-90%). The average specificity was 80% (95% confidence interval from 73-85%). Our fully automatic detection method for burst-suppression patterns showed high sensitivity and specificity on unselected consecutive long term EEG recordings and will support automated EEG evaluation in the clinical setting.

**S11**

Passive Intracranial EEG Based Localization of the Central Sulcus during Sleep
Rafeed Alkawadi, MD; Hitten Z. Zaveri, MD; Jason L. Gerard, MD; Lawrence J. Hirsch, MD, FACNS; Dennis D. Spencer, MD

We report the results of a pilot study to test the performance of a new operator-independent method for passive identification of the central sulcus (CS). We studied...
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7 patients with intractable epilepsy undergoing intra-cranial EEG (iEEG) monitoring at Yale, in whom CS localization was obtained by standard methods. Our method takes advantage of inherent properties of the primary motor cortex (MC), which exhibits enhanced iEEG-high gamma power and coherence across the CS. For each contact x we calculated the z-score of a composite power and synchrony value log10(p_x) * cx, where px is sum of the root mean square of the iEEG in the high gamma band (80-115) Hz for contact x over the 6-10 mins of NREM sleep studied, and cx is the mean magnitude squared coherence in the same band using a 500-ms Hamming window between contact x and all other contacts. Z-score values lower than threshold (th) were set to 0. Finally, we calculated a metric m = z/d, where d is the mean Euclidian distance of each contact from contacts with z scores greater than 0. The last step was implemented to emphasize local network activity. The sensori-motor (SM) cortex exhibited higher EEG-gamma power compared to non-SM cortex (p < 0.0002). There was no significant difference between the motor/pre-motor and sensory cortex (p < 0.47). CS was successfully localized in all patients with thresholds between 0.4-0.6. In 2 patients, knowledge of anatomy was needed to distinguish the MC from adjacent epileptic foci. The primary hand and leg motor areas exhibited the highest metric values consistently followed by the tongue motor area. Higher threshold values were very specific (94%) for the anterior bank of the CS but not sensitive. Intermediate threshold values achieved a reasonable trade-off (0.4: 89% specific and 70% sensitive). This method can be used for passive identification of the CS, including possible use in the OR.

**S12 What's your EEG number? Resident Training & Competency**

Lynn Liu, MD; Adam Juersivich, MD; Thomas Wychowski, MD

**Objective:** Assess resident electroencephalogram (EEG) training experience and correlate experience to self-assessment of competency.

**Background:** Many procedural specialties have a specified number of procedures required to demonstrate competency. There are no data to generate such numbers for EEG interpretation. Exposure during Neurology residency can vary between 0-3 months. Quantity and diversity of cases can be a matter of chance. A neurology graduate should be competent in reading EEGs.

**Design/Methods:** Twelve University of Rochester neurology residents over 2 years completed their 4-10 weeks EEG rotation. Each investigator scored EEG reports by degree of abnormality for each subject’s experience then assigned them a rank based on their case-mix. Using a 4-point Likert scale each subject assessed their skills in interpreting reports and basic EEG milestones: normal EEG, common artifacts, normal variants, and common and uncommon abnormalities. To correlate the degree of complexity of the EEG experience and subject self-assessment, rank correlation was attempted using Spearman’s Rank-order Correlation.

**Results:** The total EEGs read range from 88-355 studies with abnormal studies representing 34-68%. Residents felt comfortable interpreting EEG reports and normal EEGs but were less comfortable with normal variants and uncommon abnormalities. Due to lack of degree of separation in self-assessments scores Spearman’s Rank-order Correlation could not be used.

**Conclusions:** Despite the lack of correlation between self-assessment score and EEG experience, this study raises important considerations to determine competency. Program directors sign off based on supervisor opinions potentially flawed by recall bias. Self-assessments may be limited by in insight. Current summative testing lacks specificity. Formative and summative assessment during the rotation should be validated and correlated with EEG experience.

**S13 Brief Potentially Ictal Rhythmic Discharges (BIRDs) in the Epilepsy Monitoring Unit**

Ji Yeoun Yoo, MD; Lara Marcuse, MD; Madeline Fields, MD

**Background:** Brief potentially ictal rhythmic discharges (BIRDs) and their association with seizures have been described in neonates and recently in critically ill patients. We aimed to identify BIRDs in non-critically ill patients and explore their association with seizures and other findings.

**Methods:** We prospectively identified BIRDs in patients who received long term video or ambulatory EEG monitoring from July 2013 to September 2014. Patients with status epilepticus or altered mental status were excluded. BIRDs were defined as rhythmic discharges of theta or higher frequency lasting less than 10 seconds.

**Results:** BIRDs were identified in 6 out of 620 patients (1%). Typical frequency and duration of BIRDs were theta/alpha frequency and 0.5-4.0 seconds. All patients had epilepsy and 4 of them were medically refractory; 3 of the 4 were non-lesional. In the 3 patients with lesions (2 benign tumor resection, 1 prenatal cerebral hemorrhage), BIRDs co-localized to them. BIRDs were extra-temporal (1 frontal, 4 centro-parietal) in all but one (frontotemporal) who had a tumor resection. Three had recorded seizures which co-localized to the BIRDs; in these patients, BIRDs preceded the seizure onset. All had co-localizing interictal epileptiform discharges, except in one who had independent sharp waves in the other hemisphere; in this patient, clinical seizures co-localized to the BIRDs.

**Conclusion:** This data demonstrates the presence of BIRDs in patients with epilepsy, more often associated with extra-temporal in origin and medically refractory in nature. Further study is needed to better understand their clinical and prognostic significance.

**S14 The Practicality and Diagnostic Yield of Ambulatory Electroencephalography (aEEG)**

Kader AbdeleRahman, MD; Madeleine M. Grigg-Damberger, MD, FACNS

**Background:** The University of New Mexico Hospital (UNMH) inpatient video EMU covers a wide under-served area. Current waiting time at the UNAH EMU is approximately 6 months. Outpatient ambulatory EEG may serve as a cost-effective and convenient alternative to inpatient video EEG monitoring.

**Objective:** To investigate our institution’s experience, utilization, and indications for outpatient aEEG in the diagnosis and management of epilepsy and to assess the diagnostic yield and limitations of aEEG.

**Methods:** Retrospective EMR and EEG database review of all patients undergoing aEEG at UNMH from 01/2008 to 04/2014. Variables analyzed included age, gender, presence of IEDs, habitual and/or non-habitual events, diagnostic yield, and limitations.

**Results:** 46 met inclusion criteria: 61% pediatric and 39% adults, 56% of the studies performed had a recording duration < 48 hours, remainder >48 hours. 39 studies were performed to characterize spells; habitual event(s) were captured in 51%, but
only 15% of those were associated with ictal EEG correlates. 6 aEEG studies were performed for quantification of seizures and IEDs for management purposes. One aEEG study was performed in a patient undergoing epilepsy surgery evaluation in whom no seizures occurred during one week of inpatient video monitoring. Focal seizures with ictal correlates were captured by the aEEG.

Conclusion: Outpatient aEEG is under-utilized at our institution. Physicians are more likely to refer those with nonepileptic spells to aEEG than to the EMU. However, in the absence of video or a good clinical description, events without EEG correlation can only offer a presumptive diagnosis of nonepileptic spells. Outpatient aEEG can provide useful clinical information for the management and localization of patients already diagnosed with epilepsy. The addition of video to the outpatient setting will surely add to the diagnostic yield.

S15
Intracranial Recording of “14Hz positive spikes” During Stereoelectroencephalogram Evaluation- A Case Study
Lazarus Mayoglu, MD; Juan Bulacio, MD; Jorge Gonzalez-Martinez, MD; Ahsan Moosa, MD

Recognition of normal variants on EEG is critical to avoid misdiagnosis. 14Hz positive spikes are frequent in older children and are readily recognized by most electroencephalographers on scalp EEG. Literature on this variant with intracranial EEG recording is limited. We report a 7yr old child with non-lesional focal epilepsy who had an intracranial evaluation using bilateral stereo EEG implantation for EEG recording is limited. We report a 7yr old child with non-lesional focal epilepsy who had an intracranial evaluation using bilateral stereo EEG implantation for epilepsy surgery. Stereotyped 0.5 - 1s bursts of sharp spindle like discharges were recorded from body and tail of the right hippocampus. They occurred in a monomorphic fashion of around 14Hz and were noted only in certain stages of sleep. They were high amplitude, up to 4-5mV. No independent sharp waves were noted in the right hippocampus. Concomitant scalp EEG showed typical patterns of 14Hz positive spikes in the right tempo-parietal region. Invasive evaluation suggested seizure onset from the left frontal lobe that lead to a focal resection. Patient remains seizure free 4 years after surgery. Recognition of such benign patterns may be important to avoid potential misdiagnosis of the epileptogenic zone.

S16
New Quantification Methods of EEG Spikes in Patients with ESES
Ahmet Tanritanir, MD; Michele Jackson, MD; Lindsay St. Louis, MD; Jacquelyn Klehm, MD; Tobias Loddenkemper, MD, FACNS

Rationale: This study aims to describe the correlation of 3 EEG features in ESES, spike-wave index (SWI), spike frequency (SF) and one hour spike frequency (OHSF) in sleep and wakefulness periods and to present a novel quantification tool, the sleep to wakefulness ratio (SWR) for clinical use in evaluating the treatment of ESES.

Methods: We retrospectively evaluated SWI, SF and OHSF in 15 patients diagnosed with ESES who had overnight video-EEG monitoring at a tertiary center from 2012 to 2014. We included patients with at least 50% spike percentage in slow wave sleep. We determined the correlation between SWI and SF for 1st 5 min of sleep and a 5 min period of wakefulness and the correlation with OHSF for longer period of 1 hr in sleep and wakefulness. The SWR for SWI, SF and OHSF was evaluated. SWI was defined as percentage of 1-sec bins with at least 1 spike-wave complex for a 5 min period. SF was defined as spike count in same 5 min period and OHSF was defined as spike count in a 1 hr period.

Results: Median age was 7.73 (Range: 3-11, SD: 2.7) yrs and 66.7% were males. Median SWI was 70.7 (IQR: 52-83, SD: 15.4) in sleep and 24 (IQR: 9.7-43, SD: 16.8) in wakefulness. Median SF was 265.5 (IQR: 235-335, SD: 102.9) in sleep and 100 (IQR: 30-200, SD: 83.1) in wakefulness. Median OHSF was 2650 (IQR: 1976-4322, SD: 1651.4) in sleep and 879 (IQR: 326-2001, SD: 1172.6) in wakefulness. SWI and SF in sleep (Spearman correlation coefficient, R = 0.921; p=0.0001) and wakefulness (R = 0.971; p=0.0001) correlated well. OHSF and SWI correlated in sleep (R= 0.771; p=0.001) and wakefulness (R=0.874; p=0.0001). OHSF and SF correlated in sleep (R=0.711, p=0.003) and wakefulness (R=0.886, p=0.0001). The SWR for SWI, SF and OHSF was 3.83, 4.45 and 5.98 respectively.

