### ANNUAL COURSES OVERVIEW

**Wednesday, February 10, 2016**

<table>
<thead>
<tr>
<th>Time</th>
<th>Event</th>
<th>Location</th>
</tr>
</thead>
<tbody>
<tr>
<td>7:00 – 8:30AM</td>
<td>Evoked Potentials</td>
<td>Palm Ballroom 1</td>
</tr>
<tr>
<td>9:00 AM – 5:00PM</td>
<td>Neurophysiological Intraoperative Monitoring (NIOM): Part I</td>
<td>Palm Ballroom 1</td>
</tr>
<tr>
<td></td>
<td>Intensive Care Unit EEG Monitoring</td>
<td>Palm Ballroom 2</td>
</tr>
</tbody>
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**Thursday, February 11, 2016**

<table>
<thead>
<tr>
<th>Time</th>
<th>Event</th>
<th>Location</th>
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</thead>
<tbody>
<tr>
<td>7:00 - 8:30AM</td>
<td>Stereo-EEG</td>
<td>Grand Salon 1</td>
</tr>
<tr>
<td>9:00 AM – 10:30AM</td>
<td>Applied Cases in Central Neurophysiology and Video-EEG</td>
<td>Grand Salon 3</td>
</tr>
<tr>
<td>9:00AM – 5:00PM</td>
<td>Neurophysiological Intraoperative Monitoring (NIOM) Part II</td>
<td>Grand Salon 1</td>
</tr>
<tr>
<td>10:45AM – 12:45PM</td>
<td>Electrocorticography &amp; Invasive EEG</td>
<td>Grand Salon 2</td>
</tr>
<tr>
<td>11:30AM – 2:00PM</td>
<td>CNP Program Directors Symposium</td>
<td>Grand Salon 3</td>
</tr>
<tr>
<td>1:00 – 2:30PM</td>
<td>Autonomic Neurophysiology</td>
<td>Grand Salon 3</td>
</tr>
<tr>
<td>3:00 – 4:30PM</td>
<td>Business of Clinical Neurophysiology</td>
<td>Grand Salon 6</td>
</tr>
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**Friday, February 12, 2016**

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<thead>
<tr>
<th>Time</th>
<th>Event</th>
<th>Location</th>
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<tbody>
<tr>
<td>7:30 – 10:30AM</td>
<td>Video-EEG</td>
<td>Grand Salon 1</td>
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<td></td>
<td>EMG</td>
<td>Grand Salon 2</td>
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<tr>
<td></td>
<td>Neonatal &amp; Pediatric EEG</td>
<td>Grand Salon 3</td>
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</table>

### ANNUAL MEETING OVERVIEW

**Friday, February 12, 2016**

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<tr>
<th>Time</th>
<th>Event</th>
<th>Location</th>
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</thead>
<tbody>
<tr>
<td>7:30 – 10:30AM</td>
<td>Morning Workshops:</td>
<td>Grand Salon 4 &amp; 5</td>
</tr>
<tr>
<td></td>
<td>Stereoelectroencephalography (SEEG) Case-based Workshop</td>
<td>Grand Salon 4 &amp; 5</td>
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<tr>
<td></td>
<td>Electro-Clinical Approach to Epilepsy Surgery in the Setting of Multiple Lesions</td>
<td>Narcissus/Orange Blossom</td>
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<td></td>
<td>Electrocortical Functional Mapping: Tips, Techniques and Strategies in Adults and Children</td>
<td>Grand Salon 6</td>
</tr>
<tr>
<td>10:30 – 11:00AM</td>
<td>Exhibit and Poster Hall Open</td>
<td>International Ballroom</td>
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<tr>
<td>11:00AM – 12:45PM</td>
<td>Opening General Session: President’s Address &amp; Gloor Award Lecture</td>
<td>International Ballroom</td>
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<tr>
<td>12:45 – 2:00PM</td>
<td>Lunch — Visit Exhibits and Poster Tours</td>
<td>International Ballroom</td>
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<tr>
<td>2:00 – 3:30PM</td>
<td>Concurrent Sessions:</td>
<td>Grand Salon 4 &amp; 5</td>
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<tr>
<td></td>
<td>Pediatric EEG Patterns and Electroclinical Syndromes, Meanings and Realities in the Modern Era</td>
<td>Grand Salon 4 &amp; 5</td>
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<td></td>
<td>MEG: Workshop</td>
<td>Grand Salon 3</td>
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<tr>
<td></td>
<td>Intraoperative Motor Evoked Potentials Beyond the Standard Techniques (Joint ACNS/SBNC Symposium)</td>
<td>Grand Salon 2</td>
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<td></td>
<td>Non-peripheral Findings in ALS: Abnormal Excitability, Central Motor Conduction and Sleep Disorders (Joint ACNS/Mexican Clinical Neurophysiology Society Symposium)</td>
<td>Grand Salon 1</td>
</tr>
<tr>
<td>3:30 – 4:00PM</td>
<td>Coffee Break — Visit Exhibits and Posters</td>
<td>International Ballroom</td>
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<tr>
<td>4:00 – 5:30PM</td>
<td>Concurrent Sessions: Special Interest Groups</td>
<td>Grand Salon 1</td>
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<tr>
<td></td>
<td>NIOM</td>
<td>Grand Salon 2</td>
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<td></td>
<td>Imaging and Electrophysiology in Neuromuscular Diseases: A Case Based Approach</td>
<td>Grand Salon 2</td>
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<td></td>
<td>Future Methods of Payment Structures</td>
<td>Narcissus/Orange Blossom</td>
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<td>ICU EEG</td>
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<td></td>
<td>Invasive EEG</td>
<td>Grand Salon 4 &amp; 5</td>
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<tr>
<td>5:30 – 5:45PM</td>
<td>Walking Break</td>
<td>International Ballroom</td>
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<td>5:45 – 7:00PM</td>
<td>Neurophys Bowl</td>
<td>Grand Salon 4 &amp; 5</td>
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<tr>
<td>7:00 – 8:30PM</td>
<td>Welcome Reception</td>
<td>International Ballroom</td>
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**ACNS Executive Office**  
555 E. Wells St., Ste 1100  
Milwaukee, WI 53202  
Phone: (414) 918-9803  •  Fax: 414-276-3349  
info@acns.org  •  www.acns.org  
Megan M. Hille, CMP, Executive Director, mhiile@acns.org
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 2016 Annual Meeting & Courses in Orlando, Florida.

The Annual Courses, expertly fashioned by Dr. Tobias Loddenkemper and Dr. Saurabh R. Sinha will have commenced on Wednesday, February 10, 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, Brain Stimulation Technology hosted by Drs. Charles Epstein and Alexander Rotenberg, will be a great educational addition to the Annual Courses program.

The Annual Meeting begins on Friday, February 12 and continues through Sunday, February 14. The Program Committee, led by Dr. Devon I. Rubin and Dr. Stephan U. Schuele, 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 Symposium 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
Course Committee Co-Chair

Saurabh R. Sinha, MD, PhD, FACNS
Course Committee Co-Chair

Devon I. Rubin, MD, FACNS
Program Committee Co-Chair

Stephan U. Schuele, MD, MPH, FACNS
Program Committee Co-Chair
About the American Clinical Neurophysiology Society (ACNS)
ACNS is a professional association dedicated to fostering excellence in clinical neurophysiology and furthering the understanding of central and peripheral nervous system function in health and disease through education, research, and the provision of a forum for discussion and interaction.

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

Past Presidents
1947 *Herbert H. Jasper, MD, PhD
1948 *Herbert H. Jasper, MD, PhD
1949 Frederic A. Gibbs, MD
1950 *Hallowell Davis, MD
1951 *Robert Schwab, MD
1952 *James O’Leary, MD
1953 *Robert B. Aird, MD
1954 *Mary A.B. Brazier, DSc
1955 *A. Earl Walker, MD
1956 *Reginald G. Bickford, MD
1957 *John R. Knott, PhD
1958 *Robert S. Dow, MD
1959 *W. Theodore Liberson, MD
1960 *Arthur A. Ward, Jr., MD
1961 *Jerome K. Merlis, MD
1962 *Charles E. Henry, PhD
1963 *Cosimo Ajmone-Marsan, MD
1964 *Peter Kellaway, PhD
1965 *Donald B. Lindsay, PhD
1966 *David D. Daly, MD
1967 Kenneth A. Kooi, MD
1968 Gian-Emilio Chatrian, MD
1969 Robert J. Ellingson, PhD, MD
1970 Donald W. Klass, MD
1971 *Daniel Silverman, MD
1972 Eli S. Goldensohn, MD
1973 *Richard D. Walter, MD
1974 Janice R. Stevens, MD
1975 Ernst A. Rodin, MD
1976 *John S. Barlow, MD
1977 *Fernando Torres, MD
1978 *Frank Morrell, MD
1979 *Pierre Gloor, MD, PhD
1980 Richard N. Harner, MD
1981 Jack D. Grabow, MD
1982 Roger Q. Cracco, MD
1983 Cesare T. Lombroso, MD
1984 Robert J. Gurnit, MD
1985 Andrew J. Gabor, MD, PhD
1986 John A. Wada, MD
1987 Frank W. Sharbrough, MD,
1988 Joan B. Cracco, 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. Moshié, 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 Gareth 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
2014 Aatif M. Husain, MD, FACNS

* Deceased
Course Committee

Co-Chair:
Tobias Loddenkemper, MD, FACNS
Children's Hospital Boston
Saurabh R. Sinha, MD, PhD
Duke University Medical Center

Members:
Selim Benbadis, MD, FACNS
University of South Florida
Elliott Dimberg, MD
Mayo Clinic
Charles M. Epstein, MD, FACNS
Emory University
Cecil D. Hahn, MD, MPH, FACNS
Hospital for Sick Children
Susan T. Herman, MD, FACNS
Beth Israel Deaconess Medical Center
Aatif M. Husain, MD, FACNS
Duke University Medical Center
Lawrence J. Hirsch, MD, FACNS
Yale University
Jong Woo Lee, MD, PhD, FACNS
Brigham & Women's Hospital

Alan D. Legatt, MD, PhD, FACNS
Montefiore Medical Center
Jeffrey Liou, MD
Harvard Medical School
Jaime R. López, MD, FACNS
Stanford University
Michael McGurvey, MD, FACNS
Hospital of the University of Pennsylvania
Yafa Minazad, DO, FACNS
Southern California Neurology
Marc R. Nuwer, MD, PhD, FACNS
UCLA Medical Center
Phillip Pearl, MD, FACNS
Children's Hospital Boston
Claus Reinsberger, MD, PhD
University of Paderborn
Alexander Rotenberg, MD, PhD
Children's Hospital Boston
Elayna Rubens, MD, FACNS
Memorial Sloan Kettering Cancer Center
Devon I. Rubin, MD
Mayo Clinic
Mark Scher, MD
Rainbow Babies and Children’s Hospital
Michael R. Sperling, MD, FACNS
Thomas Jefferson University
Nitin Tandon, MD
University of Texas — Houston
William O. Tatum, IV, DO, FACNS
Mayo College of Medicine
Francis O. Walker, MD, FACNS
Wake Forest University
Courtney Wusthoff, MD
Stanford University

Ex-Officio:
Jeffrey Britton, MD, FACNS
Mayo Clinic
Gloria Galloway, MD, FACNS
Ohio State University
Stephan U. Schuele, MD, MPH, FACNS
Northwestern University

CME Committee

Chair:
Gloria Galloway, MD, FACNS

Members:
Nicholas S. Abend, MD, FACNS
Children's Hospital of Philadelphia
Meriem Bensalem-Owen, MD, FACNS
University of Kentucky
Jeffrey Britton, MD, FACNS
Mayo Clinic
Rohit Das, MD, FACNS
Indiana University

Charles M. Epstein, MD, FACNS
Emory University School of Medicine
Evan J. Fertig, MD
Northeast Regional Epilepsy Group
Susan T. Herman, MD, FACNS
Beth Israel Deaconess Medical Center
Pongkiat Kankirawatana, MD, FACNS
Children's of Alabama — UAB

Jong Woo Lee, MD, FACNS
Brigham & Women's Hospital
Mirela V. Simon, MD, FACNS
Massachusetts General Hospital
Saurabh R. Sinha, MD, PhD, FACNS
Duke University Medical Center
Christa Swisher, MD
Duke University Medical Center
ACNS PROGRAM COMMITTEE

Program Committee
Co-Chairs:
Devon I. Rubin, MD, FACNS
Mayo Clinic
Stephan U. Schuele, MD, MPH, FACNS
Northwestern University

Members:
Nicholas S. Abend, MD
Children’s Hospital of Philadelphia
Imran I. Ali, MD, FACNS
University of Toledo
Salah A. Almubarak, MD, FRCPC, FACNS
Royal University Hospital
Anto Bagic, MD, PhD, FACNS
University of Pittsburgh
Meriem Bensalem-Owen, MD, FACNS
University of Kentucky
Richard C. Burgess, MD, FACNS
Cleveland Clinic Epilepsy Center
Bernard Allan Cohen, PhD, FACNS
Neurological Monitoring Associates, LLC
Placido Coyac Cuautle, MD
Neurofisiologia Integral de Puebla
Rafael de Castro, MD
Neurolife Natal
Elliot Dimberg, MD
Mayo Clinic
Jonathan C. Edwards, MD, FACNS
Medical University of South Carolina
Ronald Emerson, MD, FACNS
Hospital for Special Surgery
William B. Gallentine, DO, FACNS
Duke University Medical Center
Cecil D. Hahn, MD, MPH, FACNS
The Hospital for Sick Children
Mark Hallett, MD, FACNS
National Institutes of Health
Abeer Hani, MD
Duke University Hospital Program
Danny Hilkmann, MD
Maastricht University Medical Centre
Aatif M. Husain, MD, FACNS
Duke University Medical Center
Akio Ikeda, MD, PhD
Kyoto University Graduate School of Medicine
Adam Juersivich, MD
University of Rochester School of Medicine
Mohammad MU Kabiraj, Sr., MBBS, PhD
Prince Sultan Military Medical City
Ioannis Karakis, MD, MSc
Emory University
Ekrem Kutluay, MD
Medical University of South Carolina
Gowri Lakshminarayanan, MD
Stanford University Medical Center
Suzette M. LaRoche, MD, FACNS
Emory University School of Medicine
Jong Woo Lee, MD, PhD, FACNS
Brigham & Women’s Hospital
Jaime R. Lopez, MD, FACNS
Stanford University
Daniela N. Minecan, MD
University of Michigan Health System
Heidi Munger Clary, MD, MPH
Wake Forest University
Christos Papadelis, PhD
Harvard Medical School
Eva K. Ritzl, MD
Johns Hopkins University
Mark Ross, MD, FACNS
Mayo Clinic Arizona
Raj D. Sheth, MD, FACNS
Mayo Clinic / Nemours Clinic-Florida
John Stern, MD
UCLA School of Medicine
William O. Tatum, DO, FACNS
Mayo College of Medicine
Amit Verma, MBBS
The Methodist Hospital
Courtney J. Wusthoff, MD
Stanford University
Ex-Officio:
Gloria Galloway, MD, FACNS
Ohio State University
Tobias Loddenkemper, MD, FACNS
Children’s Hospital Boston
Stephan U. Schuele, MD, MPH, FACNS
Northwestern University
GENERAL MEETING INFORMATION

<table>
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<td><strong>Tuesday, February 9, 2016</strong></td>
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<td><strong>Wednesday, February 10, 2016</strong></td>
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<td><strong>Thursday February 11, 2016</strong></td>
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<td><strong>Friday, February 12, 2016</strong></td>
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<td><strong>Saturday, February 13, 2016</strong></td>
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<td><strong>Sunday, February 14, 2016</strong></td>
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*Registration will be in the Palm Foyer on Wednesday, February 10.*

**Business Center**
The Hilton Orlando Lake Buena Vista has a full service Express Business Center located in the main lobby next to the concierge. Services include photo copying service, printers, fax machine and shipping/packaging.

**Business Meeting**
The ACNS Annual Business Meeting will be held in Grand Salon 4, from 7:00 — 7:30PM on Saturday, February 13, 2016. 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, 2016.

**Exhibits**
Those attending the Annual Meeting are encouraged to visit the Exhibit Hall located in International Ballroom. All meals and coffee breaks on Friday, February 12 and Saturday, February 13 will be held in the Exhibit Hall. Exhibit Hall hours are listed below:

- **Friday, February 12, 2016**
  - 10:00AM — 4:00PM  Exhibit Hall Open
  - 10:30 — 11:00AM  Coffee Break
  - 12:45 — 1:00PM  Lunch & Poster Tours
  - 3:30 — 4:00PM  Coffee Break

- **Saturday, February 13, 2016**
  - 7:00AM — 2:00PM  Exhibit Hall Open
  - 7:00 — 8:00AM  Continental Breakfast & Poster Tours
  - 9:30 — 10:30AM  Coffee Break
  - 12:45 — 2:00PM  Lunch

**Language**
English is the official language of the ACNS Annual Meeting & Courses. Select sessions will be presented in Spanish and are noted in the program.

**Lost & Found**
Please notify staff at the ACNS Registration Desk (International Ballroom Foyer) 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 pass 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 12:45 — 2:00PM on Friday, February 12 and 7:00 — 8:00AM on Saturday, February 13 for discussion. Poster abstracts and presentation dates can be found on page 36.

**Exhibit & Poster Hall, International Ballroom**
- **Friday, February 12, 2016**
  - 10:30AM — 4:00PM
- **Saturday, February 13, 2016**
  - 7:00AM — 2:00PM

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

Publication of Abstracts
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 (International Ballroom Foyer).

Venue Information
The Hilton Orlando Lake Buena Vista is the location for the 2016 Annual Meeting and Courses. Calls should be directed to the American Clinical Neurophysiology Society Registration Desk.
Hilton Orlando Lake Buena Vista
1751 Hotel Plaza Blvd
Lake Buena Vista, Florida 32830
(407) 827-4000

Wi-Fi Internet
Wireless internet access is available to Annual Meeting & Courses delegates throughout the meeting space. To access the internet, use the following network credentials: PSAV_Event Solutions
Password: ACNS2016
GENERAL MEETING INFORMATION

Nearby Restaurants

American
Earl of Sandwich (W)
1750 Buena Vista Dr.
407.938.1762

Miller’s Orlando Ale House (1.2 mi)
12371 Winter Garden Vineland Rd.
407.239.1800

Barbeque
American Q* (W)
1905 Hotel Plaza Blvd.
407.827.3080

International
Bahama Breeze (1.3 mi)
8735 Vineland Ave.
407.938.9010

Italian
Portobello’s* (W)
1650 Buena Vista Dr.
407.934.8888

Mexican
El Patron Mexican Restaurant & Cantina* (1.3 mi)
12167 S. Apopka Vineland Rd.
407.238.5300

Pal Campo Restaurant (1.9 mi)
13605 S. Apopka Vineland Rd.
407.778.4600

Seafood
The Boathouse* (W)
1620 Buena Vista Dr.
407.939.2628

Fulton’s Crab House* (W)
1670 Buena Vista Dr.
407.934.2628

Hemingway’s* (1.6 mi)
1 Grand Cypress Blvd.
407.239.1234

Landry’s Seafood (1.4 mi)
8800 Vineland Ave.
407.827.6466

Steakhouse
Johnnie’s Hideaway* (1.1 mi)
Crossroads, 12551 FL-535
407.827.1111

Hawk’s Landing Steakhouse & Grille* (3.3 mi)
8701 World Center Dr.
407.238.8829

Kobe Japanese Steakhouse (1.4 mi)
8460 Palm Pkwy.
407.239.1119

(W) — Walking Distance
(*) — Unique to Orlando

GENERAL MEETING INFORMATION

Discounted tickets to all Walt Disney World® theme parks available to all attendees and families.

Visit the Annual Meetings & Courses section of the ACNS website (www.acns.org/meetings/annual-meeting-and-courses/2016) or stop by the front desk for more information!

SUPPORT ACKNOWLEDGEMENT

The American Clinical Neurophysiology Society (ACNS) gratefully acknowledges Upsher-Smith Laboratories, Inc. for their support of the 2016 Annual Meeting in the form of an unrestricted educational grant.
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, neurosurgeons, trainees in these disciplines and other physicians and researchers who utilize clinical neurophysiological techniques and knowledge in the diagnosis and management of patients with disorders of the nervous system.

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

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

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

Gap 1. Emerging Areas of Practice
Neurophysiologic 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.
**Meeting Description**
The ACNS Annual Meeting & Courses are designed to provide a solid review of the fundamentals and the latest scientific advances in both “central” and “peripheral” clinical neurophysiology. Presentations at the Annual Meeting & Courses are given by leading experts in the field and have value for health care professionals who utilize clinical neurophysiology. Sessions include symposia, workshops, courses and Special Interest Groups, featuring didactic lectures, expert panels, debates and interactive formats. Poster presentations at the Annual Meeting highlight the latest work conducted at clinical neurophysiology centers around the country.

**Annual Courses Learning Objectives**
At the end of the Annual Courses, the participant will 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 18-22.

**Annual Meeting Learning Objectives**
At the end of the Annual Meeting, the participant will 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 25-35.

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

**Important Dates**
- CME Certificate Claim Deadline: April 15, 2016

**Credit Designation**
ACNS designates the Annual Meeting for a maximum 20.5 AMA PRA Category 1 Credit(s)™. Physicians should claim only credit commensurate with the extent of their participation in the activity.

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

**Evoked Potentials**
- 1.5 AMA PRA Category 1 Credit(s)™

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

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

**Intensive Care Unit EEG Monitoring (ICU EEG)**
- 6.5 AMA PRA Category 1 Credit(s)™

**Introduction Stereo-EEG**
- 1.5 AMA PRA Category 1 Credit(s)™

**Non-Invasive Brain Stimulation Technology**
- 1.5 AMA PRA Category 1 Credit(s)™

**Applied Cases in Central Neurophysiology & Video-EEG**
- 1.5 AMA PRA Category 1 Credit(s)™

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

**Electrocorticography (ECoG)/Invasive EEG**
- 6.5 AMA PRA Category 1 Credit(s)™

**Applied Cases in Peripheral Neurophysiology**
- 1.5 AMA PRA Category 1 Credit(s)™

**CNP Program Directors’ Symposium**
- 2 AMA PRA Category 1 Credit(s)™

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

**Business in Clinical Neurophysiology**
- 1.5 AMA PRA Category 1 Credit(s)™

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

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

**Tricky Neonatal & Childhood EEG Variants**
- 3 AMA PRA Category 1 Credit(s)™

Physicians should only claim credit commensurate with the extent of their participation in the activity.
CONFLICT OF INTEREST DISCLOSURES

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

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

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<tr>
<td>Nicholas Abend, MD, FACNS</td>
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<tr>
<td>Jeffrey Britton, MD, FACNS</td>
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<td>Richard Burgess, MD, PhD, FACNS</td>
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<td>Frank W. Drislane, MD, FACNS</td>
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<td>Jonathan Edwards, MD, FACNS</td>
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<td>Gloria Galloway, MD, FACNS</td>
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<td>Cecil Hahn, MD, MPH, FACNS</td>
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<tr>
<td>Selim Benbadis, MD, FACNS</td>
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<tr>
<td>Elliot Dimberg, MD</td>
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<td>Charles Epstein, MD, FACNS</td>
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<td>Susan Herman, MD, FACNS</td>
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<td>Lawrence Hirsch, MD, FACNS</td>
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<td>Jong Woo Lee, MD, PhD, FACNS</td>
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<td>Alan Legatt, MD, PhD, FACNS</td>
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<td>Jeffrey Liao, DO</td>
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<td>Jaime Lopez, MD, FACNS</td>
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<td>Michael McGarvey, MD, FACNS</td>
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<td>Yafa Minazad, DO, FACNS</td>
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<td>Phillip Pearl, MD, FACNS</td>
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<tr>
<td>Claus Reinsberger, MD, PhD</td>
<td>University of Paderborn</td>
<td>Sleepmed Inc. (b)</td>
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<td>Alexander Rotenberg, MD, PhD</td>
<td>Children’s Hospital Boston</td>
<td>Brainsway Inc. (a); Eisai Co. Ltd. (a); Neuro’motion Inc. (c); Neuroelectrics Inc. (a); NeuroRex Inc. (e)</td>
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<td>Elayna Rubens, MD, FACNS</td>
<td>Memorial Sloan Kettering Cancer Center</td>
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<td>Devon Rubin, MD</td>
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<td>AAN (g); AANEM (g);</td>
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<td>Mark Scher, MD</td>
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<td>Saurabh Sinha, MD, PhD, FACNS</td>
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<td>Cadwell, Inc. (e); Cybernics Inc. (a, g); Monteris, Inc. (b); UCB Pharmaceuticals (a); Upsher Smith Laboratories (a)</td>
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<tr>
<td>Michael Sperling, MD, FACNS</td>
<td>Thomas Jefferson University</td>
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<td>Nitin Tandon, MD</td>
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<td>Francis Walker, MD, FACNS</td>
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<td>Elsevier (g); Pfizer (a); Natus (b, g); Terason (g); Teva (a); UpToDate (g)</td>
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<td>Courtney Wusthoff, MD</td>
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<td>Imran Ali, MD, FACNS</td>
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<td>Salah Almubarak, MD, FRCP, FACNS</td>
<td>Royal University Hospital</td>
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<td>Anto Bagic, MD, PhD, FACNS</td>
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<td>Elekta Oy, Helsinky, Finland (g)</td>
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<td>Meriem Bensalem-Owen, MD, FACNS</td>
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<td>Bernard Cohen, PhD, FACNS, FASNM</td>
<td>Neurological Monitoring Associates, LLC</td>
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<td>Placido Cuautle, MD</td>
<td>Neurofisiologia Integral de Puebla</td>
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<td>Rafael da Castro, MD</td>
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<td>William Gallentine, DO, FACNS</td>
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<td>Jonathan Halford, MD</td>
<td>Medical University of South Carolina</td>
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<td>Abeer Honi, MD</td>
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<td>Danny Hilkman, MD</td>
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<tr>
<td>Akio Ikeda, MD, PhD</td>
<td>Kyoto University School of Medicine</td>
<td>Endowed Chair (g); Honorarium (g)</td>
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<tr>
<td>Adam Juersivich, MD</td>
<td>University of Rochester School of Medicine</td>
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<tr>
<td>Mohammad Kabiraj, MBB, MPhil, PhD, FACNS</td>
<td>Prince Sultan Military Medical City (PSMMC)</td>
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<td>Ioannis Karakis, MD, MSc</td>
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<td>Ekrem Kutluay, MD</td>
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<td>Gowri Lakshminarayan, MD</td>
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<td>Daniela Minecan, MD</td>
<td>University of Michigan Health System</td>
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<td>Heidi Munger Clary, MD, MPH</td>
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<td>Christos Papadeis, PhD</td>
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<td>Eva Ritzl, MD, FACNS</td>
<td>Johns Hopkins University</td>
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<tr>
<td>John Stern, MD</td>
<td>Geffen School of Medicine at UCLA</td>
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<tr>
<td>Amit Verma, MBBS</td>
<td>Houston Methodist Hospital</td>
<td>Lundbeck (b, g); Sunovion (b, g); Teva (g); UCB (b, g)</td>
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## CONFLICT OF INTEREST DISCLOSURES

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### CME Committee (if not listed above)

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<tr>
<th>Name</th>
<th>Institution</th>
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<tr>
<td>Rohit Das, MD, FACS</td>
<td>Indiana University Comprehensive Epilepsy Center</td>
<td>No Relationships</td>
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<tr>
<td>Evan J. Fertig, MD</td>
<td>Northeast Regional Epilepsy Group</td>
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<tr>
<td>Pongkit Kankirawatana, MD, FACS</td>
<td>Children’s of Alabama - UAB</td>
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<tr>
<td>Christa Swisher, MD</td>
<td>Duke University Medical Center</td>
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### Resident/Fellow Education Committee (if not listed above)

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<td>Pegah Afra, MD</td>
<td>University of Utah</td>
<td>Cyberonics (a); SAGE (a); Sunovion (a, d); UCB (a, b)</td>
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<td>Antoaneta Balabanov, MD</td>
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<td>Jean E. Cibula, MD</td>
<td>University of Florida</td>
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<td>Amy Crepeau, MD</td>
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<td>Eduardo Garcia, MD</td>
<td>Newton-Wellesley Hospital</td>
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<td>Gena Ghearing, MD, FACS</td>
<td>University of Pittsburgh Medical Center</td>
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<td>Andrea Hakimi, DO, FACS</td>
<td>University of Oklahoma</td>
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<td>Jeffrey Kennedy, MD</td>
<td>University of California, Davis Medical Center</td>
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<td>Lynn Liu, MD</td>
<td>University of Rochester School of Medicine</td>
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<td>Eika Pestana Knight, MD, FACS</td>
<td>Cleveland Clinic</td>
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<td>Mania C. Sam, MD, FACS</td>
<td>Wake Forest School of Medicine</td>
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<tr>
<td>Rani Sarks, MD, MSc</td>
<td>Brigham &amp; Women's Hospital</td>
<td>No Relationships</td>
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<tr>
<td>Norman K. So, MD</td>
<td>University of Washington</td>
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### Annual Course Directors and Faculty (if not listed above)