Conclusions: This biomarker, sleep to wakefulness ratio may provide an additional method for EEG quantification that can be used clinically in evaluating ESES.

S17
Ictal Appearing Discharges Terminated with Sensory Stimulation
Mark Callow, MD; Jane Mitchell, MD; Dr Abdullah Al Sawaf, MD; Dr Dominic Fee, MD; Meriem Bensalem-Owen, MD, FACNS

Background: The reverse phenomenon of stimulus induced rhythmic or periodic ictal discharges (SIRPDs) where ictal appearing discharges are terminated by sensory stimulations has been rarely described. In a series of 33 patients with SIRPDs, one individual whose epileptogenic activity was aborted with stimulation was reported. Four patients with this unusual electrographic pattern were identified at the University of Kentucky.

Methods: Patients with rhythmic or periodic lateralized epileptiform discharges aborted with various sensory stimulations were identified upon review of either continuous video-EEG monitoring or standard EEG.

Results: Four patients were identified over a period of 8 years. This short series included three males and one female with a mean age of 55.5 years. All patients had various cerebral insults including stroke, traumatic brain injury, dementia and metastasis to the calvarium. Two of these patients had prior SIRPDs. Demographics, history, and EEG findings were reviewed.

Conclusions: The termination of ictal appearing discharges with sensory stimulations is an interesting and rare phenomenon. The clinical significance and pathophysiology of this electrographic pattern remains however unclear. Future studies directed at determining whether sensory feedback mechanisms are involved in the termination of an ongoing seizure would be helpful and could contribute to improved patient care.

S18
Symptomatic Hepatomegaly as a Complication of Prolonged Treatment for Refractory Status Epilepticus
Derek Debricki, MD; Teneille Gafton, MD

Prolonged treatment of refractory status epilepticus (RSE) can result in significant systemic complications. The current abstract reports two patients with new onset refractory status epilepticus (NORSE) syndrome who developed symptomatic hepatomegaly during treatment for prolonged refractory seizures in the intensive care unit (ICU). In both, hepatomegaly contributed to abdominal compartment
syndrome; surgical intervention was required for one. Comparison of abdominal imaging at the time of seizure presentation and at the development of abdominal symptoms suggests that the observed hepatomegaly developed during the course of treatment. Both patients required multiple intermittent and continuous anti-epileptic drugs (AEDs) and prolonged use of anesthetic agents to maintain a burst-suppression pattern (>60 days) on continuous electroencephalography (cEEG) for seizure control. In the first case, surgical pathology demonstrated a pattern of cellular proliferation (consent for liver biopsy was declined in the second). Indeed, enzyme-inducing AEDs have been associated with hepatic cellular proliferation in animal models. Although no direct causal relationship can be determined, it is suspected that the prolonged use of AEDs (including enzyme-inducing agents) and/or anesthetic agents resulted in the iatrogenic development of symptomatic hepatomegaly. This is a significant complication that should be considered in the protracted management of RSE in the ICU.

S19
Stereo-EEG and Surgery in Bilateral Perisylvian Polymicrogyria
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Introduction: Epilepsy surgery is rarely performed in patients with bilateral perisylvian polymicrogyria, and becomes more challenging when there are additional epileptogenic lesions. We report a patient with bilateral asymmetric perisylvian polymicrogyria (BAPSPMG), septo-optic dysplasia and unilateral temporal hypoplasia, who underwent surgery after stereo-EEG (SEEG).

Material and methods: Case report

Results: A 44 year old man presented with pharmacoresistant focal dysexecutive seizures since age 23. Brain MRI showed BAPSPMG (left>right), absent septum pellucidum, hypoplastic optic nerves and chiasm, and left temporal hypoplasia. On noninvasive video-EEG monitoring, two left hemispheric onset seizures were recorded. FDG-PET showed left temporal hypometabolism. Wada test and functional MRI revealed bilateral language representation, with severely impaired left and normal right memory. SEEG was performed with 7 depth electrodes in the left temporal and perisylvian regions. Five seizures were recorded with ictal EEG onset in the left neocortical temporal and perisylvian temporal regions. An extensive left temporal lobectomy with removal of opercular tissue was performed. Four months after surgery, he remains seizure-free.

Conclusions: Epilepsy surgery can be performed in BAPSPMG on the side with more prominent MRI findings, if there are concordant, unilateral EEG and PET abnormalities. SEEG is useful to study the relationship between the epileptic focus and the lesions, and to plan the extent of resection.

S20
Lacosamide Associated Sinus Pauses Without Significant PR Change
Fawad A. Khan, MD; Michael Bernard, MD; R. Eugene Ramsay, MD; Hina Dave, MD

Introduction: Lacosamide (LCM), a newer antiepileptic drug for the adjuvant management of partial onset seizures, was recently approved for monotherapy. Cardiac conduction disturbances are a known side effect of LCM. We report a case of repeated sinus pauses with LCM.

Case Report: A 67 year old male with history of hepatocellular carcinoma and hepatitis C cirrhosis presented with acute alteration of mental status and myoclonus. Based on clinical suspicion for status epilepticus, Levetiracetam (LEV) and LCM were initiated. The EEG failed to show evidence of seizures and LEV was discontinued. Cardiac telemetry recording did not show any cardiac arrhythmias and electrocardiogram showed normal sinus rhythm and right bundle branch block. A mild increase in PR interval was noted following LCM. 5 days later multiple sinus pauses were noted on the telemetry recording requiring transcutaneous cardiac pacing. Within 48 hours of discontinuation of LCM the sinus pauses resolved.

Discussion: We observed sinus dysfunction without significant increase in PR interval likely secondary to accumulated toxicity of LCM (dose of 400 mg/day) as a consequence of hepatic dysfunction. Asystole and severe conduction blocks with intoxication of LCM have been previously reported. This case underscores vigilance in cardiac monitoring and appropriate dosing of LCM in patients with hepatic and renal dysfunction to avoid cardiac toxicity.

S21
Pyridoxine Deficiency in Adult Status Epilepticus Patients
Hina Dave, MD; Fawad A. Khan, MD; Vivek Sabharwal, MD; R. Eugene Ramsay, MD

Background: We wanted to evaluate pyridoxine levels in adult patients admitted with status epilepticus. Methods: With IRB approval, we reviewed the records on patients admitted to the neurological ICU for status epilepticus from January to November 2014 as well as clinic patients from the last three years. Reported normal pyridoxine range is 5 to 50 ug/L. Results: In the status population (48 patients), all but four patients had low normal or undetectable pyridoxine levels. 132 adult outpatients were identified. 39% had a low normal pyridoxine level. The mean pyridoxine was 5.5 ug/L in the status group and 25.2 ug/L in the outpatient group. See Figure 1. Conclusion: Pyridoxine is a water-soluble vitamin that is naturally present in many foods. The active component, pyridoxal 5’ phosphate, binds to intracerebral glutamic acid decarboxylase which is the enzyme responsible for the conversion of glutamate to GABA. See Figure 2. A pyridoxine deficiency was seen in 65.6% of status patients versus 10.6% in the outpatients, a statistical difference to the comparison group. Further studies on the effect of pyridoxine on status control are needed.

S22
Number of Allergies in Epileptic VS Non-epileptic Seizures
Aradia Fu, MD; David Denny, MD; Samara Cerven, MD; Naudia Moorley, MD; Steve Chung, MD

Objective: The primary goal was to compare the number of patient-reported allergies within adults with epileptic seizure (ES) and non-epileptic seizures (NES). The secondary goal was subgroup analysis of gender difference. Background: The cost of direct and indirect care for patients with NES are comparable to that of care for patients with intractable epilepsy. Therefore, it is critical that we have reliable indicators for NES in order to establish accurate diagnosis early.

Methods: This cohort study collected data from 1,635 epileptic monitoring unit reports of adult patients. We reviewed the number of allergies reported by each patient and the final diagnosis. Patients with ambiguous events, no captured events,
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and those with both ES and NES were excluded from this study. Patients were divided into four groups: female ES (f-ES), male ES (m-ES), female NES (f-NES), and male NES (m-NES). We used one-tailed T-test when comparing the number of allergies between NES and ES and two-tailed T-test when comparing the gender difference within each ES and NES groups.

Results: There was a total of 630 f-NES and 252 m-NES patients within NES group (n=882), and 443 f-ES and 310 m-ES patients within ES group (n=753). The mean reported number of allergies was 1.91 for f-NES (SD = 2.71), 1.14 for m-NES (SD = 1.56), 0.87 for f-ES (SD = 1.73), and 0.49 for m-ES (SD = 0.99).

Number of allergies reported by NES patients was significantly greater than that reported by the ES patients (p < 0.001), regardless of gender. However, clear gender difference was noted within the two diagnosis groups, with females reporting greater number of allergies in NES (p < 0.001) and ES (p < 0.001).

Conclusions: Patients with NES self-report greater number of allergies than patients with ES. Furthermore, significantly greater number of allergies was reported by the female than male, regardless of the diagnosis of NES or ES.

S23
Intraoperative-ECoG during MRI-guided Stereotactic Laser Thermal Ablation of Mesial Temporal Structures for Intractable Epilepsy
Michael Pietak, MD; Matthew W. Luedke, MD; Shervin Rahimpour, MD; Sandra Serahin, MD; Michael Haglund, MD; Saurabh R. Sinha, MD, PhD

MRI-guided stereotactic laser thermal ablation (Visualseal procedure) is a new modality for epilepsy surgery. In trials on patients with intractable localized resection seizures, it has been shown to successfully destroy epileptogenic lesions with less morbidity than conventional craniotomies, and potentially similar rates of seizure freedom. It is gaining favor in the treatment of mesial temporal sclerosis, where the circumscribed epileptic focus is amenable to a stereotactic approach. In some centers, including ours, intraoperative electrocorticography (ECoG) is a standard procedure during amygudolihippocampactomies. Results of intraoperative ECoG have been used to guide the extent of resection of mesial temporal structures as well as prognostication. Given the limitations of burr-hole access during Visualseal procedures, ECoG has not previously been applied. Here we present two case reports involving intraoperative ECoG monitoring with a depth electrode placed into the parahippocampal region, recording before and immediately after thermal ablation of mesial temporal seizure foci. In each case, there were changes in mesial temporal spike and burst activity after laser ablation of the hippocampus. This is the first demonstration of intraoperative neurophysiologic monitoring during stereotactic laser thermal ablation. The role of intraoperative ECoG for guiding the extent of ablation and determining prognosis during Visualseal procedures remains to be determined.

S24
Safety and Pharmacokinetics of IV Loading Dose of Lacosamide in the ICU
R E. Ramsay, MD; Vivek Sabharwal, MD; Fawad Khan, MD; Megan Irland, MD; Misty Jenkins, MD; Varsha Gusman, MD; Hina Dave, MD;

Rationale: Lacosamide (LCS) is a relatively new AED available for IV administration reported last year to be a fast, effective and safe alternative in emergency situations. This warrants further investigation to better understand the safety and doses which can be used in this situation.

Methods: With IRB approval, patients were identified that received IV LCS in the ICU for acute treatment of seizures in the past 18 months. Selected were those who were given an initial infusion of 400 mg or more. Data collected were age, gender, weight, duration of infusion, change or termination of infusion for side effects (primarily drop in blood pressure), initiation of pressor agents during or up to 2 hrs after infusion completed. On a subset of 41 patients, LCS level had been obtained about 10 minutes after completion of infusion.

Results: 94 patients were identified. Demographics were male/female 48/46, average weight 82.0 kg (range 43.7 - 182.3), and average age of 56.1 yrs (24 - 83). Doses were 400 mg (24 pts), 500 (4 pts), 600 mg (31 pts) and 800 mg (4 pts). Weight base dosing ranged was 2.68 to 13.60 mg/kg (ave 6.9). No patient had a change in 1) BP resulting in reduction in or stopping the infusion or 2) starting pressors. LCS levels were obtained in 41 patients post infusion. LCS level correlated well with weight based dosing. Doses above 7 mg/kg produced levels of 10 ug/ml. Average volume of distribution was 0.57 L/Kg.

Conclusions: IV LCS can be safely given up to 1100 mg or 13 mg/kg over 30 min. Vd in ICU patients (0.57) is similar to reported value of 0.6 L/K in healthy volunteers. Weight based dosing should be used to achieve a target plasma level. Steady state LCS levels reported in clinical trials with 200, 400 and 600 mg per day are 4.99, 9.35 and 12.46 u/ml. To achieve high “therapeutic” level post IV load, doses of 8-10 mg/kg should be used which we found to be safe to use. .

S25
Hand Signs in Primary and Secondarily Generalized Motor Seizures
Jason Siegel, MD; William O. Tatum, DO, FACNS

Introduction: Localization-related epilepsy (LRE) is diagnosed by combining electroencephalography (EEG) and clinical semiology. However EEG may be non-localizing and non-lateralizing in both seizures of focal and generalized origin. We aimed to assess hand postures to identify differences between primary and secondarily generalized motor seizures.

Methods: We retrospectively identified generalized motor seizures in 16 patients admitted to our Epilepsy Monitoring Unit (EMU) in 2014. Based on video EEG, 6 had a final diagnosis of LRE, 5 had GGE, and 5 had Psychogenic Non-epileptic Events (PNEA). Video analysis examined the angle of the first metacarpophalangeal joint and its relation to the middle finger. Other clinical seizure characteristics and EEG patterns were recorded.

Results: Twenty-three convulsions were reviewed (9 LRE, 5 GGE, 9 PNEA). Unilateral or bilateral finger pointing during any phase of the convulsion was 100% sensitive and 85.7% specific for LRE. In GGE the most common hand position was bilateral fist formation. PNEA patients had a variety of atypical and fist positions. On EEG, 4 LGE seizures had focal onset, though the other 5 had asymmetric or focal interictal activity or suspicious neuroimaging. All GGE patients had normal backgrounds with generalized interictal epileptic activity and seizure onset. All PNEA patients had normal backgrounds with no change on ictal EEG.

Discussion: Based on these data, patients with extended first metacarpophalangeal digits are likely to have a focal basis for their generalized seizures. These results could implicate a change in treatment when finger pointing >25 degrees is identified.
S26
Novel Ambulatory EMG-based GTC Seizure Detection Device for Home & Hospital Use
Jose E. Cavazos, MD; Michael Giraud, MD; Luke Whitmire, MD

Rationale: There are no FDA-cleared devices outside of an epilepsy monitoring unit (EMU) that can reliably alert for generalized tonic-clonic seizures (GTCS) and provide accurate semiological details to physicians. This study was designed to validate the effectiveness of a novel EMG-based, real-time, GTCS detection system that can be discretely worn without interfering with activities of daily living.

Methods: In this phase III double-blind controlled trial, we tested the sensitivity and specificity of the Brain Sentinel GTCS detection system as compared to vEEG detection in EMUs at 11 NAEC level IV Epilepsy Centers in the U.S.A. Independent ABPN Epilepsy certified neurologists reviewed vEEG records to determine Classic GTCS semiology and timelines.

Results: More than 6000 hours of EMG and vEEG data have been reviewed from 118 subjects. The sensitivity of the GTCS detection system is comparable to FDA-cleared automated EEG seizure detection algorithms while maintaining a high level of specificity. Recorded EMG data preserves clinically relevant semiology of events such as temporal components of motor recruitment during a GTCS and activity following events.

Conclusions: Brain Sentinel’s system promises to provide people with epilepsy greater independence and peace of mind in a home or clinical setting while providing physicians with useful data for seizure management.

S27
A Retrospective Study of Continuous EEG Monitoring in a Single Tertiary Care Pediatric Center
Arnold Sansevere, MD; Rejean Guerriero, MD; Ivan Sanchez Fernandez, MD; Lindsay St. Louis, MD; Jacquelyn Klehm, MD; Tobias Loddenkemper, MD, FACNS

Rationale: The main goal of this study is to describe the clinical and electroencephalographic (EEG) characteristics of children who underwent continuous EEG (cEEG) monitoring in the intensive care unit (ICU).

Methods: This is a retrospective descriptive study of patients aged 1 month to 21 years who underwent cEEG (>3 hours) in the ICUs at Boston Children’s Hospital in the period of 2011-2013. Patients were excluded if the cEEG was performed as part of an elective admission or if monitoring occurred in the setting of epilepsy surgery evaluations. In patients with multiple episodes of cEEG, only the first trial was considered.

Results: 414 patients with a median age of 4.2 years were included. Thirty-five percent of patients had epilepsy, while 45% of patients had another neurologic disorder. Neuro-imaging was performed in 95% of patients (Table 1). Clinical suspicion of convulsive seizures/characterization of events was the main indication for cEEG (70.5%), while encephalopathy/concern for nonconvulsive seizures was the second most common indication (Table 2). The etiology of the episode leading to cEEG monitoring was structural in 213 (51%) patients. A burst suppression pattern was seen in 4.4% of patients while 14.3% of patients had a normal awake and asleep background. Overall, 98 (24%) patients had EEG seizures, of which 22 (22.4%) were solely electrographic. 12 out of 96 (11.5%) of patients met criteria for status epilepticus, defined as a continuous seizure lasting >30 minutes in 6 patients or recurrent seizures totaling >50% of a one hour epoch in the remainder. Mortality was 15% (59 patients).

Conclusions: We describe a large series of pediatric patients in the ICU who underwent cEEG monitoring. Twenty-three percent of patients had recorded seizures. Solely electrographic seizures are very common, affecting one-fifth of children with recorded seizures.

S28
Electrodiagnostic Before and After Spinal Surgery
Fernando Rivera-Martinez, MD

Introduction. Although neuroimaging offers extremely precise anatomic evaluation, EDX testing is still useful. Specifically, SEP can complement and therefore improve the evaluation of patients with lumbosacral pathologies. Objective. Describe EDX results, pre and post surgical in lumbosacral pathologies, with emphasis on findings of tibial SSEP and dermatomal L5/S1. Methods. EMG, NCS, F-Wave/H-Reflex and SEP to 32 adults whose disease had surgical judgment. Results. All patients showed alterations in the EMG neurogenic-pattern characterized by MUAPs markedly polyphase, high amplitude and signs of denervation at rest. NCS with prolongation of latencies and slowed conduction velocities in both motor and sensory nerves. In general, EMG-NCS and late-responses showed improvement at follow-up 6 months after surgery. Cortical responses SEP with marked alterations in the pre surgical evaluation showed more replicable and given lowest latency after spinal surgery answers changes. Conclusions. Clinical and images were accompanied by alterations in the EDX findings, being possible through the latter demonstrate the improvement in response to surgical treatment. Specifically, SEP changes suggest potential markers of functional changes that occur in the posterior cord pathway and that it is only possible to detect a longitudinal study, although it is required to replicate these findings by studying a larger sample.

S29
Neurophysiological Profile in Mexican Children with Congenital Hypothyroidism
Gabriela Romero, MD; Carmen Sanchez, MD

Congenital hypothyroidism (CH) is a systemic disease caused by absent or decreased thyroid hormones during pre and postnatal development; is associated with neuronal migration, morphology and connectivity axonal and dendritic growth and myelination, its absence may explain the presence of sequelae such as mental retardation. Objective: to find functional profiles through EEG, BAEP and VEP in children with treated CH.

Materials and methods: Included 33 children, 26 female and 7 male, 14 under one year, 11 between two and tree years and 8 four years; 18 athyreosis and 15 with sublingual nodule. EEG, BAEP and PEVC were performed. The condition at birth, hypothyroidism type, treatment start and hormone levels were obtained.

Results: EEG. Delta activity >70%, amplitude of 51.06 µV, lower voltage in the left hemisphere.

VEP. Binocular visual cortical responses were statistically different associated with hypothyroidism type: athyreosis vs. sublingual nodule (p<0.05). The athyreosis children had the longest visual responses.
ABEP: The latency of waves and interwave intervals were abnormal delayed in children with athyreosis (p <0.05).

Conclusions: There influence of the type of HC on the functional expression of children. The neurophysiological profiles show functional processes altered, indicators of the commitment of SNC.

S30
Continuous Theta burst stimulation (cTBS) applied to the dorsolateral prefrontal cortex modulates late somatosensory evoked potentials (SEP) in normal subjects.
Robert J. Varipapa, MD

Background: Transcranial magnetic stimulation (TMS) is a well-studied and widely employed method for non-invasive stimulation and study of the human brain. The motor cortex is the most prominent of these areas likely due to the ability definitively measure cortical excitability with the motor evoked potential. Due to the lack of an overt physiologic measurement, TMS has been poorly studied in other brain regions. The TMS theta burst protocol developed by Huang et al. has been shown to induce transient physiologic changes in the motor cortex. Specifically, the continuous theta burst protocol was demonstrated to diminish MEPs when applied to the motor cortex. Previous research has demonstrated that individuals with a lesion to the DLPFC present with altered SEPs.