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<td>Edward Avila, DO</td>
<td>Memorial Sloan Kettering Cancer Center</td>
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<td>Deborah Briggs, MD, FACS</td>
<td>SBSI/UTSW - Austin</td>
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<td>Jan Claassen</td>
<td>Columbia University Medical Center</td>
<td>Actelion Pharmaceuticals (e); SAGE Therapeutics (e)</td>
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<td>Jay Gavvala, MD</td>
<td>Northwestern University</td>
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<td>Elizabeth Gerard, MD, FACS</td>
<td>Northwestern University</td>
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<td>Jorge Gonzalez-Martinez, MD</td>
<td>Cleveland Clinic</td>
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<td>Brent Goodman, MD</td>
<td>Mayo Clinic</td>
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<tr>
<td>Alexandra Hovagimian, MD</td>
<td>Beth Israel Deaconess Medical Center</td>
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<tr>
<td>Randa Jarrar, MD</td>
<td>Phoenix Children’s Hospital</td>
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<td>Lara Jehi</td>
<td>Cleveland Clinic</td>
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<tr>
<td>Barbara Jobst, MD</td>
<td>Dartmouth Hitchcock Medical Center</td>
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<tr>
<td>Giridhar Kalamangalam, MD, DPhil, FACS</td>
<td>University of Texas</td>
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<td>Emily Kale, BS</td>
<td>Duke University</td>
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<td>Ruple Loughlin, MD</td>
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<tr>
<td>George Lee, MD, AIS, FACS</td>
<td>Real Time Neuromonitoring Associates</td>
<td>See addendum.</td>
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<tr>
<td>Daniel Menkes, MD, FACS</td>
<td>William Beaumont Hospital</td>
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<tr>
<td>Pradeep Modur, MD, FACS</td>
<td>UT Southwestern Medical Center</td>
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<td>L. Elizabeth Mullikin, MPA FACHE</td>
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### CONFLICT OF INTEREST DISCLOSURES

\[ a. \text{Grants/Research Support}; b. \text{Consultant}; c. \text{Stock/Shareholder (self-managed)}; d. \text{Speaker's Bureau}; e. \text{Advisory Board or Panel}; f. \text{Salary, Contractual Services}; g. \text{Other Financial or material Support (royalties, patents, etc.)} \]

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<td>Alexander Rotenberg, MD, PhD</td>
<td>Children's Hospital Boston</td>
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<td>Demos Medical Publishing (g)</td>
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<td>Leo Happel, PhD, FACNS</td>
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<td>Mandy Harris, MD</td>
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<td>Luis Mayor, MD</td>
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**Staff**

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<td>Megan Hille, CMP</td>
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<td>Laura Konop</td>
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AWARD RECIPIENTS & LECTURES

Friday, February 12, 2016
11:00AM – 12:45PM
Location: Grand Salon 4

2016 Pierre Gloor Award Presentation & Lecture
“The Possibilities and Perils of Transcranial Brain Stimulation”
John Rothwell, PhD

The Gloor Award is presented annually for outstanding current contributions to clinical neurophysiology research. Prof. Rothwell will be recognized and will deliver the 2016 Gloor Address on Friday, February 12, 2016. Dr. Rothwell is currently a professor of Human Neurophysiology at UCL Institute of Neurology in London, England. He was also elected a Fellow of the Academy of Medical Sciences in 1994.

Saturday, February 13, 2016
10:00 – 11:00AM
Location: Grand Salon 4

2016 Herbert H. Jasper Award Presentation & Lecture
“My Memories of Dr. Jasper and My View of the Future of Clinical Neurophysiology as a Discipline”
Donald L. Schomer, 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. Schomer will be recognized during a general session on Saturday, February 13, 2016. Dr. Schomer is a professor of Neurology at Harvard University and director of clinical neurophysiology at Beth Israel Deaconess Medical Center in Boston, Massachusetts.

5:30 – 7:00PM
Location: Grand Salon 4

2016 Robert S. Schwab Award
“Livy and the Future of Clinical Neurophysiology”
Morris Fisher, MD, FACNS

The Schwab Award is presented annually to an individual who has made significant contributions in the area of clinical neurophysiology. Dr. Fisher will be recognized and deliver the 2015 Schwab Award address on Saturday, February 13, 2016. Dr. Fisher is a professor of neurology at Loyola University Medical Center in Chicago, Illinois. He completed a two-year epilepsy fellowship at the Graduate Hospital Comprehensive Epilepsy Center affiliated with the University of Pennsylvania. After training, he relocated to Tampa, Florida and pioneered the Tampa General Hospital-University of South Florida Comprehensive Epilepsy Center before joining the Mayo Clinic in 2009 where he serves as the Director of the Epilepsy Monitoring Unit.

He is board certified by the American Board of Psychiatry and Neurology (ABPN) in Neurology, Epilepsy, and Clinical Neurophysiology by the American Board of Clinical Neurophysiology (ABCN). He is currently a fellow in the American Academy of Neurology, the American Clinical Neurophysiology Society (ACNS), and the American Neurological Association. He has served on the board of directors for the American Board of Registration of EEG Technologists and Evoked Potentials and currently serves on the board of the Epilepsy Foundation of America. He is the past president of the board of the American Board of Clinical Neurophysiology and the current president of the American Clinical Neurophysiology Society. He has authored multiple abstracts, peer-reviewed editorials and journal articles, book chapters and has edited/co-edited several books in the field of clinical epilepsy and neurophysiology and is the Book Editor for the Journal of Clinical Neurophysiology. His research interests include seizure semiology, drug-resistant epilepsy, and EEG/clinical neurophysiology.

2016 President’s Address
Mobile Clinical Neurophysiologic Monitoring in Epilepsy
William O. Tatum, IV, DO, FACNS

Dr. William O. Tatum IV is a professor of Neurology in the Mayo Clinic College of Medicine. He is director of the epilepsy monitoring unit at Mayo Clinic in Florida. He attended medical school at the College of Osteopathic Medicine and Surgery in Des Moines, Iowa and neurology residency at Loyola University Medical Center in Chicago, Illinois. He completed a two-year epilepsy fellowship at the Graduate Hospital Comprehensive Epilepsy Center affiliated with the University of Pennsylvania. After training, he relocated to Tampa, Florida and pioneered the Tampa General Hospital-University of South Florida Comprehensive Epilepsy Center before joining the Mayo Clinic in 2009 where he serves as the Director of the Epilepsy Monitoring Unit.

He is board certified by the American Board of Psychiatry and Neurology (ABPN) in Neurology, Epilepsy, and Clinical Neurophysiology by the American Board of Clinical Neurophysiology (ABCN). He is currently a fellow in the American Academy of Neurology, the American Clinical Neurophysiology Society (ACNS), and the American Neurological Association. He has served on the board of directors for the American Board of Registration of EEG Technologists and Evoked Potentials and currently serves on the board of the Epilepsy Foundation of America. He is the past president of the board of the American Board of Clinical Neurophysiology and the current president of the American Clinical Neurophysiology Society. He has authored multiple abstracts, peer-reviewed editorials and journal articles, book chapters and has edited/co-edited several books in the field of clinical epilepsy and neurophysiology and is the Book Editor for the Journal of Clinical Neurophysiology. His research interests include seizure semiology, drug-resistant epilepsy, and EEG/clinical neurophysiology.

NETWORKING & SOCIAL EVENTS

Welcome Reception
Friday, February 12, 2016
7:00 – 8:30PM
Location: International Ballroom

Dr. William Tatum, DO, FACNS formally invites all Annual Meeting delegates to attend the ACNS Welcome Reception on Friday, February 12, from 7:00 – 8:30PM in the ACNS Exhibit Hall, International Ballroom. 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 14, 2016
7:00 — 8:00AM
Location: Azalea, 2nd Floor

If you signed up to be a mentor or mentee, there will be a designated meeting area in the Exhibit Hall on Friday and Saturday during breaks and lunches. Please look for the tables marked with balloons as a place to meet up!

Also, on Sunday morning, feel free to grab your coffee and bagel and head up to Azalea, on the 2nd floor, for an opportunity to network during breakfast.
## Annual Courses Overview

### Wednesday, February 10, 2016

<table>
<thead>
<tr>
<th>Time</th>
<th>Event</th>
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<tbody>
<tr>
<td>7:00 – 8:30AM</td>
<td>Evoked Potentials</td>
<td>Palm Ballroom 1</td>
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<td>EMG and EEG Technology</td>
<td>Palm Ballroom 2</td>
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<tr>
<td>9:00AM – 5:00PM</td>
<td>Neurophysiologic Intraoperative Monitoring (NIOM): Part I</td>
<td>Palm Ballroom 1</td>
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<td>Intensive Care Unit EEG Monitoring</td>
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### Thursday, February 11, 2016

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<tr>
<td>7:00 - 8:30AM</td>
<td>Stereo-EEG</td>
<td>Grand Salon 1</td>
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<td>Non-Invasive Brain Stimulation Technology</td>
<td>Grand Salon 2</td>
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<tr>
<td>9:00AM – 10:30AM</td>
<td>Applied Cases in Central Neurophysiology and Video-EEG</td>
<td>Grand Salon 3</td>
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<tr>
<td>9:00AM – 5:00PM</td>
<td>Neurophysiologic Intraoperative Monitoring (NIOM) Part II</td>
<td>Grand Salon 1</td>
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<td>Electrocorticography &amp; Invasive EEG</td>
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<tr>
<td>10:45AM – 12:45PM</td>
<td>Applied Cases in Peripheral Neurophysiology</td>
<td>Grand Salon 3</td>
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<td>11:30AM – 2:00PM</td>
<td>CNP Program Directors Symposium</td>
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<td>1:00 – 2:30PM</td>
<td>Autonomic Neurophysiology</td>
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<td>3:00 – 4:30PM</td>
<td>Business of Clinical Neurophysiology</td>
<td>Grand Salon 6</td>
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### Friday, February 12, 2016

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<tr>
<td>7:30 – 10:30AM</td>
<td>Video-EEG</td>
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<td>EMG</td>
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<td></td>
<td>Neonatal &amp; Pediatric EEG</td>
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### Evoked Potentials

**7:00 – 8:30AM**  
Location: Palm Ballroom 1  
**Co-Chairs:** Alan Legatt, MD, PhD, FACNS & Elayna Rubens, MD, FACNS

**Objectives:**
At the conclusion of this course, participants should be able to:
1. Identify the components of visual, somatosensory, and brainstem auditory evoked potentials;
2. Classify an evoked potential study as normal versus abnormal;
3. Provide an anatomical localization of the dysfunction when the evoked potential study is abnormal.

**Agenda:**
- 7:00AM  Brainstem Auditory Evoked Potentials (BAEPs)  
  Alan Legatt, MD, PhD, FACNS
- 7:30AM  Visual Evoked Potentials (VEPs)  
  Elayna Rubens, MD, FACNS
- 8:00AM  Somatosensory Evoked Potentials (SEPs)  
  Edward Avila, DO

### EMG and EEG Technology

**7:00 – 8:30AM**  
Location: Palm Ballroom 2  
**Co-Chairs:** Susan T. Herman, MD, FACNS & Seward Rutkove, MD

**Objectives:**
At the conclusion of this course, participants should be able to:
1. Describe the fundamental operation of neurophysiologic recording equipment, including differential amplifiers, common mode noise rejection, ground and analog and digital filters;
2. Explain the concepts of analog-to-digital conversion, aliasing and general frequency analysis;
3. Explain the concepts involved in electrical stimulation of nerve and muscle;
4. Evaluate and select neurophysiologic equipment based on knowledge of appropriate technical specifications for clinical or research use;
5. Appropriately select and utilize developing technologies for peripheral nerve and muscle assessment.

**Agenda:**
- 7:00AM  EMG Technology  
  Seward Rutkove, MD
- 7:45AM  EEG Technology  
  Susan T. Herman, MD, FACNS

### Neurophysiologic Intraoperative Monitoring (NIOM): Part I

**9:00AM – 5:00PM**  
Location: Palm Ballroom 1  
**Co-Chairs:** Aatif M. Husain, MD, FACNS & Michael McGarvey, MD, FACNS

**Objectives:**
At the conclusion of this course, participants should be able to:
1. 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;
2. Recognize changes in intraoperative neurophysiologic tests which indicate damage to neural structures, and distinguish these from common technical artifacts;
3. Communicate normal and abnormal results to the surgical team, and incorporate results into clinical recommendations that may alter the surgical technique to avoid, limit or reverse injury to neural structures;
4. Apply knowledge of advanced NIOM techniques, such as D wave recordings, brain mapping and other techniques to their practice.