Objective: Here we apply the TMS protocol of cTBS to the dorsolateral prefrontal cortex of normal subjects. We expect that the application of cTBS to the DLPFC will produce transient SEP changes consistent with persons with a lesion in the same area.

Methods: Normal subjects aged 18 to 65 were recruited from the Roanoke Valley. Baseline SEPs were elicited via stimulation of the median nerve and recorded via a 64 electrode EEG. cTBS was then be applied to the subjects contralateral DLPFC. SEPs were then measured at 7 and 14 minutes post stimulation.

Results: Group and individual subject data was analyzed. ANOVA failed to show any significant change between pre cTBS and post cTBS SEPs consistent with a real brain lesion in the DLPFC.

Conclusion: The application of cTBS to the DLPFC failed to elicit a significant change in SEPs consistent with a lesion in this area. This result suggests that cTBS does not sufficiently generate an area of suppression in the DLPFC capable of altering SEPs as observed in persons with a real lesion in this brain area.

S31
Evaluation of Idiopathic Sudden Sensorineural Hearing Loss with Brain Stem Auditory EP and ECOG
Syed S. Habib, MD

Objective: Idiopathic sudden sensorineural hearing loss (ISSNHL) is a frightening symptom that often prompts an urgent visit to physicians. The present project aimed to study brain stem auditory evoked potential (BSAEP) and electrocochleographic (ECOG) findings in patients presenting with Idiopathic sudden sensorineural hearing loss.

Methods: This Observational Study was conducted in the Department of Clinical Physiology at KAUH, King Saud University Riyadh, KSA. We studied 23 cases with idiopathic sudden sensorineural hearing loss (ISSNHL). The diagnosis of ISSNHL was made on the basis of sudden nonfluctuating hearing loss, etiology that remained unknown after clinical, laboratory and imaging studies, severity of the hearing loss averaging at least 30 dB HL for three subsequent one octave steps in frequency and blank otological history in an otherwise healthy individual. We performed BSAEP & ECOG in these cases according to standard protocols.

Results: Left Ear was affected in 9 (39.1%) patients, Right Ear in 13 (56.5%) and was bilateral in 1 (4.3%). Both Wave I and Wave V were significantly prolonged in affected ear compared to unaffected ears (p=0.0031), while in interpeak latency I-V latency was significantly higher in affected ears versus unaffected ears (p=0.0544). Six patients (26.1 %) had type II diabetes mellitus, five cases (21.7%) had hypertension and 5 cases (21.7%) had dyslipidemia. ECOG revealed absence of Summation Potential (SP) and Action Potential (AP) response even at 95 dB in 17 out of 23 cases (73.9%). In 31.8 % cases, unaffected ear had mild while 31.8% had high hearing threshold in the unaffected ear.

Conclusion: BSAEP and ECOG provide useful diagnostic information in patients with ISSNHL in addition to radiological and other investigations. It could also provide a guide for future cochlear implants in patients especially when it is bilateral.

S32
Lateral Femoral Cutaneous Nerve Somatosensory Evoked Potentials in Meralgia Paresthetica in Sri Lanka
Vajira S. Weerasinghe, MD; Nimal Senanayake, MD

Lateral femoral cutaneous nerve (LFCN) entrapment at the inguinal canal is referred to as meralgia paresthetica (MP). Previous studies have reported recording of nerve conduction along this nerve which is practically a difficult procedure to perform. Somatosensory evoked potentials (SSEP) have been reported. But the results of those studies have not been consistent. In a group of patients with numbness of lateral aspect of the thigh, somatosensory evoked potentials were recorded from the lateral femoral cutaneous nerve to assess whether there is any evidence of compression of the nerve at the inguinal canal.

Materials and Methods: This was an observational descriptive study conducted at the Teaching Hospital, Peradeniya, Sri Lanka. Cutaneous site on the upper lateral thigh region innervated by the lateral femoral cutaneous nerve was stimulated using standard stimulating electrodes and the SSEP were recorded at Cz-Fz electrode positions using a Natus electromyographic machine.

Results: There were 68 patients with an age range of 19-81 yrs. Right side was affected in 51%, P38 wave was absent in 11 pts while it was delayed in 31 patients (4.5 to 20.1 ms delay). A significant amplitude difference was found in 11 patients. There was no latency or amplitude difference in 15 patients. According to these results in 53 patients (78%), SEP was able to detect a compression while in 15 patients (22%) a compression could not be detected.

Conclusions: This study shows that in clinically suspected meralgia paresthetica, somatosensory evoked potential recording is a relatively useful technique to detect a compression of the lateral femoral cutaneous nerve at the inguinal canal.
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S33
Cross-Modal-Plasticity: study with Evoked Potential
Lidia Charroo Ruiz, MD

This research focuses on Cross-Modal-Plasticity in deaf and deaf-blind children candidates for Cochlear Implants (CI) through the evaluation of the maps topography of the Visual-Flash (VEP) and Somatosensory-of median (SSEP-N20) and tibial (SSEP-P40) nerves Evoked Potentials. In deaf children (n=14) and deaf-blind children (n=12) topographic maps SSEP-N20 showed expansion of activation. Cortical areas: temporal in deaf and temporal-occipital in deafblind children were activated. These regions under normal physiological conditions processed information auditory and visual but no somatosensory. SSEP-P40 no showed changed with localized in the central-parietal mid-line (area of somesthetetic representation of foot). Maps topographic of the VEP no showed changes. The expansion of the cortical response of the SSEP-N20 to the left temporal region in deaf and deafblind children was interpreted as evidence of Cross-Modal-Plasticity, although in deaf children the over-representation of SSEP-N20 was less extensive that in deafblind children. These results are pointing neuroplastic changes that occur as result of the loss input of the sensory information during the critical period of neurodevelopment, as well as, the effect that may have the use of the hands for communication in these children, with consequent implications of the Cross-Modal-Plasticity to the optimal use of the CI after implantation.

S34
Synergistic Effect of Theanine and Caffeine on Visual Reaction Time, Evoked Potentials and Cognitive Event Related Potentials
Vajira S. Weerasinghe, MD; Chanaka Kahathuduwa, MD; Tissa Amarakoon, MD; Tharaka Dassanayake, MD

Theanine and caffeine have shown improvements in behavioral cognitive studies1-3. We examined the neurophysiologic effects of theanine and caffeine on perception, cognitive processing and expression. In a double-blind, five-way cross-over trial, we administered 200 mg theanine (T), 160 mg caffeine (C), a combination of T and C (TC), 10 g Ceylon black tea (Tea) and distilled water (placebo) prepared as 150 ml solutions on five separate days to 20 healthy males with an age range 18-28 years. We recorded recognition visual reaction time (RVRT) before and 55 minutes after each administration and visual evoked potentials (P100), motor evoked potentials4 (MEP) and auditory P300 event-related potentials4-5 (oddball paradigm) within 30-55 minutes after each administration. We calculated cognitive processing time4 (CPT) using RVRT, P100 and MEP. RVRT and CPT improved with T (22.4 ms), C (21.6 ms) and TC (38.4 ms) (p<0.05). A substance-time interaction was seen in CPT (p=0.048). T and C increased P300 amplitudes compared to placebo (p=0.001). TC further increased P300 amplitude to a level greater than both T and C (p<0.05). P100, MEP and P300 latency did not differ across substances. Thus, theanine and caffeine seem to improve speed and neural resource allocation in cognitive tasks synergistically.

S35
Pseudo-random Pairs in Functional Brain Networks Using fMRI-based Adjacency Matrices.
Ioannis P. Pappas, MD

Clustering of the brain network in a neurological context consists mainly of identify clusters of nodes that as totalities behave independently and exhibit a uniform behavior within their body. The subsystem segregation model considers the existence of dense, and highly functional subgraphs. In this paper we extend this framework by identifying areas of the brain that form pseudo-random pairs regardless of their topological position; these pairs are ε-close to being completely random in terms of their edge density. From a probabilistic point of view, a node in any of these clusters has a probability proportional to ε to be connected with an arbitrary node of any other cluster. A bit of information can travel between these with a strictly defined probability and, potentially, following a specific pattern. The theoretical framework behind these pseudo-random measures can establish facts about the existence of patterns in the brain network (such as triangles) that can populate, at least “primafacie”, non-segregated areas. Eventually, quantifying pseudo-random behavior can reveal functional deviations in patients with abnormal connectivity such as Alzheimer patients where it has been showed that there is a transition from the normal small-world networks to highly random networks. Quantifying such deviations can be replaced by comparing the pseudo-random interpolations between the clusters of the two graphs and arguing probabilistically about the information exchanged between different parts of the brain. Our method consists of two parts: Firstly, we construct the adjacency matrix of the fMRI data based on the linear correlation of the time series of each voxel. Secondly, we apply a modified version of Szemerédi’s algorithm that produces ε-regular partitions of the aforementioned graph.

S36
Fully Automatic Head Modelling for EEG Source Imaging
Gerhard Gitsch, MD; Franz Fürbass, MD; Manfried Hartmann, MD; Hannes Perko, MD; Tilman Kluge, MD

EEG source imaging can provide clinical relevant localization results especially if individual head models are used. We developed and evaluated a fully automatic procedure for head model extraction. A high resolution T1 weighted structural MRI including the entire head is used to automatically determine the scalp surface, the electrode positions and source space points which are defined as regular distributed subset of the grey matter voxels of the cerebrum. Based on this information a Lead Field Matrix is calculated automatically using SMAC [1]. The head modelling procedure was evaluated using 14 MRIs. We visually assessed the position of the source space points and the electrodes. To show source localization accuracy, we compared localization results using the calculated head models with localization results using complex BEM head models [2] and clinical findings. The evaluation shows that our method is capable of automatically determine the electrode positions and the source space points with high accuracy. Localization of ictal and interictal events using automatically derived head models showed equivalent results compared to localization with BEM head models.


S37
Utilization of SSEPs Collision Test for Spinal Cord Stimulator Lead Placement
Ning Zhang, MD; Justin Cheongsattanay, MD; Pedro Coutin-Churchman, MD; Nader Pouratian, MD; Marc R. Nower, MD, FACNS

Background Spinal cord stimulation is a procedure involving direct epidural electrical stimulation to the dorsal column of the spinal cord. Utilization of spinal cord stimulators
(SCS) has become an evidence-based treatment for various chronic pain disorders. Optimization of SCS lead placement and appropriate stimulation of the dorsal column are required for optimal pain control. By strategically inducing collision of antidromic impulses from the SCS with orthodromic impulses evoked by peripheral nerve stimulation, we can optimize SCS placement by intraoperative monitoring somatosensory evoked potentials (SSEPs). Patients & Methods: Three patients with different pain presentation and pathology were studied. Both median and posterior tibial SSEPs monitoring were established after induction of anesthesia, SSEPs were collected continuously throughout the procedure. Collision tests were achieved by stimulating the dorsal column while recording both subcortical and cortical SSEPs. Results: Baseline median and posterior tibial SSEPs were within normal limits in all three patients. Continuous SSEP monitoring was reproducible and stable during the procedures. Positive collision was observed in all three cases. A significant unilateral reduction (50% or greater) or abolishment of SSEP cortical responses was interpreted as lateralized placements of the stimulating electrodes during the collision test. A reduction (50% or greater) or abolishment of SSEP cortical responses was interpreted as lateralized placements of the stimulating electrodes during the collision test. Successful SSEP’s collision may help to improve pain control outcomes.

S38
Dexmedetomidine in Spine Surgeries: A Friend or Foe?
Shanka Rajan, MD; Diana Braver, MD; Mirela V. Simon, MD, FACNS; Dinesh Nair, MD
Introduction: In this study, we report our experience on the effects of Dexmedetomidine on motor and somatosensory evoked potentials (MEPs and SSEPs) during intraoperative neuro-monitoring (IOM) for scoliosis surgery.
Method: Between 2010 and 2014 we identified 102 patients who underwent scoliosis surgery with IOM. Six of them received Dexmedetomidine at an infusion rate ranging from 0.2-1.0 mcg/kg/hr. Twelve age and sex-matched scoliosis patients, who did not receive Dexmedetomidine, was used as control.
Results: In 5/6 patients, MEPs amplitude on average decreased by 67% of baseline in the upper and 21% in the lower limbs, approximately three hours after starting Dexmedetomidine (Figure 1). Final MEPs recorded at maximal stimulation intensity, continued to remain suppressed, despite discontinuing Dexmedetomidine. In 1/6 patients, surgery lasted only 2h 45 min and the stimulus intensity to elicit MEPs changed from 85mA (baseline) to 180mA (end of surgery). SSEPs remained stable in all. Among control subjects, MEPs remained stable and at conclusion of surgery, stimulus intensity on average increased only 13.86% over baseline.
Conclusion: Our data does not support the use of Dexmedetomidine in surgeries requiring continuous monitoring of MEPs. More data needs to be analyzed to determine the dose-dependent effects of Dexmedetomidine on MEPs and SSEPs.

S39
Accidental Spinal Cord Contusions During Spine Deformity Surgeries
María del Mar Moreno, MD; Lidia Cabanes, MD; Gema de Blas, MD; Miguel Anton, MD; Vicente Garcia, MD; Jesus Burgos, MD
Introduction: Accidental spinal cord contusions are a rare event in the surgical correction of spinal deformities. We present a study in a large series of patients which underwent these surgeries with the aim of establish the clinical and neurophysiologic pattern of this complication, that to our knowledge is not yet well described in the literature.
Methods: Multicenter (5 centers), observational, retrospective (2008-2013) study. A total of 691 patients presenting complex spinal deformities who underwent posterior instrumented surgical correction were studied. Intraoperative neurophysiologic monitoring of spinal cord function was performed with motor (MEPs) and somatosensory (SSEPs) evoked potentials.
Results: 23 out of 691 patients (3%) suffered a spinal cord trauma, which become evident by a high blood pressure peak, as well as a neurophysiologic event that followed a constant sequential pattern. Ipsilateral MEPs were lost in the first place. Following that, contralateral MEPs were lost, and finally, SSEPs dropped. In the 19 cases (83%) with MEPs lost and preserved SSEPs, MEPs recovered during surgery. 4 (20%) of these patients presented a transient post-operative paresis (3-7 days) with complete recovery, and the rest were asymptomatic. In the four cases (17%) which presented complete loss of MEPs and significant changes in the SSEPs, the evoked potentials did not recover during the surgery, and the four patients presented some degree of post-operative paraparesis. Three of them were completely recovered after 3, 4 and 12 months respectively, whereas one patient presented only a partial recovery.
Conclusions: Intraoperative accidental spinal cord contusions which produce a selective MEPs loss with intraoperative recovery have an excellent prognosis. When the contusions also produce changes on the SSEPs, they have a worst outcome, and produce transient neurological sequelae.

S40
Proximal Weakness During Cervical Spine Surgery
Parastou Shilian, MD; Bong Su Kang, MD; Justin Cho, MD; Andres A. Gonzalez, MD
Introduction: Unilateral deltoid and/or biceps weakness is one of the complications of cervical spine surgery. It is reported to be more frequent for posterior (8.6-18.4%) than anterior approaches (1.6-12.1%). Etiology is thought to be a C5 root injury, however, the exact mechanism has not been established.
Materials and Methods: A retrospective chart review was performed of patients that had intraoperative monitoring at Keck Hospital of USC, January 2010 and June 2012. 243 patients had cervical spine surgery, four of which developed postoperative biceps and/or deltoid weakness.
Results: The incidence of upper extremity postoperative proximal weakness was 1.64% (4/243), 1.23% (2/162) for anterior and 2.46% (2/81) for posterior approaches. In three cases, intraoperative neurophysiologic monitoring did not detect any changes in SEP and MEP; however, EMG discharges were reported but not necessarily logged. In one case, there was a transient decrease amplitude in left hand and left lower extremity MEP. Of our four patients, one had deltoid, another had deltoid/triceps, and two had deltoid/biceps weakness. Patients developed
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weakness 1 to 2 days after surgery with recovery was seen within 1-5 months, although one was lost to follow up.

Discussion: This study shows that the use of monitoring is associated with lower incidences of proximal arm weakness in cervical procedures than reported. The variety of clinical presentations suggest different mechanisms of injury: C5 nerve root traction, brachial plexus injury, segmental spinal cord injury, and reperfusion injury. By understanding the mechanism, intraoperative monitoring could be tailored to detect such injuries and improve surgical outcomes. Segmental cord injury and axillary nerve compression may be better detected by MEP in the deltoid and biceps, while traction to the C5 root could be best detected using EMG. Further study is needed to better understand the mechanism of injury.

S41
Hemorrhagic Complications with Subdermal Needle Electrodes in Intraoperative Neurophysiologic Monitoring (IONM) of Endovascular Procedures
Eric Jones, MD; S Charles Cho, MD; Scheherazade Le, MD; Leslie Lee, MD; Viet Nguyen, MD; Jaime R. Lopez, MD, FACNS

IONM presents many unique challenges but is usually not associated with significant complications. Nonetheless, complications from IONM have been reported, amongst others, tongue bites and seizures from transcranial and cortical electrical stimulation, respectively. However, excessive and prolonged hemorrhage from routine subdermal needle placement has not been reported. We present a case series of 5 patients where the planned postoperative medical management caused unexpected, excessive needle-site hemorrhage, impacting postoperative care. All cases occurred during endovascular treatment of cerebrovascular disorders where high-dose combination antiplatelet agents were used. Neuroradiologists using somatosensory and motor evoked potentials (EPs), and EEG was performed in all patients, using standard subdermal needle electrodes. Discussion with the treating neuroradiologist and chart review identified a correlation between high dose anti-platelet treatment and post-procedure needle-site hemorrhagic complication. No short or long-term neurologic deficits or prolonged sequelae from bleeding were identified. Hemorrhagic short-term complications included subcutaneous hematomas, discomfort and patient concern. Treatment included prolonged manual pressure to bleeding sites and pressure head bandage. These short-term complications led us to change our IONM protocol, using only surface electrodes in high risk patients on planned high-dose anti-platelet therapy. We have eliminated post-procedure hemorrhagic complications while successfully recording EEG and EPs since the change.

S42
Pulse-train Stimulation Technique for Implantation of Thoracic Pedicle Screws: Preliminary Results of a Brazilian Monitoring Group
Paulo A. Kimaid, MD; Rafael De Castro, MD; Charles Nascimento, MD; Rodrigo N. Cardoso, MD; Rinaldo Claudino, MD; Marcondes Franca, MD

Purpose: The effectiveness of detecting mal-positioning of pedicle screws varies widely in the literature. Recently, a new technique of intraoperative neuromonitoring with high accuracy in preventing medial mal-positioning of thoracic pedicle screws was published. We present our preliminary results using the proposed technique in 20 cases of idiopathic scoliosis.

Methods: Patients were submitted to posterior arthrodesis and scoliosis correction under total intravenous anesthesia and intraoperative neuromonitoring with our standard protocol updated with the new multi-train stimulation technique (MTST). MTST consists of a repetitive train of pulses applied with a ball-tip probe in the track and in the implanted pedicle screw. Warn criteria were thresholds below 15mA for track and 30mA for screw evoking a muscle response in lower limb muscles. We analyzed the warn criteria, the surgeon decision and the outcome of the patient.

Results: From 315 tracks/screws tested, threshold was below the lower limit in 22, especially for T9 (6) and T8 (4) pedicle screws. In 9 cases (45%) the warn criteria was not achieved. The time expended to perform the MTST was between 3-5 seconds for each level. When warned, it was checked with radioscopy and surgeon feedback. Screws were repositioned successfully in 19 cases and due to their persistent lower thresholds, three screws were removed definitely. All patients awoke with no new deficits.

Conclusion: Despite the small number of cases the technique showed a good relation with medial mal-positioning of the probe or screw. It was not time consuming and the surgeons felt confident with the warn criteria, reducing the x-ray exposition. In our opinion the technique should be considered as a standard of care for the implantation of thoracic pedicle screws.

S43
Motor Evoked Potential Double Train Stimulation: Optimal Number of Pulses Per Train
Emily B. Kole, BS, CNIM Jose Devesa, MD; Aatif M. Hussien, MD, FACNS

Introduction: Double train stimulation for motor evoked potentials (MEP) consists of one train of testing stimuli and one train of facilitating stimuli. The optimal number of pulses per train has not been determined. We wanted to determine the optimal combination of pulse trains that produces the highest MEP amplitude.

Methods: Double train stimulation was attempted in 10 patients. Stimulation trains of 4 + 4 stimuli, 2 + 7 and 7 + 2 stimuli were attempted. The MEP response from the right foot muscles was used to determine the amplitude. Additionally, the MEP amplitude obtained with a single train of 7 stimuli was compared to the double trains.

Results: Trains with 2+7 pulses resulted in higher amplitudes than trains with 7 + 2 and 4 + 4 pulses. Single trains of 7 pulses resulted in amplitudes comparable to 7+2 double trains and amplitudes lower than 2+7 double trains.

Conclusions: A shorter initial priming train and a longer train for testing improved MEP responses. This can result in lower stimulation intensity and less patient movement due to MEP testing during surgery.