**Agenda:**
- 9:00AM  SEP Monitoring  
  Aatif M. Husain, MD, FACNS
- 9:45AM  MEP Monitoring  
  Aatif M. Husain, MD, FACNS
- 10:25 Break
- 10:40AM  BAEP Monitoring  
  Alan Legatt, MD, PhD, FACNS
- 11:20AM  EEG Monitoring  
  Michael McGarvey, MD, FACNS
- 12:15PM Lunch (delegates on their own, see page 8 for nearby restaurants)
- 1:15PM  EMG and Peripheral Nerve Monitoring  
  Viet Nguyen, MD
- 1:55PM  Cranial Nerve Monitoring  
  Jaime R. Lopez, MD, FACNS
- 2:35PM  Troubleshooting  
  Emily B. Kale, BS, CNIM
- 3:15PM Break
- 3:30PM  Anesthesia  
  Steve Robicsek, MD, PhD
- 4:10PM  Billing Issues  
  Marc R. Nuwer, MD, PhD, FACNS
Intensive Care Unit EEG Monitoring  
9:00AM – 5:00PM  
Location: Palm Ballroom 2  
Co-Chairs: Cecil D. Hahn, MD, MPH, FACNS & Jong Woo Lee, MD, PhD, FACNS  
Objectives:  
At the conclusion of this session, participants should 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, FACNS  
9:40AM  Guidelines and Logistics of ICU EEG Monitoring  
Susan T. Herman, MD, FACNS  
10:20AM Break  
10:40AM  cEEG Interpretation: Assessment of Background, Sleep, Reactivity & Artifacts  
Suzette LaRoche, MD, FACNS  
11:10AM  cEEG Interpretation: The Ictal-Interictal Continuum  
Lawrence J. Hirsch, MD, FACNS  
11:40AM  cEEG Interpretation: Neonates  
Courtney J. Wusthoff, MD  
12:10PM Lunch (delegates on their own, see page 8 for nearby restaurants)  
1:10PM  Quantitative EEG for Ischemia Detection  
M. Brandon Westover, MD, PhD  
1:40PM  Quantitative EEG for Seizure Detection  
Cecil D. Hahn, MD, MPH, FACNS  
2:10PM  Finances, Billing & Coding  
Marc R. Nuwer, MD, PhD, FACNS  
2:40PM Break  
3:00PM  Treatment of Nonconvulsive Seizures, Status Epilepticus, and Postanoxic Myoclonus  
Jong Woo Lee, MD, PhD, FACNS  
3:30PM  Multimodality Monitoring in Acute Brain Injury  
Jan Claassen, MD, PhD  
4:00PM  ICU EEG Reading Session: Neonatal Cases  
Ronit Pressler, MD  
4:20PM  ICU EEG Reading Session: Pediatric Cases  
William B. Gallentine, DO, FACNS  
4:40PM  ICU EEG Reading Session: Adult Cases  
Elizabeth Gerard, MD, FACNS

Non-Invasive Brain Stimulation Technology  
7:00 – 8:30AM  
Location: Grand Salon 2  
Co-Chairs: Charles Epstein, MD, FACS and Alexander Rotenberg, MD, PhD  
Objectives:  
At the conclusion of this course, participants should be able to:  
1. Identify the basics of TMS and tDCS equipment and the physical principles that govern electrical intracranial electrical current distribution;  
2. Explain TMS and tDCS effects at the broad network level, and at the cellular level;  
3. Identify safety considerations in noninvasive brain stimulation;  
4. Explain the ways in which current knowledge, available technology, and physics guide experimental design and interpretation, focusing on several classic and contemporary TMS and TDCS experiments.  
Agenda:  
7:00AM  The Basics of TMS and TDCS: Circuits, Physics, and Specific Neuronal Elements  
Charles Epstein, MD, FACNS  
7:40AM  Putting the Pieces Together: Ion-Channels, Transmitters, Classic and Contemporary Studies in Non-Invasive Brain Stimulation  
Alexander Rotenberg, MD, PhD
**Program Agenda | Annual Courses**

**Applied Cases in Central Neurophysiology and Video-EEG**

9:00 – 10:30 AM  
**Location:** Grand Salon 3  
**Chairs:** William O. Tatum, IV, DO, FACNS & Selim Benbadis, MD, FACNS  

**Objectives:**  
At the conclusion of this course, participants should be able to:  
1. Recognize the features of epileptic seizures and their mimics and address management in the care of patients with this condition;  
2. Demonstrate knowledge in the clinical practice of approaching patients with seizures and spells;  
3. Evaluate the appropriateness of the clinical information as it applies to the care of patients.  

This session will be a fast-paced, interactive “show and tell” where audience participation is encouraged. Several challenging EEGs and videos will be presented and lively discussion between the session directors and the audience.

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

9:00 AM – 5:00 PM  
**Location:** Grand Salon 1  
**Chairs:** Aatif M. Husain, MD, FACNS & Michael McGarvey, MD, FACNS  

**Objectives:**  
At the conclusion of this course, participants should be able to:  
1. 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;  
2. Recognize changes in intraoperative neurophysiologic tests which indicate damage to neural structures, and distinguish these from common technical artifacts;  
3. Communicate normal and abnormal results to the surgical team, and incorporate results into clinical recommendations that may alter the surgical technique to avoid, limit or reverse injury to neural structures;  
4. Apply knowledge of advanced NIOM techniques, such as D wave recordings, brain mapping and other techniques to their practice.

**Agenda:**  
9:05 AM  Pre-Operative Motor Pathway Assessment  
**Gloria M. Galloway, MD, FACNS**  
9:45 AM  D Wave Monitoring with Case Discussions  
**Eva K. Ritzl, MD, FACNS**  
10:25 AM Break  
10:40 AM  Sensory and Motor Spinal Cord Mapping  
**Mirela V. Simon, MD, FACNS**  
11:00 AM  Monitoring During Movement Disorders Surgery  
**Jay L. Shils, PhD, D.ABNM, FACNS**  
12:15 PM Lunch (delegates on their own, see page 8 for nearby restaurants)  
1:15 PM  Pedical Screw Evaluation  
**Bernard Cohen, PhD, FACNS, FASNM**  
1:55 PM  Pelvic Floor Monitoring  
**Stanley Skinner, MD, FACNS**  
2:35 PM  Language and Motor Mapping  
**Jonathan C. Edwards, MD, FACNS**  
3:15 PM Break  
3:30 PM  Value of NIOM  
**John P. Ney, MD, MPH**  
4:10 PM  Medicolegal and Business Issues  
**George R. Lee, III, MD, MS, FACNS**

**Electrocorticography & Invasive EEG**

9:00 AM – 5:00 PM  
**Location:** Grand Salon 2  
**Chairs:** Stephan U. Schuele, MD, MPH, FACNS & Lawrence J. Hirsch, MD, FACNS  

**Objectives:**  
At the conclusion of this course, the participant should be able to:  
1. Identify patients from noninvasive evaluations that are good candidates for intracranial EEG evaluation;  
2. Decide which type of intracranial EEG recordings, if any, are most appropriate for a given patient, including consideration of risks of complications;  
3. Explain which invasive EEG patterns correlate with specific pathologies and which have prognostic importance;  
4. Understand techniques for mapping eloquent cortex via intracranial EEG stimulation and recordings;  
5. Understand the current state of knowledge regarding high frequency oscillations and DC shifts;  
6. Demonstrate an updated understanding of ongoing research into basic physiology of focal onset epilepsy based on invasive EEG techniques.

**Agenda:**  
9:15 AM  Non-Invasive Evaluations that Lead to Invasive EEG Testing  
**Giridhar Kalamangalam, MD, DPhil, FACNS**  
9:45 AM  Choosing Intracranial Electrodes  
**Stephan U. Schuele, MD, MPH, FACNS**  
10:15 AM  Surgical Techniques Including Risks of Invasive Implantations  
**Jorge Gonzalez-Martinez, MD, PhD**  
10:45 AM Break  
11:00 AM  Patterns of Seizure Onsets & Spread, Underlying Pathological Substrates, Surgical Outcomes in Adults  
**Lawrence J. Hirsch, MD, FACNS**  
11:30 AM  Discussion and Demonstrations of Patterns of Seizure Onsets & Spread, Underlying Pathological Substrates, Surgical Outcomes in Children  
**Tobias Loddenkemper, MD, FACNS**  
12:00 PM Lunch (delegates on their own, see page 8 for nearby restaurants)  
1:00 PM  Concept of “Epileptogenic Zones/Networks” and Identifying Them with Invasive EEG  
**Lara E. Jehi, MD**  
1:45 PM  Case Discussion I: Non-Lesional Neocortical Epilepsy  
**Jay Gavvala, MD**  
2:05 PM  Case Discussion II: FCD: ECoG vs. Invasive Implantation  
**Lawrence J. Hirsch, MD, FACNS**
2:25PM  Case Discussion III: Bi-Temporal Lobe Epilepsy
  Lawrence J. Hirsch, MD, FACNS

3:00PM  Break

3:15PM  Functional Mapping with Intracranial EEG
  Anthony Ritaccio, MD, FAAN, FANA

4:00PM  Wideband Intracranial EEG and Localization of Seizure Foci
  Gregory Worrell, MD

Applied Cases in Peripheral Neurophysiology
10:45AM — 12:45PM
Location: Grand Salon 3
Co-Chairs: Jaime R. López, MD, FACNS & Eliott Dimberg, MD

Objectives:
At the conclusion of this course, the participant 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:
10:45AM  Applied Cases in Peripheral Neurophysiology
  Seward Rutkove, MD
11:15AM  Applied Cases in Peripheral Neurophysiology
  Randa Jarrar, MD
11:45AM  Applied Cases in Peripheral Neurophysiology
  Elliot Dimberg, MD

CNP Program Directors Symposium
11:30AM — 2:00PM
Location: Grand Salon 6
Chair: Jeffrey 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.

Objectives:
At the conclusion of this course, participants should be able to:
1. Explain and understand recent changes to CNP fellowship program requirements;
2. Develop and employ milestone assessment approaches;
3. Recite the steps in conceptualizing and implementing a quality improvement project.

Agenda:
11:30AM  Welcome & Lunch
12:00PM  ACGME Program Requirement Review
  Jeffrey Britton, MD, FACNS
12:30PM  Milestone Update - Evaluations and Other Methods
  Ruple Laughlin, MD and Jeffrey Britton, MD, FACNS
1:00PM  Quality Improvement Projects
  Andrea Hakimi, DO, FACNS
1:30PM  ACGME Review Committee Q&A
  Imran Ali, MD

Autonomic Neurophysiology
1:00 — 2:30PM
Location: Grand Salon 3
Co-Chairs: Claus Reinsberger, MD, PhD and Jeffrey Liou, MD

Objectives:
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:
1:05PM  Autonomic Testing
  Jeffrey Liou, MD
1:35PM  Neurological Disorders with Central Autonomic Failure
  Alexandra Hovaguimian, MD
2:00PM  Peripheral Autonomic Failure
  Brent Goodman, MD
PROGRAM AGENDA | ANNUAL COURSES

**Business of Clinical Neurophysiology**
3:00 – 4:30PM  
Location: Grand Salon 3  
Co-Chairs: Marc R. Nuwer, MD, PhD, FACNS and Yafa Minazad, MD, FACNS

Objectives:
At the conclusion of this course, participants should be able to:
1. Identify the components that should go into Fair Market Value analysis for neurophysiologists;
2. Cost out services that are provided to the hospital as a neurophysiologist.

Agenda:
3:00PM What am I Worth: Overview of Fair Market Value  
   L. Elizabeth Mullikin, FACHE  
3:40PM Untangling Compensation  
   Pradeep Modur, MD, MS  
4:20PM Discussion

**Video-EEG**
Friday, February 12, 2016
7:30 – 10:30AM  
Location: Grand Salon 1  
Co-Chairs: Phillip Pearl, MD, FACNS and Michael Sperling, MD, FACNS

Objectives:
At the conclusion of this activity, participants will be able to:
1. Discuss the challenges and principles of the diagnosis of PNES;  
2. Review the principles of scalp video-EEG monitoring in epilepsy evaluation;  
3. Outline the limitations of non-invasive video-EEG monitoring; and  
4. Discuss the importance of safety during monitoring.

Agenda:
7:30AM Technical Aspects of Video EEG/Building and EMU  
   Barbara Jobst, MD  
8:00AM Psychogenic Nonepileptic Seizures  
   Selim R. Benbadis, MD, FACNS  
8:30AM Scalp Video EEG in Epilepsy Evaluation  
   Meriem Bensalem-Owen, MD, FACNS  
9:00AM Video EEG in Pediatrics  
   Phillip Pearl, MD, FACNS  
9:30AM Intracranial EEG and Video Correlation: HFOs Interictal and Ictal Events  
   Michael R. Sperling, MD, FACNS  
10:00AM Discussion

**EMG**
7:30 – 10:30AM  
Location: Grand Salon 2  
Co-Chairs: Devon Rubin, MD and Francis 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 evaluations of radiculopathies and polyneuropathies; and  
3. Recognize characteristic EMG patterns and approaches to neuromuscular junction disorders.

Agenda:
7:30AM Electrodiagnostic Assessment of CTS and Mononeuropathies  
   Francis O. Walker, MD, FACNS  
8:15AM Electrodiagnostic Evaluation of Polyneuropathy  
   Daniel L. Menkes, MD, FACNS  
9:00AM Electrodiagnostic Assessment of Radiculopathies  
   Seward Rutkove, MD  
9:45AM Electrodiagnostic Evaluation of Neuromuscular Junction Disorders  
   Devon Rubin, MD

**Tricky Neonatal & Pediatric EEG Variants**
7:30 – 10:30AM  
Location: Grand Salon 3  
Co-Chairs: Mark Scher, MD and Courtney Wusthoff, MD

Objectives:
At the conclusion of this activity, participants will be able to:
1. Recognize normal features of EEG unique to premature newborns, and distinguish those expected at different gestational ages;  
2. Identify abnormal findings on EEG for premature newborns;  
3. Evaluate recent data regarding the frequency of neonatal seizures in premature newborns.

Agenda:
7:30AM Neonatal EEG  
   Ronit M. Pressler, MD, PhD  
8:30AM Childhood “Epileptiform” Variants and Tricky Findings  
   Adam Hartman, MD  
9:30AM Childhood EEG Background & Common Abnormalities  
   Asim Shahid, MD
# Annual Meeting Overview

**Friday, February 12, 2016**

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<th>Time</th>
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<td>7:30 – 10:30AM</td>
<td>Morning Workshops:</td>
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<td></td>
<td>Stereoelectroencephalography (SEEG) Case-Based Workshop</td>
<td>Grand Salon 4 &amp; 5</td>
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<td>Electro-Clinical Approach to Epilepsy Surgery in the Setting of Multiple Lesions</td>
<td>Narcissus/Orange Blossom</td>
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<td>Electrocortical Functional Mapping: Tips, Techniques and Strategies in Adults and Children</td>
<td>Grand Salon 6</td>
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<td>10:30 – 11:00AM</td>
<td>Exhibit and Poster Hall Open</td>
<td>International Ballroom</td>
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<td>11:00AM – 12:45PM</td>
<td>Opening General Session: President’s Address &amp; Gloor Award Lecture</td>
<td>Grand Salon 4 &amp; 5</td>
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<td>12:45 – 2:00PM</td>
<td>Lunch — Visit Exhibits and Poster Tours</td>
<td>International Ballroom</td>
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<td>2:00 – 3:30PM</td>
<td>Concurrent Sessions:</td>
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<td>Pediatric EEG Patterns and Electroclinical Syndromes, Meanings and Realities in the Modern Era</td>
<td>Grand Salon 4 &amp; 5</td>
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<td>MEG: Workshop</td>
<td>Grand Salon 3</td>
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<td>Intraoperative Motor Evoked Potentials Beyond the Standard Techniques (Joint ACNS/SBNC Symposium)</td>
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<td>Non-peripheral Findings in ALS: Abnormal Excitability, Central Motor Conduction and Sleep Disorders (Joint ACNS/Mexican Clinical Neurophysiology Society Symposium)</td>
<td>Grand Salon 1</td>
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<td>3:30 – 4:00PM</td>
<td>Coffee Break — Visit Exhibits and Posters</td>
<td>International Ballroom</td>
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<td>4:00 – 5:30PM</td>
<td>Concurrent Sessions: Special Interest Groups</td>
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<td>NIOM</td>
<td>Grand Salon 1</td>
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<td>Imaging and Electrophysiology in Neuromuscular Diseases: A Case Based Approach</td>
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<td>Future Methods of Payment Structures</td>
<td>Narcissus/Orange Blossom</td>
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<td>ICU EEG</td>
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<td>Invasive EEG</td>
<td>Grand Salon 4 &amp; 5</td>
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<td>5:30 – 5:45PM</td>
<td>Walking Break</td>
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<td>5:45 – 7:00PM</td>
<td>Neurophys Bowl</td>
<td>Grand Salon 4 &amp; 5</td>
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<tr>
<td>7:00 – 8:30PM</td>
<td>Welcome Reception</td>
<td>International Ballroom</td>
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**Saturday, February 13, 2016**