S44
Unexpected Neurophysiological Changes during Intracranial Revascularization Procedure
Holly G. MacCollum, MD; Parastou Shilian, DO; Andres A. Gonzalez, MD

Background: Cerebral hyperperfusion syndrome (CHS) is an uncommon complication of intracranial revascularization procedures. If untreated, CHS can result in devastating events including cerebral edema, seizures, and intracranial hemorrhage, and therefore must be promptly recognized.

Clinical presentation: 37 year old woman presented with a two month history of weakness and numbness on the right more than left upper extremity. MRI
showed bilateral watershed infarcts. Cerebral angiogram demonstrated bilateral supraclinoid ICA stenoses consistent with Moya Moya disease. Patient underwent a high flow extracranial to MCA bypass. Intraoperative monitoring included SEPs and MEPs. Following anastomosis, significant amplitude reduction was seen in the bilateral hand MEPs. Blood pressures had remained in the normal range during the intraoperative and postoperative periods. Postoperatively, patient had worsening bilateral arm weakness, blurry vision and headache. CT perfusion revealed increased rCBF suggesting hyperemia in the brainstem and bilateral occipital lobes. Esmolol drip was started, and within thirty minutes, patient’s symptoms resolved.

Discussion: Bilateral changes are often dismissed as they are not thought to be anatomical or in the corresponding vascular distribution at risk. Commonly, hyperemia occurs at the site of bypass, however, in our case, hyperemia additionally affected the posterior circulation. Due to the patient’s bilateral ICA stenoses, the posterior circulation was providing collateral flow. We postulate at the time of anastomosis, a change in blood flow dynamics resulted in hyperemia. Hyperemia is an uncommon complication that needs to be in the differential when changes occur in evoked potentials in order to prevent catastrophic complications.

Methods: Spinal cord was exposed through a large laminectomy in 13 experimental animals (domestic pigs) with mean weight of 35 kg. Dural sac (T7-T11) was exposed. The dural sac and spinal cord widths were measured at the level where the compression was going to be performed. Progressive compression of the spinal cord was performed with a precise compression device with a pair of parallel blades that were set up antero-posteriorly or to both sides of the spinal cord between T8 and T9 roots, and then sequentially approximated 0.25 mm every 2 minutes to cause a progressive cord compression. Epidural catheters were placed cranial and caudal to the compression level, and spinal cord to spinal cord evoked potential (EP), D-wave recordings and somatosensory epidural evoked potential (SSEP) were obtained for each approach of the sticks.

Results: Mean width of the dural sac was 7.5 mm. For progressive compression, increasing latency and decreasing amplitude of the evoked potentials were observed after a mean displacement of the sticks of 1.5±1 mm for the motor EP, 1.5±0.7 mm for the cord to cord EP, and 2.5±1.3 for the SSEP when provoking an antero-posterior compression; and 2.9±1.1 mm for the motor EP, 2.7±1 mm for the cord to cord EP, and 4.1±1.3 for the SSEP when performing the lateral compression.

Conclusion: The spinal cord is more sensitive to antero-posterior compression than to lateral compression. In both cases, cord to cord EP and D-wave are the first neurophysiologic parameters to detect the injury, whereas the SSEPs are less sensitive to compression. D-wave and cord to cord EP are equally accurate for the detection of spinal cord damage.

S46
Balloon Test Occlusion with IONM & Exams for Surgical Planning
Viet Nguyen, MD; David Morales, MD; S Charles Cho, MD; Leslie Lee, MD; Scheherazade Le, MD; Jaime R. Lopez, MD, FACS

Surgical treatment of head and neck tumors, vascular lesions, and traumatic injuries sometimes involves the sacrifice or permanent occlusion of an artery to the brain. Balloon test occlusions (BTOs) performed with intraoperative neurophysiologic monitoring (IONM) before sacrifice can help determine if there will be enough collateral blood flow to avoid cerebral infarction. We investigated whether combining neurophysiology awake physical examinations could improve the efficacy of BTOs. Seventy-three BTO procedures between 2005 and 2013 were examined. Fifty-five patients underwent awake physical exams and neurophysiologic testing. Eighteen patients under general anesthesia underwent neurophysiologic testing only. IONM changes during the BTO were identified, as well as immediate postoperative deficits related to the final procedure. One patient demonstrated IONM changes during the BTO and had postoperative deficits; 15 demonstrated changes but had no postoperative deficits; 49 neither demonstrated changes nor had postoperative deficits; and 8 did not demonstrate changes but did have postoperative deficits.

False negative outcomes were significantly lower (p=0.0042) when performed with physical examinations. IONM with BTOs can help decrease the risk of neurologic deficits from arterial sacrifice for vascular lesions and tumors. Adding physical exams to neurophysiologic testing decreases false negatives compared to neurophysiologic testing alone.
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The majority of procedures did not use total intravenous anesthesia and included a halogenated agent.

Conclusion: Tce-MEP may be safely performed in patients with seizures and epilepsy, including patients with medically refractory epilepsy. We advocate routine use of Tce-MEPs in this patient population.

S48
Diffuse Optical Monitoring of Spinal Cord Blood Flow and Oxygenation
Angela Kogler, MD; Thomas Floyd, MD

Introduction: Spinal cord ischemia can occur as a result of spinal trauma and spine and vascular surgery, often leading to paralysis or paresis. Somatosensory and motor evoked potentials are an indirect measure of spinal cord integrity. We have developed a prototype optical monitor, based on the principles of Diffuse Correlation Spectroscopy (DCS) and Diffuse Optical Spectroscopy (DOS), and hypothesized that this device would be capable of continuously monitoring changes in spinal cord blood flow and oxygenation concentration. This portable monitor could potentially be used intraoperatively and in the critical care units to immediately detect loss of blood flow to the spinal cord and to monitor the effects of interventions aimed at ameliorating ischemia.

Materials and Methods: We tested the efficacy of the device in 28 adult Dorsett sheep models by recording the response of spinal cord blood flow and oxygenation to pharmacological interventions and aortic occlusion. Temporal resolution in detecting ischemia after aortic occlusion was compared between the optical monitor and evoked potentials.

Results and Discussion: The monitor immediately detected increased blood flow (+51±11%) and oxygenation in response to hypertension and decreased flow (-39±11%) and oxygenation due to hypotension. Aortic occlusion resulted in an immediate fall in spinal cord blood flow and oxygenation. Upon aortic occlusion, the optical device detected a decrease in blood flow by 50% in 3 min whereas evoked potentials took 5 min - 22 min to notice a drop in signal.

Conclusions: Optical monitoring of spinal cord blood flow and oxygenation is feasible and the results of initial testing are promising. This monitoring tool potentially represents an important step forward, offering a new level of accuracy and immediacy in detecting spinal cord ischemia intraoperatively, and in the neurocritical care setting.

S49
IONM of Cranial Nerves Using EMG Hookwire Electrodes
Santiago Avilov, Jaime R. Lopez, MD, FACNS; S Charles Cho, MD; Leslie Lee, MD; Scheherazade Le, MD; Viet Nguyen, MD

Electromyography (EMG) can be used in intraoperative neurophysiologic monitoring (IONM) to monitor cranial nerves at risk during brainstem or cavernous sinus surgical procedures. The safety and clinical utility of EMG monitoring of cranial nerves III, IV, VI, and IX, which requires special hookwire electrodes, are not well studied. We hypothesize that cranial nerve EMG monitoring using hookwire electrodes is safe and useful in minimizing new neurological deficits. 78 cases from 2012 to 2014 were retrospectively examined. Preoperative and postoperative neurological deficits were compared, IONM changes were noted, and clinical complications were analyzed using electronic medical records. Complications were defined as any deviation from the normal perioperative course. Complications due to IONM, and more specifically due to the hookwire electrodes, were juxtaposed to other operative complications. A total of 19 cases with perioperative complications were recorded. Of these, 2 (2.6%) had complications due to the IONM hookwire electrodes — both perioperative ecchymosis. 17 (20.5%) had complications of non-IONM etiology. Cranial nerve monitoring using hookwire electrodes proved to be safe, and its clinical utility outweighs the risk for harm. IONM complications accounted for only a small amount of perioperative complications.

S50
Value of Stimulus-triggered EMG of Track vs Screw for the Detection of Lumbar Radiculopathy in Scoliosis Surgery.
Gema de Blas, MD; Lidia Cabanes, MD; Maria del Mar Moreno, MD; Guillermo Martin Palomeque, MD; Miguel Anton, MD; Vicente Garcia, MD; Jesus Burgos, MD

Question: One of the complications in scoliosis surgery are radiculopathies due to pedicular screw malposition. Our aim is to analyze our cases of lumbar radiculopathies after scoliosis surgery, in which screw malposition was not detected by the usual neurophysiologic techniques.

Methods: We have studied 294 surgeries (6765 screws, 30% lumbar). Intraoperative monitoring (IONM) was performed with muscular MEPs after rTES, SEP and t-EMG of the screws. 8 patients presented lumbar radiculopathy after surgery, despite t-EMG values were normal, and screw malposition was confirmed by CT-scans. In the surgery to withdraw these screws, t-EMG was determined after stimulation of the depth and the middle part of the track.

Results: All of the malpositioned screws showed normal t-EMG thresholds after stimulating the screw. When stimulating the depth of the track, values were also normal, but stimulation in the middle of the track showed very low thresholds in most of the cases. All of the patients improved with the screws withdrawal.

Conclusion: Malpositioned lumbar screws are infrequent, but very symptomatic. t-EMG of the track improves the detection of these cases.

S51
Intraoperative Vasoactive Treatment Reverses Loss of BAEP in MVD
Ning Zhong, MD; Rafael J. Lopez-Baquero, MD; Pedro Coutin-Churchman, MD; Marc R. Nuwer, MD, PhD, FACNS

Background Postoperative sensorineural hearing loss (up to 20%) is a major co-morbidity in microvascular decompression (MVD) procedure when surgically treating the patient with hemifacial spasm, trigeminal or geniculate neuralgia. For MVD procedure, there has been no study of utilizing perioperative vasoactive treatment, which has been established for vestibular schwannoma surgery to prevent post-operative hearing loss. Patient & Method A 58-year-old female with left facial twitching was found to have a prominent PICA with possible left CN VII compression. The patient underwent left retrosigmoid craniotomy MVD, intraoperative brainstem auditory evoked potential (BAEP) were monitored. Results Baseline latencies and amplitudes for BAEP waves I, III and IV/V were seen bilaterally within normal limits. During left brainstem MVD, an over 40% prolongation in absolute latencies for waves III, IV/V; and a 75% reduction of wave V amplitude were observed on the left side. Such changes were not reversed after releasing the retractor. Nimodipine was added into the surgical field, which resulted in normalization of latencies.
and amplitudes for left BAEP waves. Post-operatively the patient did not have hearing loss. Conclusion We observed that focally applying calcium channel blocker nimodipine helped reverse the loss of BAEP. This may have helped to preserve the patient’s hearing. Stretching the eighth nerve from cerebellar retraction is thought to be the most common mechanism of postoperative sensorineural hearing loss, and persistent loss of BAEP is an independent indicator of post-operative hearing loss. Our case suggested that microcirculation disturbance or vasospasm may also happen during MVD, which may be an additional pathophysiological factor for hearing loss in the patients undergoing vestibular schwannoma surgery. Local application of vasoactive treatment may be beneficial for hearing preservation in MVD.
EXHIBIT HALL & EVENING PROGRAMS

Exhibit Hall Hours
Friday, February 6, 2015
7:00AM – 5:00PM    Exhibit Hall Open
7:00 – 8:00AM    Continental Breakfast
10:00 – 10:20AM    Coffee Break
11:45AM – 1:00PM    Lunch
3:00 – 4:00PM    Coffee Break
7:00 – 8:00PM    Welcome Reception

Saturday, February 7, 2015
7:00 – 2:00PM    Exhibit Hall Open
7:00 – 8:00AM    Continental Breakfast
10:15 – 10:30AM    Coffee Break
12:45 – 2:00PM    Lunch

Exhibit Hall Floorplan

Exhibitors

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<td>ABCN/ABRET</td>
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<td>Ad-Tech Medical Instrument Corp</td>
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<td>Nihon Kohden America</td>
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<td>Persyst Development Corporation</td>
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<td>PMT Corporation</td>
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<td>Rhythmlink International, LLC</td>
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<td>Rochester Electro-Medical, Inc.</td>
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<td>Wolters Kluwer Health</td>
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</tbody>
</table>
ABERN/ABRET
2509 W. Iles Avenue, Suite 102
Springfield, IL 62704
Phone: 217.726.7980
Fax: 217.726.7989
www.theabcn.org
ABRET Neurodiagnostic Credentialing & Accreditation offers five credentials (R. EEG T®, R. EP T®, CNIM®, CLTM®, CAP) and three laboratory accreditation programs, LAB-EEG, LAB-NIOM, and LAB-LTM. Learn more about laboratory accreditation, view resources for technologists and receive assistance in recruiting neurodiagnostic technologists.

The American Board of Clinical Neurophysiology (ABCN) has a 65 year history of promoting excellence in Clinical Neurophysiology and offers examinations with added competency in Epilepsy Monitoring or Neurophysiologic Intraoperative Monitoring. A General Clinical Neurophysiology Track is also available. A Critical Care EEG Track will be offered in 2015. International testing is available.

Ad-Tech Medical Instrument Group
1907 William Street
Racine, WI 53404
Phone: 800.776.1555
Fax: 262.634.5668
customersupport@adtechmedical.com
www.adtechmedical.com

For over 25 years, Epilepsy Centers have made Ad-Tech their choice for invasive electrodes for brain mapping and epilepsy monitoring. We offer a large variety of electrodes and accessories to meet you and your patient’s needs. Visit our booth to discover why Ad-Tech is your best choice.

ACMEGS
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Milwaukee, WI 53202
Phone: 414.918.9804
Fax: 414.276.3349
info@acmegs.org
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 Road, Suite A
Kansas City, MO 64131-3019
Phone: 816.931.1120
Fax: 816.931.1145
info@aset.org
www.aset.org
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

Blackrock Neuromed
630 Kommas Drive, Suite 200
Salt Lake City, UT 84108
Phone: 801.994.5661
Fax: 877-623-6027
damon@blackrockneuromed.com
www.blackrockneuromed.com

Blackrock Neuromed’s Cervello® EEG/LTM systems provide advanced EEG technology including remote monitoring capabilities, Bluetooth connectivity and flexible integration and sampling rates for advanced research. All systems are supported with a comprehensive service package with start-to-finish integration, installation and expert IT support.

Cadwell Laboratories, Inc.
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Kennewick, WA 99336
Phone: 509.735.6481
Fax: 509.783.6503
info@cadwell.com
www.cadwell.com
Cadwell has been focused on the development of useful and innovative devices for neurophysiologists, physiatrists, neurologists, and technologists who want the best devices to provide superior patient care since 1979. Based in Kennewick, Washington, our products include the Cascade family for IONM, the Easy family for routine, ambulatory, LTM, and critical care EEG monitoring, the Easy PSG for in-lab and ApneaTrak for HST and the Sierra family for EMG, NCV and EP for both research and practice environments. We proudly introduce our new vision of EEG, the ARC family of products. Be sure to stop and take a look.
EXHIBIT HALL & EVENING PROGRAMS

Compumedics USA, Inc.
6605 W. WT Harris Boulevard
Charlotte, NC 28269
Phone: 704.714.3200
Fax: 704.714.3298
support@compumedics.com.au
www.compumedics.com.au

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.

Cortech Solutions
1409 Audubon Boulevard, Suite B1
Wilmington, NC 28403 USA
Phone: 910.362.1143
Fax: 910.378.3443
sales@cortechsolutions.com
www.cortechsolutions.com

Visit Cortech Solutions to learn about our wide range of sophisticated research and diagnostic EEG / EP / ERP systems and more. This year, we will highlight these g.tec products: g.Hiamp high-resolution EEG / ERP / ECoG system with 80 / 144 / 256 channels for research and diagnostic applications; cortiQ system for automated functional mapping with ECoG; mindBEAGLE system for screening, monitoring and communication in disorders of consciousness. We represent g.tec, Biosemi, Cambridge Research Systems and other leading brands from around the world. We look forward to meeting you!

Cyberonics
100 Cyberonics Boulevard
Houston, TX 77058
Phone: 800.332.1375
Fax: 281.218.9332
ir@cyberonics.com
www.cyberonics.com

Cyberonics, Inc. is the leader in device solutions for epilepsy and is committed to consistently delivering innovative and effective solutions for physicians, caregivers and people with epilepsy. Cyberonics’ VNS Therapy® is available in more than 70 countries for the treatment of drug-resistant epilepsy, with more than 80,000 patients implanted worldwide.

Electrical Geodesics, Inc.
1600 Millrace Drive
Suite 200
Eugene, OR 97403
Phone: 541.687.7962
Fax: 541.687.7963
jmterrell@gmail.com
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.

Elekta Neuromag
50-400 Perimeter Center Terrace
Atlanta, GA 30346
Phone: 770.300.9725
Fax: 770.448.6338
Info.america@elekta.com
www.elekta.com/neuromag

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.

Ives EEG Solutions
25 Storey Avenue, Suite 118
Newburyport, MA 01950
Phone: 978.358.8006
Fax: 978.358.7825
patrick@iveseegsolutions.com
www.iveseegsolutions.com

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.
EXHIBIT HALL & EVENING PROGRAMS

Lifelines Neurodiagnostic Systems, Inc.
411 Edwardsville Road, Suite A
Troy, IL 62294
Phone: 618.667.6445
Fax: 618.667.1982
sales@lifelinesneuro.com
www.lifelinesneuro.com

Cloud-based technology is the next step in the evolution of EEG, and Lifelines Neurodiagnostic Systems is leading the industry with innovative, new products. iEEG Cloud is a complete, video telemetry solution. Record and upload the EEG/video data to the Cloud for review over the internet in near-real time. We maintain servers with our hosted solution. Contact us at info@lifelinesneuro.com for more information.

MOBE, LLC
2020 Fieldstone Parkway
Suite 900-261
Franklin, TN 37069
Phone: 904.814.3764
info@mobemotion.com
www.mobeforlife.com

MOBE is a personalized program that empowers people on their path to a better life. Our ambition is to guide individuals toward enhanced physical, emotional, and social well-being. This involves engaging in simple, achievable, healthy lifestyle activities.

Moberg
224 S. Maple Street
Ambler, PA 19002
Phone: 215.283.0860
Fax: 215.283.0859
info@moberg.com
www.moberg.com

The Moberg 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 neurophysiological monitoring is available today with the CNS Monitor.

Natus
Corporate Headquarters
6701 Koll Center Parkway, Suite 120
Pleasanton, CA 94566 USA
Phone: 800.356.0007
Fax: 608.829.8775
madison.info@natus.com
www.natus.com

Natus Neurology is a leading provider of healthcare instrumentation used for the diagnosis and monitoring of neurological conditions include epilepsy, sleep disorders, cerebral vascular disorders and stroke, neuropathies, neuromuscular diseases, and myopathies, as well as for neurosurgical procedures and neurophysiologic research. In addition to computerized neurodiagnostic systems and software, Natus offers a complete line of supplies and accessories utilized in settings from private practice to hospitals. Natus product brands include: Bio-Logic, Dantec, Deltramed, Emblo, Grass, Nicolet, Schwarzer, Stellate, Teca, and Xltek. For more information, please visit us at www.natus.com

Neuralynx
105 Commercial Drive
Bozeman, Montana 59715
Phone: 406.585.4542
Fax: 866.585.1743

Neurotech
15285 Watertown Plank Road, Suite 2
Elm Grove, WI 53122
Phone: 262.754.0898
Fax: 262.754.0897
vwolfe@neurotecheeg.com
www.neurotecheeg.com

Nihon Kohden America
15353 Barranca Parkway
Irvine, CA 92618
Phone: 800.325.0283
Fax: 800.580.1550
info@nkusa.com
www.nkusa.com

EXHIBIT HALL & EVENING PROGRAMS

Persyst Development Corporation
12625 High Bluff Drive
Suite 213
San Diego, CA 92130
Phone: 858.461.4542
Fax: 858.461.4531
sale@persyst.com
www.persyst.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.

PMT Corporation
1500 Park Road
Chanhassen, MN 55317
Phone: 952.470.0866
Fax: 952.470.0865
info@pmtcorp.com
www.pmtcorp.com

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.

RhythmLink International, LLC
1140 First Street South
Columbia, SC 29209
Phone: 803.252.1222
Fax: 803.252.1111
sales@rhythmlink.com
www.rhythmlink.com

RhythmLink International is a medical device manufacturing company specializing in devices that connect patients to machines to record or elicit physiologic information. RhythmLink is the first to receive FDA clearance for Disposable MR Conditional Electrodes. Visit us to learn about the Disposable MR Conditional Cup, Webb and innovative PressOn™ Electrodes and our newly released MR Conditional LeadLock™.

Rochester Electro-Medical, Inc.
4212 Cypress Gulch Drive.
Lutz, FL 33559 USA
Phone: 813.963.2933
Fax: 813.994.8793
info@rochestermed.com
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.