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<th>Time</th>
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<tr>
<td>7:00 – 8:00AM</td>
<td>Continental Breakfast — Visit Exhibits and Poster Tours</td>
<td>International Ballroom</td>
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<td>8:00 – 9:30AM</td>
<td>Concurrent Sessions:</td>
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<td></td>
<td>Montage Matters: Designing EEG Montages for Optimal Localization</td>
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<td>Stand-Alone Home Video: A New Tool for the Diagnosis of Seizures?</td>
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<td>Interpreting Challenging Electrophysiological Findings - An Interactive Case-Based Approach</td>
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<td>Sleep and Sudden Death</td>
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<td>9:30 – 10:30AM</td>
<td>Coffee Break — Visit Exhibits and Posters</td>
<td>International Ballroom</td>
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<td>10:00 – 11:00AM</td>
<td>General Session: Travel Award Presentation &amp; Jasper Award Lecture</td>
<td>Grand Salon 4 &amp; 5</td>
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<td>11.00 – 11:15AM</td>
<td>Walking Break</td>
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<td>11:15AM – 12:45PM</td>
<td>Concurrent Sessions:</td>
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<td>New Insights in the Mechanism of Sudden Unexpected Death in Epilepsy</td>
<td>Grand Salon 4 &amp; 5</td>
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<td>The New Landscape of Neurostimulation a Myriad of Choices</td>
<td>Grand Salon 1</td>
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<td>Can Clinical Neurophysiology Makes Sports Healthier (and Better)?</td>
<td>Grand Salon 2</td>
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<td>History and Future of Clinical Neurophysiology</td>
<td>Grand Salon 3</td>
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<td>12:45 – 2:00PM</td>
<td>Lunch — Visit Exhibits and Poster Tours</td>
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<td>2:00 – 3:30PM</td>
<td>Concurrent Sessions:</td>
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<td>Clinical Neurophysiology Trials and Tribulations in Neurocritical Care</td>
<td>Grand Salon 4 &amp; 5</td>
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<td>Remote EEG Monitoring: Building a Network</td>
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<td>Toxic and Drug Induced Peripheral Neuropathies</td>
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<td>REM Sleep Behavior Disorder and REM Sleep without Atonia: Diagnosis and Treatment</td>
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<td>Walking Break</td>
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<td>3:45 – 5:15PM</td>
<td>Concurrent Sessions: Special Interest Groups</td>
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<td>The Wisdom and Vision from the ACMEGS Inaugural Decade</td>
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<td>Non-invasive Methods of Cortical Mapping</td>
<td>Grand Salon 4 &amp; 5</td>
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<td>Neonatal and Infantile EEG and Seizure Patterns: When do we Need an EEG?</td>
<td>Grand Salon 1</td>
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<td>The EEG a Great Tool (Note: This session will be in Spanish)</td>
<td>Grand Salon 3</td>
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<td>5:15 – 5:30PM</td>
<td>Walking Break</td>
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<td>5:30 – 7:00PM</td>
<td>General Session: Research Highlights &amp; Schwab Award Lecture</td>
<td>Grand Salon 4 &amp; 5</td>
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<td>7:00 – 7:30PM</td>
<td>Annual Business Meeting</td>
<td>Grand Salon 4 &amp; 5</td>
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<td><strong>Sunday, February 14, 2016</strong></td>
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<tr>
<td>7:00 – 8:00AM</td>
<td>Continental Breakfast</td>
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<td>8:00 – 9:30AM</td>
<td>Concurrent Sessions:</td>
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<td>Functional Neurosurgery and NIOM — From Spine to Brain</td>
<td>Grand Salon 1</td>
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<td>The Future of EMG in the Current Medical Environment</td>
<td>Grand Salon 2</td>
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<td>Multimodal Strategies in Surgical Epilepsy Planning (Estrategias Multimodales en la Planeación de Cirugía de Epilepsia) (Joint ACNS/Latin American Chapter of IFCN) (Note: This session will be presented in Spanish)</td>
<td>Grand Salon 3</td>
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<td>New Frontiers in Electrocorticography</td>
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<td>9:30 – 10:00AM</td>
<td>Coffee Break</td>
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<td>10:00 – 11:30AM</td>
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<td>Clinical Neurophysiology of Insular Cortex Epilepsy</td>
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<td>Functional Brain Mapping: Overview and Future Directions</td>
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<td>What Can We Learn from the Postictal State?</td>
<td>Grand Salon 3</td>
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Friday, February 12, 2016

WORKSHOPS
7:30 – 10:30AM

**Electro-clinical Approach to Epilepsy Surgery in the Setting of Multiple or Multilobar Lesions**
7:30 – 9:00AM*
Location: Narcissus/Orange Blossom, 2nd Floor
Chair: Ahsan Moosa Naduvil Valappil, MD

**Objectives:**
At the conclusion of this session, participants should be able to:
1. Identify potential candidates for epilepsy surgery who do not require invasive monitoring despite multilobar lesions;
2. Recognize the various approaches to invasive monitoring in the setting of multilobar lesions;
3. Appreciate the benefits and pitfalls of intra-operative electrocorticogram in the setting of multilobar/multifocal lesions.

**Agenda:**
7:30AM Electro-Clinical Features of Refractory Epilepsy Due to Multi-Lobar/Large Hemispheric Lesions in Young Children
Elaine Wyllie, MD
8:00AM Multi-lobar/multiple Lesions: Any Role for Limited Resection Using Extra/Intra-Operative Electrocorticogram
Douglas R. Nordli, MD, FACNS
8:30AM Multiple/Multi-Lobar Lesions: Any Role for Limited Resection Using Stereoelectroencephalogram?
Ahsan Moosa Naduvil Valappil, MD

**Electrocortical Functional Mapping: Tips, Techniques and Strategies in Adults and Children**
Location: Grand Salon 6
Co-Chairs: David Gloss, MD and Elson Lee So, MD, FACNS

**Objectives:**
At the conclusion of this session, participants should be able to:
1. Conduct ECSM within the safety limits of the procedure, and yet optimize the ability to determine the presence or absence of cortical function;
2. Recognize after discharges and the expected normal and abnormal background from different areas of the cortex;
3. Know the differences in cortical excitability and responses between children and adults, and employ the correct techniques to maximize the outcome and safety of the procedure in children;
4. Use specific language testing paradigms and materials for evaluating the presence or absence of language function at the cortical areas tested;
5. Employ measures to address the occurrence of after discharges, especially when widespread or frequent.

Agenda:
7:30AM Introduction
7:40AM Electrocorticography for Functional Mapping: The Evidence
David Gloss, MD
8:20AM Electrocortical Functional Mapping: Special Issues in Children
Prasanna Jayakar, MD, PhD
8:50AM Question & Answer
9:10AM Electrocorticography for Functional Mapping: Step-by-Step Strategy
Elson Lee L. So, MD, FACNS
9:40AM Adult Case Discussion
Eva K. Ritzl, MD, FACNS
10:00AM Cases of Direct Cortical SSEPs for Sensorimotor Mapping
Mirela V. Simon, MD, MSc, FACNS
10:20AM Discussion and Closing Remarks

**Stereoelectroencephalography (SEEG) Case-Based Workshop**
Location: Grand Salon 4 & 5
Co-Chairs: Giridhar Kalamangalam, MD, DPhil, FACNS and Stephan Schuele, MD, MPH, FACNS

**Objectives:**
At the conclusion of this session, participants should be able to:
1. Appreciate the typical indications for SEEG in surgical epilepsy syndromes;
2. Obtain an idea for hypothesis generation for selected electroclinical syndromes;
3. Understand SEEG data interpretation;
4. Appreciate the planning of surgical resections based on SEEG data.

Agenda:
7:30AM Introduction & Case I
Giridhar Kalamangalam, MD, DPhil, FACNS
8:15AM Case II
Stephen Thompson, MD
8:45AM Surgical Aspects and Electrode Visualization
Nitin Tandon, MD
9:15AM Case III
Christopher Skidmore, MD
9:45AM Case IV & Expert Panel
Stephan Schuele, MD, MPH, FACNS

**COFFEE BREAK – Visit Exhibits & Posters**
10:30 – 11:30AM
Location: International Ballroom
PROGRAM AGENDA | ANNUAL MEETING

OPENING GENERAL SESSION: PRESIDENT'S ADDRESS & GLOOR AWARD LECTURE
11:00AM – 12:45PM
Location: Grand Salon 1
Co-Chairs: Devon Rubin, MD, FACSNS and Stephan U. Schuele, MD, MPH, FACSNS
Agenda:
11:00AM Introduction of ACNS President
Jonathan C. Edwards, MD, FACNS
11:15AM Presidential Lecture: Mobile Clinical Neurophysiologic Monitoring in Epilepsy
William O. Tatum, DO, FACNS
11:55AM Introduction and Gloor Award Presentation
12:00PM Gloor Award Lecture: The Possibilities and Perils of Transcranial Brain Stimulation
John Rothwell, PhD

LUNCH — Visit Exhibits & Poster Tours
12:45 – 2:00PM
Location: International Ballroom

CONCURRENT SESSIONS
2:00 – 3:30PM
Pediatric EEG Patterns and Electroclinical Syndromes, Meanings and Realities in the Modern Era
Location: Grand Salon 4 & 5
Chair: Heather Olson, MD and Elaine Wyllie, MD

Objectives:
At the conclusion of this session, participants should be able to:
1. Recognize hypsarrhythmia patterns and its variant and the importance of these patterns for the treatment algorithm;
2. Identify the clinical and electroencephalographic features that distinguish CDKL5 disease from other genetic epilepsies with onset in the first years of life;
3. Discuss the electroencephalographic characteristics of migrating partial seizures of infancy and etiological work up;

Agenda:
2:00PM Hypsarrhythmia Classic and Modified Variations: Implications for Current Clinical Practice
John J. Millichap, MD
2:20PM CDKL5 disease: Seizure Types and EEG Markers of the Disease
Heather Olson, MD
2:40PM Migrating Partial Epilepsy of Infancy: Implications for Etiological Diagnosis
Elaine Wyllie, MD
3:00PM Panayiotopoulos Syndrome: Expanding the Clinical Spectrum or When Not to Treat!
Christelle Moufaward El Achkar, MD
3:20pm Discussion

Intraoperative Motor Evoked Potentials Beyond the Standard Techniques (Joint ACNS/Brazilian Society of Clinical Neurophysiology Symposium)
Location: Grand Salon 2
Chair: Paulo Kimaid, MD, PhD

Objectives:
At the conclusion of this session, participants should be able to:
1. Describe each MEP techniques and paradigms;
2. Describe the potential role of such techniques to IONM;
3. Identify the pros and cons of each technique.

Agenda:
2:00PM Transcranial MEP: Single or Double Trains
Aatif M. Husain, MD, FACNS
2:20PM Transcranial MEP: Utility of Recording From Cranial Nerve Innervated Muscles
Paulo A. Kimaid, MD, PhD
2:40PM Spinal MEP or Multipulse Thoracic Pedicle Track Stimulation Utility
Rafael de Castro, MD
3:00PM Direct Cortical Stimulation MEP
Aatif M. Husain, MD, FACNS
3:20pm Discussion

Non-Peripheral Findings in ALS: Abnormal Excitability, Central Motor Conduction and Sleep Disorder (Joint ACNS/Mexican Clinical Neurophysiology Society Symposium)
Location: Grand Salon 1
Chair: Jaime Ramos-Peek, MD

Objectives:
At the conclusion of this session, participants should be able to:
1. Understand the clinical importance of extra peripheral ALS symptoms;
2. Elaborate on different clinical neurophysiology tests used to evaluate non-peripheral ALS signs and symptoms;
3. Increase the knowledge of non-peripheral ALS signs and symptoms.

Agenda:
2:00PM Motor Evoked Potentials in ALS Patients
Armando Tello-Valdes, MD, PhD
2:25PM Upper Motor Neuron Effects on the EMG of ALS Patients
Jorge Burgos, MD
2:50PM Sleep Breathing Disorders in ALS
Jaime Ramos-Peek, MD
3:15PM Discussion
Jaime Ramos-Peek, MD
MEG Workshop
Location: Grand Salon 3
Chair: Richard Burgess, MD, PhD, FACNS
Objectives:
At the conclusion of this session, participants should be able to:
1. Recognize when and how a MEG will be helpful in the evaluation of their epilepsy patients;
2. Identify what to expect from a MEG report, as well as what information to provide to the magnetoencephalographers;
3. Interpret the information and figures received in a MEG report;
4. Incorporate the results of the MEG into the care plan for the patient, and understand how to handle differences between the MEG findings and those from other studies.

Agenda:
2:00PM  Indications for MEG: What Should the Referring Physician Know About MEG? When and Why to Order a MEG?  
Robert C. Knowlton, MD, MSPH
2:25PM   MEG Results: What Should the Referring Physician Expect from the MEG Report? How to Ascertain that the Abnormalities Recorded in the MEG Were the Same as Those Recorded in the Requestor's Office?  
Richard C. Burgess, MD, PhD, FACNS
2:50PM  How the MEG Results Fit Into the Workup: Multi-Modality Data Concordance, Practical Image Integration  
Anto Bagic, MD, PhD, FACNS
3:15PM   Panel Discussion

COFFEE BREAK – Visit Exhibits & Posters
3:30 – 4:00PM
Location: International Ballroom

SPECIAL INTEREST GROUPS
4:00 – 5:30PM
Neurophysiologic Intraoperative Monitoring (NIOM)
Location: Grand Salon 1
Chair: Jaime R. Lopez, MD, FACNS
Objectives:
At the conclusion of this session, participants should be able to:
1. Review new insights in spinal cord arterial supply;
2. Discuss some of the limitations of TcMEPs;
3. Review practical dilemmas encountered when performing NIOM.

Agenda:
4:00PM  Introduction  
Jaime R. López, MD
4:05PM  A Different View of the Spinal Cord Arterial Supply  
Leo T. Happel, PhD
4:30PM  The Problem with TcMEPs-cross-activation  
Andras A. Gonzalez, MD
4:55PM  Continuous Neuromonitoring: Fact or Fiction?  
Leslie H. Lee, MD
5:20PM  Discussion

Imaging and Electrophysiology in Neuromuscular Diseases: A Case-Based Approach
Location: Grand Salon 2
Chair: Suraj Muley, MD
Objectives:
At the conclusion of this session, participants should be able to:
1. Identify different patterns of conduction slowing and their significance;
2. Describe the use of ultrasound in neuromuscular disorders;
3. Identify how to diagnose motor neuron disease.

Agenda:
4:00PM  The Many Faces of Conduction Slowing  
Suraj Muley, MD
4:20PM  Extending the Reach of Clinical Neurophysiology - Seeing What You Feel with Ultrasound  
Francis O. Walker, MD, FACNS
4:40PM  Approach to Motor Neuron Disease  
Mark Ross, MD, FACNS
5:00PM  Ultrasound in Neuropathy  
Erik Ortega, MD
5:20PM  Discussion

ICU EEG SIG: Applying the ACNS ICU EEG Consensus Statement to Clinical Practice
Location: Grand Salon 3
Co-Chairs: Nicholas Abend, MD, FACNS and Elizabeth Gerard, MD, FACNS
Objectives:
At the conclusion of this session, participants should be able to:
1. Review the key recommendations of the ACNS consensus statement of continuous EEG monitoring;
2. Identify challenges in implementing these recommendations in various clinical settings;
3. Discuss strategies to phase in guidelines in various practices and to overcome common barriers to consistent monitoring practices.

Agenda:
4:00PM  Introduction to the ACNS ICU EEG Consensus Statement  
Susan T. Herman, MD, FACNS
4:10PM  Adult – Implementation in an Existing University Practice  
Jennifer Hopp, MD
4:20PM  Adult – Implementation in a Community Hospital  
Suzette M. LaRoche, MD, FACNS
4:30PM  Adult – Implementation at a Remote Monitoring Site  
Stephen Hantus, MD, FACNS
4:40PM  Neonatal – Implementation in a Neonatal ICU  
Tammy Tsuchida, MD, PhD, FACNS
4:50PM  Pediatric – Implementation in a Pediatric ICU  
Nicholas S. Abend, MD, FACNS
5:00PM  Discussion
**Invasive EEG: Nonlesional Temporal Lobe Epilepsy**
Location: Grand Salon 4 & 5  
Chair: Gregory Worrell, MD and Stephan Schuele, MD, MPH, FACNS  

Objectives:
At the conclusion of this session, participants should be able to:
1. Discuss algorithms to select patients with nonlesional TLE for direct resection, with and without the use of electrocorticography;
2. Explain various invasive strategies to evaluate patients with nonlesional TLE;
3. Discuss pros and cons of surgical approaches other than a standard anterior temporal resection for patients with nonlesional TLE.

Agenda:
4:00PM  Introduction
4:05PM  Structural Imaging Negative, Functional Imaging Positive TLE
Elson Lee L. So, MD, FACNS
4:15PM  Discussion
4:20PM  Intraoperative ECOG: What Does it Add?
Andres M. Kanner, MD, FANA
4:30PM  Discussion
4:35PM  Extraoperative Grids: When and Where?
Stephan Schuele, MD, MPH, FACNS
4:45PM  Discussion
4:50PM  SEEG: Temporal and Temporal Plus
Samden Lhatoo, MD
5:00PM  Discussion
5:05PM  And Now, What Would You Do? Four Brief Vignettes
Jay Gavvala, MD
5:15PM  Discussion

**Future Methods of Payment Structures**
Location: Narcissus/Orange Blossom, 2nd Floor  
Co-Chairs: Deborah Briggs, MD, FACNS and Yafa Minazad, MD, FACNS  

Objectives:
1. Understand current compensation models for neurophysiologists;
2. Understand requirements for alternative payment models;
3. Identify possible quality metrics to include in future payment models.

Agenda
The SIG will feature a panel discussion on various aspects of compensation in neurophysiology as well as a brainstorming session with the group and sharing of ideas about challenges in reimbursement and how ACNS could collect the data members need to optimize their individual compensation.

Panel:
Deborah Briggs, MD, FACNS
Yafa Minazad, MD, FACNS
Pradeep Modur, MD, MS
L. Elizabeth Mullikin, FACHE
Marc R. Nuwer, MD, PhD, FACNS

**NEUROPHYS BOWL**
5:45 – 7:00PM  
Location: Grand Salon 4 & 5  
Co-Chairs: Susan T. Herman, MD, FACNS and Sarah Schmitt, MD

**WELCOME RECEPTION**
7:00 – 8:30PM  
Location: International Ballroom
**Montage Matters: Designing EEG Montages for Optimal Localization**  
Location: Grand Salon 4 & 5  
Chair: Jayant Acharya, MD  

Objectives:  
At the conclusion of this session, participants should be able to:  
1. Describe the general guidelines for creating standard montages, and the principles of localization using different types of montages;  
2. Discuss the use of additional electrodes and special montages for optimal localization during noninvasive and invasive video-EEG monitoring;  
3. Describe the clinical utility of specific montages in individual epileptic syndromes.  

Agenda:  
8:00AM Overview: Standard Montages and Principles of Localization  
Jayant Acharya, MD  
8:20AM Montages for Noninvasive Presurgical Video-EEG Monitoring  
Giridhar Kalamangalam, MD, DPhil, FACNS  
8:40AM Montages for Invasive Presurgical Video-EEG Monitoring  
Juan Bulacio, MD  
9:00AM Clinical Utility of Specific Montages for Epilepsy Syndromes  
Ekrem Kutluay, MD  
9:20AM Question & Answer  

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**Sleep and Sudden Death**  
Location: Grand Salon 3  
Chair: Stephan Schuele, MD, MPH, FACNS  

Objectives:  
At the conclusion of this session, participants should be able to:  
1. Discuss how sleep contributes to cardiorespiratory mortality;  
2. Explain the relationship between sleep and epilepsy;  
3. Discuss the sleep related factors which may predispose to SUDEP.  

Agenda:  
8:00AM Sleep and Death  
Hrayr Attarian, MD  
8:25AM Discussion  
8:30AM Sleep and Epilepsy  
Tobias Loddkenkemper, MD, FACNS  
8:55AM Discussion  
9:00AM Sleep and SUDEP  
Brian K. Gehlbach, MD  
9:25AM Discussion
**GENERAL SESSION: TRAVEL AWARD PRESENTATION & JASPER AWARD LECTURE**

**10:00 – 11:00AM**  
**Location:** Grand Salon 4 & 5  
**Co-Chairs:** Devon Rubin, MD and Stephan U. Schuele, MD, MPH, FACNS

**Agenda:**

10:00AM  **Young Investigator Travel Award Recognition**  
Devon Rubin, MD, FACNS and Stephan U. Schuele, MD, MPH, FACNS

10:15AM  **Introduction and Jasper Award Presentation**  
Frank W. Drislane, MD, FACNS

10:15AM  **Jasper Award Lecture: My Memories of Dr. Jasper and My View of the Future of Clinical Neurophysiology as a Discipline**  
Donald L. Schomer, MD, FACNS

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**WALKING BREAK**  
11:00 – 11:15AM

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**CONCURRENT SESSIONS**  
11:15AM – 12:45PM

**History and Future of Clinical Neurophysiology**  
**Location:** Grand Salon 3  
**Chair:** Ioannis Karakis, MD, MSc

**Objectives:**

At the conclusion of this session, participants should be able to:
1. Identify the historical landmarks in the development of CNP and appreciate the trajectory to current practice and future directions.

**Agenda:**

11:15AM  **History and Future of EEG**  
Andrew J. Cole, MD

11:40AM  **Discussion**

11:45AM  **History and Future of EMG**  
Ioannis Karakis, MD, MSc

12:10PM  **Discussion**

12:15PM  **History and Future of IOM**  
Mirela V. Simon, MD, MSc, FACNS

12:40PM  **Discussion**

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**The New Landscape of Neurostimulation a Myriad of Choices**  
**Location:** Grand Salon 1  
**Chair:** Dawn Eliashiv, MD, FACNS

**Objectives:**

At the conclusion of this session, participants should be able to:
1. Identify which patients with medically refractory epilepsy are appropriate for neurostimulation;
2. Identify which specific neurostimulator is most appropriate in a specific patient;
3. Demonstrate basic knowledge of RNS programming;
4. Discuss with patients pipeline neurostimulators as future options.

**Agenda:**

11:15AM  **The Clinical and Neurophysiological Basis of Neurostimulation**  
Dawn Eliashiv, MD, FACNS

11:35AM  **Cranial Neurostimulation: Cumulative Evidence Based Experience and Evolving Options**  
Christopher DeGiorgio, MD

11:55AM  **Responsive Neurostimulation: Choosing Parameters and Clinical Update**  
Lawrence J. Hirsch, MD, FACNS

12:15PM  **Deep Brain Stimulation**  
Dawn Eliashiv, MD, FACNS

12:35PM  **Discussion**

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**New Insights in the Mechanisms of Sudden Unexpected Death in Epilepsy**  
**Location:** Grand Salon 4 & 5  
**Chair:** James Tao, MD, PhD, FACNS

**Objectives:**

At the conclusion of this session, participants should be able to:
1. Describe the incidence and major risk factors of SUDEP;
2. Identify the potential roles of periictal cardiac arrhythmia, central apnea, neurogenic pulmonary edema, and impaired arousal in the pathogenesis of SUDEP;
3. Discuss measures pertinent to SUDEP prevention;
4. Recognize the current limitations and future directions of SUDEP research by better understanding the varied potential mechanisms of SUDEP.

**Agenda:**

11:15AM  **Spreading Depolarization in the Brainstem and SUDEP**  
Brian K. Gehlbach, MD

11:30AM  **Cardiac and Autonomic Mechanisms Contributing to SUDEP**  
Adriana Bermeo-Ovalle, MD

11:45AM  **Electrical Cerebral Shutdown and Central Apnea in the Mechanism of SUDEP**  
Samden Lhatoo, MD

12:00PM  **Neurogenic Pulmonary Edema and Hypoxemia in the Mechanism of SUDEP**  
Jeffrey Kennedy, MD

12:15PM  **Postictal EEG suppression and impaired arousal in the mechanisms of SUDEP**  
James Tao, MD, PhD, FACNS

12:30PM  **Discussion**
Can Clinical Neurophysiology Make Sports Healthier (and Better)?
Location: Grand Salon 2
Chair: Claus Reinsberger, MD, PhD
Objectives:
At the conclusion of this session, participants should be able to:
1. Explain the role of neurophysiology in the pathophysiology, diagnosis and treatment of concussion;
2. Describe the use of clinical neurophysiology in the assessment of overuse / dystonia;
3. Describe the multimodal utility of clinical neurophysiology in sports using the example of soccer and learn how it can contribute to performance optimization and detection of overtraining syndrome.
Agenda:
11:15AM Neurophysiology of Concussion
   Jeffrey Kutcher, MD
11:40AM Neurophysiological Assessment of Overuse/Dystonia
   Mark Hallett, MD, FACNS
12:05PM Neurophysiology of Soccer: Performance Optimization, Overtraining and More
   Claus Reinsberger, MD, PhD
12:30PM Diagnostic and Prognostic Neurophysiologic Evaluation of Nerve Injuries in Sports
   Claus Reinsberger, MD, PhD

LUNCH — Visit Exhibits & Posters
12:45 — 2:00PM
Location: International Ballroom

CONCURRENT SESSIONS
2:00 — 3:30PM
Remote EEG Monitoring: Building a Network
Location: Grand Salon 1
Chair: Amy Crepeau, MD
Objectives:
At the conclusion of this session, participants should be able to:
1. Outline the need for remote EEG reading;
2. Discuss the approach to developing contracts;
3. Review the technical considerations for a remote EEG network;
4. Discuss the communication with local providers, and limitations of the service.
Agenda:
2:00PM Increasing Demand for remote EEG Services: How We Got Here
   Amy Crepeau, MD
2:20PM Contract Considerations for Remote EEG Services
   Matthew Hoerth, MD
2:40PM Making it Happen: Technicians and IT
   Susan D. Agostini, R. EEG T, R. EP T, CLTM, FASET
3:00PM Communications and Challenges in Remote EEG Interpretation
   Stephen Hantus, MD, FACNS
3:20PM Discussion

Toxic and Drug Induced Peripheral Neuropathies
Location: Grand Salon 2
Chair: Alessia Nicotra, MD PhD
Objectives:
At the conclusion of this session, participants should be able to:
1. Describe the neurotoxic mechanisms of drug-induced PNs;
2. Describe drug induced PN objective measures, including physical examination and neurophysiological testing, and subjective measures including grading scales and patient-reported outcome measures;
3. Achieve a better understanding of drug induced PNs pathophysiology, evaluation, and management in order to improve patient’s care.
Agenda:
2:00PM Pathophysiology of Peripheral Neuropathies
   John England, MD
2:30PM Evaluation and Management of Drug-Induced Peripheral Neuropathies
   Tihomir V. Ilić, MD, PhD
3:00PM Drug-induced Peripheral Neuropathy and Other Adverse Effects: The Thalidomide Disaster
   Alessia Nicotra, MD, PhD

REM Sleep Behavior Disorder and REM Sleep without Atonia: Diagnosis and Treatment
Location: Grand Salon 3
Chair: Erik St. Louis, MD
Objectives:
At the conclusion of this session, participants should be able to:
1. Recognize the clinical importance of RBD and its strong association of RBD with neurodegenerative disorders;
2. Understand quantitative EMG REM sleep without atonia (RSWA) analysis methods, and the possible role for RSWA in discriminating RBD subtypes;
3. Choose efficacious and tolerable RBD treatments.
Agenda:
2:00PM REM Sleep Behavior Disorder: Clinical Presentation, Neurological Features, and Treatment
   Michael Howell, MD
2:45PM REM Sleep Without Atonia: Scoring Methods, Associations, and Clinical Implications
   Erik K. St. Louis, MD
Clinical Neurophysiology Trials and Tribulations in Neurocritical Care
Location: Grand Salon 4 & 5
Chair: Jong Woo Lee, MD, PhD, FACNS
Objectives:
At the conclusion of this session, participants should be able to:
1. Identify common challenges encountered while designing, implementing, and completing a clinical neurophysiological/pharmacological trial in the neurological ICU;
2. List adult and pediatric clinical neurophysiology trials being planned or currently underway;
3. Describe optimal clinical trial designs for addressing important clinical questions in neurocritical care.