Signal Gear, LLC
27 Sweetwater Drive
Prosperity, SC 29127
Phone: 630.272.5812
Fax: 800.878.9804
team@signalgear.com
www.signalgear.com

Signal Gear® is a medical device company focused on engineering innovative neurodiagnostic accessories. Founded on the premise that passionate, thoughtful study is key to innovation and creativity, we study the scientific literature, the patient, and you - the clinical practitioner. Let us demonstrate the advantages of our MEMG Needles and unique IONM angled HOOK Intra-Muscular Needle Pairs.

SpecialtyCare
3100 West End Avenue
Suite 800
Nashville, TN 37203
Phone: 615.345.5400
Fax: 615.345.5405
info@specialtycare.net
www.specialtycare.net

SpecialtyCare is the world’s leading provider of clinical services to hospitals. We partner with hospitals to drive, sustain, and accelerate high performance. We offer a portfolio of solutions that include IONM, Perfusion, Surgical Assist, Autotransfusion, Minimally Invasive Surgical Support, and Sterile Processing. SpecialtyCare serves more than 825 hospitals and health systems across a broad geographical area that includes 44 states, Puerto Rico, and Germany. We employ more than 1,500 clinicians who support over 350,000 surgical procedures annually.
**EXHIBIT HALL & EVENING PROGRAMS**

**Sunovion Pharmaceuticals**
84 Waterford Drive
Marlborough, MA 01752
Phone: 508.481.6700
joseph.felice@sunovion.com
www.sunovion.com

Aptiom (eslicarbazepine acetate) indicated for the treatment of partial onset seizure for epilepsy.

---

**UCB**
1950 E. Park Drive
Smyrna, GA 30080
Phone: 770.970.7500
Fax: 770.970.8857
uscommunications@ucb.com
www.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) is indicated as monotherapy or adjunctive therapy in patients with partial onset-seizures; Vimpat Injection is indicated as short term replacement when oral administration is not feasible.

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**Wolters Kluwer Health**
Two Commerce Square
2001 Market Street
Philadelphia, PA 19103
Phone: 612.259.8114
Fax: 612.677.3059
customerservice@lww.com
www.lww.com

Wolters Kluwer Health is a global provider of information, business intelligence and point-of-care solutions for the healthcare industry. Brands include Lippincott Williams & Wilkins, a leading international publisher of medical books, journals, and electronic media. We are the proud publisher of the Journal of Clinical Neurophysiology, official journal of the American Clinical Neurophysiology Society. We offer specialized publications and software for physicians, nurses, students and clinicians. Please visit our booth to browse our comprehensive product line.
EXHIBIT HALL & EVENING PROGRAMS

Evening Programs
ACNS is pleased to introduce the following Evening Programs. Each 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 – 7:00PM.

EEG REVIEW AND ANALYSIS USING PERSYST qEEG TRENDING, ARTIFACT REDUCTION, SEIZURE DETECTION, AND SPIKE DETECTION
Presented by: Persyst Development Corporation
Location: Salon B
Instructors: Mark L. Scheuer, MD, (Chief Medical Officer, Persyst)
            Linda Santilli-Mitchell, R.EEG.T. (Senior Clinical Application Specialist, Persyst)
            Additional presenters TBD

Through the use of sample EEG cases, participants in this session will gain basic practical experience with Persyst 12 software. An introductory talk will focus on EEG review utilizing qEEG trending graphs, automated detection algorithms, and artifact reduction. Workflow methods to streamline cEEG review will be discussed. A hands-on session will follow in which participants can use their own laptop computers to review and interpret several sample EEG studies, customize qEEG trend graphs, and interactively ask questions about what they’re seeing and best use practices. Illustrative case studies will conclude the session.
# Annual Meeting Overview

**Friday, February 6, 2015**

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<th>Time</th>
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<td>7:00 - 8:00AM</td>
<td>Breakfast — Visit Exhibits and Posters</td>
<td>Liberty Hall, 1st floor</td>
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<td>8:00 - 10:00AM</td>
<td>Concurrent Sessions:</td>
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<td>SEEG Stimulation</td>
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<td>Beyond Seizure Detection in Critical Care EEG Monitoring</td>
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<td>Pelvic Floor Neurophysiology (Joint ACNS/Mexican Society of Clinical Neurophysiology Symposium)</td>
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<td>10:00 - 10:20AM</td>
<td>Coffee Break — Visit Exhibits and Posters</td>
<td>Liberty Hall, 1st floor</td>
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<tr>
<td>10:20 - 11:45AM</td>
<td>General Session: Presidential Lecture, Gloor Award Presentation &amp; Lecture</td>
<td>Salon B, 2nd floor</td>
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<tr>
<td>11:45AM - 1:00PM</td>
<td>Lunch — Visit Exhibits and Posters</td>
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<td>1:00 - 3:00PM</td>
<td>Concurrent Sessions:</td>
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<td>Four Ways of Looking at Seizure Networks</td>
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<td>Myoclonic Status Following Cardiac Arrest</td>
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<td>Advanced Electrodiagnostic Techniques</td>
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<td>3:00 - 4:00PM</td>
<td>Coffee Break — Visit Exhibits and Poster Tours</td>
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<td>3:00 - 4:00PM</td>
<td>Special Interest Groups</td>
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<td>IOM</td>
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<td>ICU EEG: Periodic and Rhythmic Patterns</td>
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<td>Challenging EMG Cases</td>
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<tr>
<td>4:00 - 5:30PM</td>
<td>General Session: “Brain on Fire”</td>
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<td>5:30 - 7:00PM</td>
<td>Neurophys Bowl</td>
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<td>7:00 - 8:00PM</td>
<td>Welcome Reception</td>
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**Saturday, February 7, 2015**

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<th>Time</th>
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<tr>
<td>7:00 - 8:00AM</td>
<td>Breakfast — Visit Exhibits and Posters</td>
<td>Liberty Hall, 1st floor</td>
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<tr>
<td>8:00 - 9:30AM</td>
<td>Concurrent Sessions:</td>
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<td></td>
<td>Innovative Electronic Devices in Clinical Neurophysiology</td>
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<td>Electrocllinal Features of Autoimmune-Mediated Epilepsies</td>
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<td>Pedicle Screw Stimulation</td>
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<tr>
<td>9:30 - 10:15AM</td>
<td>General Session: Jasper Award Presentation &amp; Lecture</td>
<td>Salon B, 2nd floor</td>
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<tr>
<td>10:15 - 10:30AM</td>
<td>Coffee Break — Visit Exhibits and Posters</td>
<td>Liberty Hall, 1st floor</td>
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<tr>
<td>10:30 - 11:15AM</td>
<td>General Session: Schwab Award Presentation &amp; Lecture</td>
<td>Salon B, 2nd floor</td>
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<td>11:15AM-12:45PM</td>
<td>Concurrent Sessions:</td>
<td>Salon A, 2nd floor</td>
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<td>Peripheral Nerve Injury and Evaluation</td>
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<td>Probing Cortical Physiology: The Use of TMS-EEG in Epilepsy and Psychiatry</td>
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<td>Epilepsy Case Study in Video EEG Monitoring (Joint ACNS/Canadian Society of Clinical Neurophysiologists Symposium)</td>
<td>Salon D, 2nd floor</td>
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<td>12:45-2:00PM</td>
<td>Lunch — Visit Exhibits and Poster Tours</td>
<td>Liberty Hall, 1st floor</td>
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<td>2:00-3:30PM</td>
<td>Concurrent Sessions:</td>
<td>Salon A, 2nd floor</td>
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<td></td>
<td>Advanced Artifactology</td>
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<td></td>
<td>EEG — fMRI in Epilepsy (Joint ACNS/Brazilian Society of Clinical Neurophysiology Symposium)</td>
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<td>Evidence, Ethics and Epiphany in IOM</td>
<td>Salon D, 2nd floor</td>
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<td>3:30 - 4:30PM</td>
<td>Special Interest Groups</td>
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<td>Economics of MEG: How to Keep a MEG Center Afloat?</td>
<td>Salon B, 2nd floor</td>
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<td>Intracranial EEG</td>
<td>Salon C, 2nd floor</td>
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<td>Neonatal and Pediatric EEG Patterns That May be Clues to the Specific Diagnosis: Case Examples and Discussion</td>
<td>Salon D, 2nd floor</td>
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<td>4:30 - 6:00PM</td>
<td>Concurrent Sessions:</td>
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<td>Reflex Epilepsy: How to Define and Determine It</td>
<td>Salon B, 2nd floor</td>
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<td>Electroencephalography Overview and Future Directions</td>
<td>Salon C, 2nd floor</td>
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<td></td>
<td>Autonomic Function (Joint ACNS/IFCN Latin American Chapter Symposium)</td>
<td>Salon D, 2nd floor</td>
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<tr>
<td>6:00 - 7:00PM</td>
<td>Research Highlights</td>
<td>Salon B, 2nd floor</td>
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<tr>
<td>7:00 — 7:30PM</td>
<td>Annual Business Meeting</td>
<td>Salon B, 2nd floor</td>
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**Sunday, February 8, 2015**

<table>
<thead>
<tr>
<th>Time</th>
<th>Event</th>
<th>Location</th>
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<tbody>
<tr>
<td>7:00 - 8:00AM</td>
<td>Breakfast — Visit Exhibits and Posters</td>
<td>Salon B Foyer, 2nd floor</td>
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<td>8:00 - 9:30AM</td>
<td>Concurrent Sessions:</td>
<td>Salon A, 2nd floor</td>
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<td>Interooperative Communication Strategies: Case Workshop</td>
<td>Salon B, 2nd floor</td>
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<td>Skills Workshop: How to Record and Analyze Wide-Band EEG in the ICU</td>
<td>Salon C, 2nd floor</td>
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<td>Clinical Epilepsy: Slow Shifts and HFO</td>
<td>Salon D, 2nd floor</td>
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<td>Minimally Invasive Epilepsy Surgery: Case Based Discussion on the Role of Laser Ablation</td>
<td>Salon A, 2nd floor</td>
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<td>9:30 - 10:00AM</td>
<td>Coffee Break</td>
<td>Salon B Foyer, 2nd floor</td>
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<td>10:00 - 11:30AM</td>
<td>Concurrent Sessions:</td>
<td>Salon A, 2nd floor</td>
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<td>Clinical Neurophysiology Trials and Tribulations in the ICU</td>
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<td>A Practical Approach to Stereo EEG for Different Types of Focal Epilepsy</td>
<td>Salon C, 2nd floor</td>
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<td>When Surgery is not an Option, Neurostimulation and Beyond</td>
<td>Salon D, 2nd floor</td>
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</table>
SAVE THE DATE!

2016 ANNUAL MEETING & COURSES

February 10-14, 2016 • Orlando, FL
Hilton Orlando Lake Buena Vista

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