Agenda:
2:00PM  Introduction
   Jong Woo Lee, MD, PhD, FACNS
2:05PM  Lessons from TRENdS
   Aatif M. Husain, MD, FACNS
2:20PM  Finishing NEMO
   Ronit M. Pressler, MD, PhD
2:40PM  Neurosteroids in Status Epilepticus
   Henrikas Vaitkevicius, MD
3:00PM  Notes from TELSTAR
   Michel J. van Putten, MD MSc PhD
3:20PM  Discussion

WALKING BREAK
3:30 – 3:45PM

SPECIAL INTEREST GROUPS
3:45 – 5:15PM
Novel Non Invasive Methods of Cortical Mapping
Location: Grand Salon 4 & 5
Chair: Daniel Orta, MD
Objectives:
At the conclusion of this session, participants should be able to:
1. Describe the novel non invasive neurophysiological tools to mapping the brain function;
2. Identify state of art of nanotechnology and single unit recordings;
3. Describe uses and limitations of clinical cortical mapping.

Agenda:
3:45PM  Use and Limitations of Cortical Mapping
   Mirela V. Simon, MD, MSc, FACNS
4:15PM  Novel Non-invasive Methods of Cortical Mapping
   Goded Shahaf, MD
4:45PM  Extraoperative and Intraoperative ECoG Based Functional Mapping
   Gerwin Schalk, PhD

Spanish Symposium: El Electroencephalograma, una Gran Herramienta (The EEG, A Great Tool)
*This session will be presented in Spanish.
Location: Grand Salon 3
Chair: Juan Ochoa, MD
Objectives:
At the conclusion of this session, participants should be able to:
1. Identify abnormal EEG patterns and understand the significance;
2. Recognize clinical indications and limitations of EEG ICU monitoring;
3. Identify epileptiform abnormalities on EEG and clinical correlation;
4. Recognize the uses of EEG as a tool to find the source of the epileptic activity.

Agenda:
3:45PM  Uso de conceptos basicos de EEG en la practica clinica
   Luis C. Mayor, MD
4:05PM  Uso de monitoreo de EEG en cuidado intensivo
   Adriana Bermeo-Ovalle, MD
4:25PM  EEG como herramienta para localizar el foco epileptico
   Juan G. Ochoa, MD
4:45PM  Estereo EEG: La ventaja de ver la actividad epileptica en 3D comparado con el registro de superficie cortical
   Juan Bulacio, MD
5:05PM  Discusión

The Wisdom and Vision from the ACMEGS Inaugural Decade
Location: Grand Salon 2
Chair: Anto Bagic, MD, PhD, FACNS
Objectives:
At the conclusion of this session, participants should be able to:
1. Understand the complex grave circumstances that led to an inevitable self-organization of the USA clinical magnetoencephalographers into the American Clinical MEG Society (ACMEGS) in 2006;
2. Recognize and appreciate the profound positive changes within and surrounding clinical MEG field and practice resulting from the ACMEGS activity during its inaugural decade (2006-2016);
3. Appreciate and understand the direction of the future developments of clinical MEG field and ACMEG’s role in it.

Agenda:
3:45PM  Once Upon A Time: Clinical MEG Before ACMEGS
   Michael Funke, MD, PhD
4:15PM  How We Influenced Clinical MEG Field Over the Inaugural Decade
   Anto Bagic, MD, PhD, FACNS
4:45PM  The Vision of the Future of Clinical MEG and ACMEGS’s Role In It
   Richard C. Burgess, MD, PhD, FACNS
Neonatal and Infantile EEG, Seizure Patterns and Outcomes in the Setting of Congenital Heart Disease: When do we Need an EEG?
Location: Grand Salon 1
Co-Chairs: Tobias Loddenkemper, MD, FACNS and Heather Olson, MD

Objectives:
At the conclusion of this session, participants should be able to:
1. Review findings from EEG monitoring in the peri-operative period in neonates/infants undergoing surgery for congenital heart disease, including frequency and types of seizures;
2. Discuss parameters for EEG monitoring after cardiac surgery for congenital heart disease;
3. Review long term epilepsy and EEG outcome in children who have undergone surgery for congenital heart disease.

Agenda:
3:45PM  Neonatal and Infantile EEG and Seizure Patterns: When Do We Need an EEG?
Rohit Das, MD, FACNS
Mandy Harris, MD

4:20PM  EEG Monitoring in Neonates/infants Undergoing Cardiac Surgery for Congenital Heart Disease
Arnold Sansevere, MD

4:50PM  Discussion
Sunday, February 14, 2016

**BREAKFAST**
7:00 – 8:00AM  
Location: Grand Salon Foyer

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**CONCURRENT SESSIONS**
8:00 – 9:30AM  
**Functional Neurosurgery and IONM - From Spine to Brain**  
Location: Grand Salon 1  
Chair: Jay Shils, PhD, FACNS

**Objectives:**
At the conclusion of this session, participants should be able to:
1. Define mapping methods;
2. Describe the goals of these methods as they relate to planning the next phase of surgery;
3. Discuss the difference between the interpretation of data and the description of the data.

**Agenda:**
- 8:00AM  
  Functional Neurosurgery and IONM  
  Jay L. Shils, PhD, FACNS
- 8:25AM  
  Functional Neurosurgery and IONM  
  Jeff Arle, MD, PhD FAANS
- 8:50AM  
  Functional Neurosurgery and IONM  
  Mark Stecker, MD, PhD, FACNS
- 9:15AM  
  Interactive Real Life Scenarios

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**New Frontiers in Electrocorticography**
Location: Grand Salon 4 & 5  
Chair: Anthony Ritaccio, MD

**Objectives:**
At the conclusion of this session, participants should be able to:
1. Describe the transformative role of the epilepsy patient undergoing electrocorticography as a portal into novel clinical, therapeutic, and basic neurobiological investigations;
2. List essential neuroscientific and technical principles that open up these new research opportunities;
3. Engage in some of these investigations with their own ECoG patients.

**Agenda:**
- 8:00AM  
  Decodification of the Brain by Electrocorticography  
  Anthony L. Ritaccio, MD
- 8:30AM  
  Brain-Computer Interfacing Using Electrocorticographic (ECoG) Activity  
  Aysegul Gunduz, PhD
- 9:00AM  
  Modern Understanding of ECoG-Based Neurophysiology  
  Gerwin Schalk, PhD

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**The Future of EMG in the Current Medical Environment**
Location: Grand Salon 2  
Chair: Morris Fisher, MD, FACNS

**Objectives:**
At the conclusion of this session, participants should be able to:
1. Describe the development of the current practice of EMG within an historical context;
2. Identify current challenges for performing high quality EMG service;
3. Explain how EMG studies are now being approached in different types of practice environments;
4. Develop ideas as to how a career investment in EMG may be most rewarding in the future.

**Agenda:**
- 8:00AM  
  Current EMG: An Historical Viewpoint  
  Morris Fisher, MD, FACNS
- 8:20AM  
  EMG: A Future Perspective  
  Francis O. Walker, MD, FACNS
- 8:40AM  
  EMG: The Present and the Future - A Mayo Clinic Perspective  
  Devon Rubin, MD, FACNS
- 9:00AM  
  EMG and Private Practice: Going the Way of the Dinosaur?  
  John Wilson, MD
- 9:20AM  
  Discussion

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**Spanish Symposium: Estrategias Multimodales en la Planeación de Cirugía de Epilepsia**
(Multimodal Strategies in Surgical Epilepsy Planning)
*This session will be presented in Spanish*
Location: Grand Salon 3  
Chair: Adriana Bermeo-Ovalle, MD

**Objectives:**
At the conclusion of this session, participants should be able to:
1. Understand the principles for the use of semiology and non invasive EEG analysis for the identification of the epileptogenic zone in surgical epilepsy candidates;
2. Understand the principles for the use of non invasive diagnostic techniques such as Ictal Spect and MEG for the identification of the epileptogenic zone in surgical epilepsy candidates;
3. Describe the indications for Invasive EEG as well the advantages and challenges of the different approaches to invasive EEG;
4. Discuss the available data regarding outcomes in epilepsy surgery.

**Agenda:**
- 8:00AM  
  Uso de Semiología y EEG No-invasivo en la Localización del Área Epileptogénica (Use of Semiology and Non-Invasive EEG for the Localization of the Epileptogenic Zone)  
  Luis C. Mayor, MD
- 8:20AM  
  Herramientas No-Invasivas Para la Evaluación Diagnóstica Multimodal en Epilepsia Refractaria: Estudios de Medicina Nuclear y MEG (Non-Invasive Tools for Multimodal Evaluation in Refractory Epilepsy: Nuclear Medicine Studies and MEG)
## PROGRAM AGENDA | ANNUAL MEETING

**Adriana Bermeo-Ovalle, MD**

8:40AM  Como Planear una Evaluación Invasiva de EEG y Como Usar la Información Adquirida (How to Plan an Invasive EEG Evaluation and How to Use the Information Acquired)

Juan Bulacio, MD

9:00AM  Actualización Sobre Resultados en Cirugía de Epilepsia (Update on Outcomes in Epilepsy Surgery)

Andres M. Kanner, MD, FANA

9:20AM  Discussion

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### COFFEE BREAK

9:30 – 10:00PM
Location: Grand Salon Foyer

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### CONCURRENT SESSION

10:00 – 11:30AM

**Functional Brain Mapping: Overview and Future Directions**
Location: Grand Salon 2
Chair: Daniela Minecan, MD

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

1. Describe the underlying neurophysiology of brain function;
2. Demonstrate a grasp of the awake language and motor mapping techniques;
3. Formulate a mapping plan for a specific patient, based on the clinical history (epilepsy case versus lesional case) and region of the brain involved;
4. Describe the challenges and barriers to care associated with treating patients that have epilepsy or a specific lesion involving eloquent cortex.

Agenda:

10:00AM  Functional Brain Mapping Review: Neurophysiology, Methodologies, Cortical versus Subcortical
   Daniela N. Minecan, MD

10:15AM  Functional Brain Mapping: Are Children Different?
   Tobias Loddenkemper, MD, FACNS

10:30AM  Functional Brain Mapping in Stereo EEG
   Patrick Chauvel, MD

10:45AM  Intra-Operative Awake Cortical and Subcortical Mapping in Brain Tumor Cases
   Matthew Tate, MD, PhD

11:00AM  Advances in Neuroengineering: What does the Future Hold for Functional Brain Mapping?
   Gregory Worrell, MD

11:15AM  Discussion

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**Clinical Neurophysiology of Insular Cortex Epilepsy**
Location: Grand Salon 1
Chair: Dang Nguyen, MD, PhD, FRCPC

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

1. Describe insular functions with respect to its functional anatomy and its structural connectivity;
2. Explain the clinical manifestations of epileptic seizures and their electrophysiological correlates, both invasive and non-invasive;
3. Recognize the contribution of novel non-invasive imaging techniques for understanding and diagnosing insular cortex epilepsy.

Agenda:

10:00AM  The Structure and Functions of the Insula
   Olivier Boucher, PhD

10:20AM  Video-scalp EEG Investigation of Insular Cortex Epilepsy
   Dang K. Nguyen, MD, PhD, FRCPC

10:40AM  Value of MEG and EEG-fMRI in the Investigation of Insular Cortex Epilepsy
   Younes Zerouali, PhD

11:00AM  Invasive EEG Investigations of Insular Interictal and Ictal Abnormalities
   Philippe Ryvlin, MD, PhD

11:20AM  Discussion

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**What Can We Learn From the Postictal State?**
Location: Grand Salon 3
Chair: Meriem Bensalem-Owen, MD, FACNS

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

1. Discuss potential endogenous mechanisms involved in the postictal period and seizure termination;
2. Recognize postictal EEG and clinical manifestations;
3. Identify postictal autonomic manifestations.

Agenda:

10:00AM  Endogenous Mechanisms Resulting in the Postictal Period
   Steven Schachter, MD, FACNS

10:20AM  EEG Aspects of the Postictal State
   Ambica M. Tumkur, MD

10:40AM  Clinical Manifestations of the Postictal State
   Meriem Bensalem-Owen, MD, FACNS

11:00AM  Postictal Autonomic Manifestations and SUDEP
   Jeffrey Britton, MD, FACNS

11:20AM  Discussion

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**ADJOURN**
11:30AM
POSTER ABSTRACTS

Friday, February 12, 2016

Display Time: 10:00AM – 4:00PM
Poster Tours: 12:45 – 2:00PM
Location: International Ballroom, Lobby Level

Categories: F1 Autonomic Function and its Disorders
F2 – F17 Critical Care Monitoring
F18 – F19 Deep Brain and Cortical Stimulation
F20 – F29 EEG
F30 – F39 Epilepsy: Clinical
F40 – F50 Intraoperative Monitoring
F51 – F54 Peripheral Neuropathy/Neuromuscular
F55 – F56 Sensory/Motor Physiology
F56 – F60 Video-EEG Monitoring for Epilepsy

F1
Autonomic Failure from Radiation Induced Myelopathy: A Case Report
Priya Dhawan, MD; Brent Goodman, MD

Introduction: Clinical, radiographic, and autonomic nervous system test findings are described in a case of progressive autonomic failure resulting from a radiation-induced cervico-thoracic myelopathy.

Methods: Case: A 22-year-old female was referred to our institution for evaluation of progressive autonomic failure. She was diagnosed 3 years prior with thoracic region Hodgkin lymphoma; treated initially with chemotherapy, followed by autologous stem cell transplantation, and ultimately external beam radiation to the neck and chest. Several months later she developed ascending lower limb numbness and weakness, eventually becoming paraparetic.

Results: MRI studies demonstrated extensive, confluent T-2 signal change involving the cervical and thoracic spinal cord, and ultimately she was diagnosed with a radiation-induced myelopathy. Several months later she developed recurrent syncope and near-syncope, and a pacemaker was placed without benefit. Her neurological examination revealed a spastic paraparesis with a 14 sensory level. Autonomic testing revealed intact postganglionic sympathetic sudomotor function, marked cardiovascular impairment, and cardiovascular adrenergic failure with orthostatic hypotension on tilt-table testing. Symptomatic stabilization was achieved following the initiation of fludrocortisone and midodrine.

Conclusion: Myelopathy is an uncommon complication of radiation to the neck and thorax. Progressive autonomic dysfunction, resulting in cardiovascular, genitourinary, and cardiovascular adrenergic failure has not been well characterized in radiation-induced myelopathy. Timely recognition of this condition will facilitate implementation of potentially beneficial symptomatic treatment and avoidance of unnecessary diagnostic tests and treatments.

F2
Neural Correlates of Spatial and Non-spatial Attention Determined using Intracranial Electroencephalographic Signals in Humans
Joong Koo Kang, MD, PhD; Ga Young Park, PhD; Taekyung Kim; Jinsick Park; Sun I. Kim; In Young Kim; Dong Pyo Jang; Masud Husain; Eun Mi Lee

Introduction: Few studies have directly compared the neural correlates of spatial attention (i.e., attention to a particular location) and non-spatial attention (i.e., attention to a feature in the visual scene) using well-controlled tasks. Here, we investigated the neural correlates of spatial and non-spatial attention in humans using intracranial electroencephalography.

Methods: The topography and number of electrodes showing significant event-related desynchronization (ERD) or event-related synchronization (ERS) in different frequency bands were studied in 13 epileptic patients.

Results: Performance was not significantly different between the two conditions. In both conditions, ERD in the low-frequency bands and ERS in the high-frequency bands were present bilaterally in the parietal cortex (prominently on the right hemisphere) and frontal regions. In addition to these common changes, spatial attention involved right-lateralized activity that was maximal in the right superior parietal lobe (SPL), whereas non-spatial attention involved wider brain networks including the bilateral parietal, frontal, and temporal regions, in spite of the activity was maximal in the right parietal lobe. Within the parietal lobe, spatial attention involved ERD or ERS in the right SPL, whereas non-spatial attention involved ERD or ERS in the right inferior parietal lobe.

Conclusion: These findings reveal that common as well as different brain networks are engaged in spatial and non-spatial attention. Acknowledgments: This work was supported by The National Research Foundation of Korea, funded by the Korean Government (grant number 2013R1A2A2A04015925).

F3
Seizure Management Following Neonatal Cardiac Surgery
Shavonne L. Massey, MD; Nicholas Abend, MD, FACCNS; Sudha Kilaru Kessler, MD; Dennis Dlugos, MD; Daniel Licht, MD; J. William Gaynor, MD; Maryam Y. Naim, MD

Introduction: We previously reported an electrographic seizure incidence of 8% in neonates with congenital heart disease undergoing surgery with cardiopulmonary bypass (CPB). We now describe anti-seizure medication (ASM) use in this cohort.

Methods: We conducted a single-center retrospective observational study of a prospectively enrolled cohort of neonates who underwent continuous EEG monitoring after cardiac surgery with CPB from 1/13 – 12/14. Data collected included timing and type of ASM administration and seizure response.

Results: Twenty-one of 22 neonates with electrographic seizures were treated with an ASM. Phenobarbital was administered first-line to 17 neonates (81%) with seizure cessation in 12 neonates (71%). Levetiracetam was administered first-line to 4 neonates (19%) and seizures stopped in 2 neonates (50%). As second-line ASM, 4 received levetiracetam, 2 received phenobarbital, and 1 received phenytoin. Seizures continued after second-line ASM in 4 of 7 neonates (57%). There were no adverse effects with levetiracetam and two adverse effects with phenobarbital (sedation, hypotension).

Conclusion: Phenobarbital and levetiracetam were the most commonly administered first-line ASMs to neonates with seizures following cardiac surgery. A small response differential favored phenobarbital but the adverse event profile favored levetiracetam. These data will aid design of prospective ASM efficacy studies.
F4
Quantitative EEG Detects Subclinical Ischemic Pressure-Responsiveness
Gregory Kapinos, MD, MS; Heustein L. Sy, MD; Maryam Sheikh, DO; Michael Nissenbaum, MD; Willie Walker, REEGT; Sean T. Hwang, MD

Introduction: Continuous quantitative EEG (cqEEG) has been used in critical care of acute brain injury for prognostication in coma, or to detect non-convulsive seizures and early ischemic secondary injury. We report on our broader usage of cqEEG in a neuro-ICU for 1) finer detection of rampant subclinical ischemia, 2) defining subclinical hypoperfusion with positive response to mild hemodynamic augmentation (HDA) i.e. perfusion optimization-responding syndrome (PORS) and 3) adjustment in blood pressure (BP) for penumbral salvage using electrographically defined goal-directed therapy.

Methods: In ten subarachnoid hemorrhage (SAH) patients, cqEEG was used because of discordant alarm level based on other modalities announcing vasospasm. In 3 other patients, 1 with acute ischemic stroke (AIS) and 2 in vasospasm after SAH, we used cqEEG to ascertain the need for HDA for penumbral salvage. In these 3 patients, PORS was confirmed and cqEEG was continued to gauge our induced modulations in blood pressure (BP) tailored to individual patient’s evolving ischemic thresholds.

Results: Relative alpha variability (RAV), alpha-delta ratio (ADR) and alpha power (AP) by quadrant, hemispheric suppression ratio (HSR), all need to be combined to yield finer detection of subclinical ischemia. PORS was identified on EEG by an increase in AP or decrease in HSR, before the subsequent clinical response to HDA. PORS helped us select patients mandating further HDA. Once HDA was initiated, fine-tuning BP goals was based on quadrant AP & ADR, which resulted in earlier and gentler adjustments compared to changes guided by clinical parameters alone.

Conclusion: cqEEG is a valuable tool in the neuro-ICU for AIS and SAH patients. It helps in determining the need for “pressing” a patient and in tailoring and gauging induced hypertension. This modality holds promise for finer and continuous ischemia detection compared to clinical scrutiny.

F5
A Spectrogram-Based Classification System for Seizure Identification: A Pilot Study
Edilberto Amorim, MD; Craig A. Williamson, MD; Lidia Moura; Mouhsin Shafi, MD, PhD; Nicolas Gaspard, MD, PhD; Eric S. Rosenthal, MD; Venkatakrishna Rajajee, MBBS; M. Brandon Westover, MD, PhD

Introduction: Seizure screening employing compressed spectral arrays (CSAs) can facilitate and expedite review of continuous EEG. Despite the expansion of its use in EEG monitoring, no standardized classification system of spectrogram signatures associated with ictal EEG patterns is available.

Methods: Two neurophysiologists and one neuro-intensivist reviewed 40 two-hour CSA segments of continuous EEG. Reviewers scored CSA displays for the presence of seizures (initial seizure score) and concurrently rated the displays using the CSA classification system we have developed. EEG background is scored in three categories: “broadband” if the background has high power (yellow/red on dB color scale) beyond the 5Hz frequency-band, “narrow” (blue/green). Discrete events were scored in three categories: “solid flame” when there were short discrete increments in power and frequency with a flame-like appearance that has smooth borders, “irregular flame” if the flame had irregular borders, “artifact” if the highest power of the spectrogram event had 15-20Hz frequency-band. The displays in which broadband or solid flames were present were categorized as “CSA category-based seizure.”

Two experienced clinical neurophysiologists blinded to the CSA data performed conventional visual analysis of the raw EEG and served as the gold standard.

Results: Seizure screening accuracy was similar between the CSA category-based seizure and the initial seizure scoring by experts (69.2% vs. 63.3%). Seizure detection sensitivity based on CSA category was 68% and 50% with the initial seizure score; specificity was 70.8% and 76.7%, respectively.

Conclusion: In this pilot-study, seizure screening using a CSA-based category system had similar performance to spectrogram scoring by experts. The limited interrater agreement indicates that improvements on the classification definitions and education methodology are needed.

F6
Association of Rhythmic/Periodic EEG Patterns and Mortality
Carlos F. Muniz, MD; Andrea Synowiec, DO; Kevin M. Kelly, MD, PhD

Introduction: ICU outcomes and performance models, such as APACHE and ICOM, often use the Glasgow Coma Scale (GCS) as a marker of acute brain dysfunction and added morbidity. The present study explores the association of abnormal rhythmic and periodic patterns and in-hospital mortality by using EEG as a biomarker of acute brain dysfunction in the ICU.

Methods: A total of 2,084 ICU EEG studies were identified for a two-year period. EEGs were reviewed using the ACNS standard ICU EEG terminology. Demographics, discharge summary, length of stay, disposition, medications, laboratory data and ICD/DRG codes were programmatically extracted. The primary endpoint was in-hospital mortality and secondary endpoints were length of stay and discharge disposition. The two comparison groups were patients that had EEGs with rhythmic delta activity or periodic discharges versus those that did not demonstrate these patterns during their ICU stay.

Results: An abnormal rhythmic or periodic pattern as defined by the standardized ACNS ICU EEG terminology was detected in 150 of 1,061 patient visits (14%). Overall, 239 out of 1,061 patients (23%) died during the hospital admission. In the exposed group, 67/150 (44.7%) experienced in-hospital death versus 172/911 (18.9%) in the unexposed group. The odds of death in the exposed group were 67/83 (0.807) while in the unexposed group they were 172/739 (0.233) for an unadjusted odds ratio of 3.46 (95% CI 2.41-4.98, p<0.000). Logistic regression was performed to control for cardiac arrest status, ICU status and sex. The adjusted OR was 1.86 (95% CI 1.15-2.99, p=0.011).

Conclusion: The presence of abnormal rhythmic or periodic EEG patterns in the ICU seems to be an independent risk factor for in-hospital mortality. The presence of abnormal EEG patterns, as defined by the ACNS ICU EEG terminology, can be considered as a biomarker of acute brain dysfunction and tested in more comprehensive ICU performance and risk prediction models.

F7
Continuous EEG after Traumatic Brain Injury
Peter Wrigley, MD; Erin Silva, CNP; Norberto Andaluz; Brandon Foreman, MD

Introduction: cEEG is an essential tool for patients with critical illness. Seizures (Sz) and periodic discharges (PD) have been described after subarachnoid hemorrhage,
intracerebral hemorrhage (ICH), and sepsis. The last large series of patients with traumatic brain injury (TBI) was prior to widespread adoption of cEEG and standardized descriptive terminology.

Methods: From 5/2014-5/2015 all TBI patients at the University of Cincinnati were screened. Clinical information was gathered retrospectively for patients evaluated by Neurotrauma. We recorded the presence of PD, Sz and other characteristics in those undergoing cEEG monitoring. Descriptive statistics and univariate analysis were carried out using R.

Results: 631/993 patients with TBI were seen by Neurotrauma; 75 patients underwent cEEG. Median age was 57 (IQR 30-69); 56/75 (75%) were male. Median total GCS was 7 on arrival (IQR 3-13). 47/75 (63%) were admitted to the NeuroICU. cEEG was started a median of 1 day (IQR 1-2) from trauma, for a duration of median 3 days (IQR 2-4). 12/75 (16%) had PD/Sz, including 8/75 (11%) with Sz. 2/8 had myoclonus due to pre-hospital cardiac arrest; all other Sz were electrographic. PD/Sz were associated with the presence of ICH (p=0.04). In-hospital mortality was 27/75 (36%). Of those with PD/Sz, 9/12 (75%) died. The presence of PD/Sz was associated with poor outcome (death or discharge to hospice; p=0.01). Although patients with PD/Sz were made comfort care more frequently (50% vs 32%, p=0.05), patients with PD/Sz were also more likely to die as a result of their injuries (33% vs 5%, p=0.04).

Conclusion: In hospitalized TBI patients undergoing cEEG PD/Sz occurred in 16%. Sz were almost exclusively electrographic and would be missed without cEEG. PD/Sz were associated with poor outcome, although this was confounded by end-of-life decisions. Further study is warranted to understand PD/Sz after TBI and their impact on decision-making and outcome.

F8

Impact of an ICU EEG Monitoring Pathway on Management Timing
Ryan P. Williams; Brenda Banwell; Robert A. Berg; Dennis Dlugos, MD; Maureen Donnelly; Rebecca Ichord; Sudha Kilaru Kessler; Jane Lavelle; Shavonne L. Massey; Allison Parker; Alexis Topjian; Nicholas S. Abend, MD, FACNS

Introduction: We aimed to determine whether implementation of a multidisciplinary EEG monitoring pathway could improve the timeliness of anti-seizure medication administration in response to electrographic seizures in encephalopathic critically ill children.

Methods: A multidisciplinary team aimed to decrease the time electrographic seizure onset to anti-seizure medication administration by developing a pathway involving staff education and streamlined communication. Measurements were obtained prior to and after implementation.

Results: We collected data on 41 patients before and 21 after the implementation of a pathway. There were no differences between the groups in demographic characteristics, acute encephalopathy etiologies, or anti-seizure medications utilized. The median duration from seizure onset to anti-seizure medication administration was significantly shorter for patients treated with the pathway (64 minutes [50, 101]) compared to patients treated prior to pathway implementation (139 minutes (71, 189]) (p=0.0006). Seizure termination was more likely to occur following initial anti-seizure medication administration in the pathway than baseline group (57% vs. 24%, p=0.01).

Conclusion: Implementation of the pathway resulted in a significant reduction in the duration between seizure onset and anti-seizure medication administration and in the rate of seizure termination. Further study is needed to determine whether these changes improve neurobehavioral outcomes.

F9

Electrographic Seizures in Children Undergoing ECMO
Jain-Jimm Lin; Dennis Dlugos, MD; Maureen Donnelly; Rebecca Ichord; Todd Kilbaugh; Roxanne E. Kirsch; Matthew Kirschen; Daniel J. Licht; Shavonne L. Massey; Maryam Y. Naim; Natalie E. Rintoul; Alexis Topjian; Nicholas S. Abend, MD, FACNS

Introduction: We aimed to determine the prevalence and risk factors for electrographic seizures in neonates and children undergoing extracorporeal membrane oxygenation (ECMO).

Methods: Since July 2014 patients requiring ECMO also undergo 1-2 days of continuous EEG monitoring. As a quality improvement study, we are evaluating the impact of EEG monitoring on clinical management for consecutive patients from two years. We describe an interim analysis for July-June 2015.

Results: 60 patients underwent ECMO and 52 (87%) underwent continuous EEG monitoring. ECMO was veno-arterial in 44 (85%) and veno-venous in 8 (15%). Electrographic seizures occurred in 12 (23%) including 9 (75%) with electrographic status epilepticus. Seizures were exclusively non-convulsive in 9 (75%). Seizures occurred in 9/18 (50%) in the CICU, 3/16 (16%) in the NICU, and 0/6 (0%) in the PICU. Seizures occurred in 27% with veno-arterial ECMO and none with veno-venous ECMO (p=0.09). Seizures occurred in 9/32 (28%) without and 3/8 (27%) with prior neurologic conditions, respectively (p=0.7). Mortality was 67% and 38% in those with and without electrographic seizures, respectively (0.07).

Conclusion: Electrographic seizures and status epilepticus occur in about one-quarter of neonates and children undergoing ECMO. Additional data will be gathered to assess electrographic seizure risk factors.

F10

Diagnostic Accuracy between Readers for Identifying Electrographic Seizures in Critically-Ill Adult
Bin Tu, MD, PhD; Gordon Bryan Young, MD; Agnieszka Kokoszka; Andres Rodriguez-Ruiz, MD; Jay K. Varma, MD; Linda M. Eerikäinen, MSc; Nadege Assassi, BS; Stephan A. Mayer, MD, FCCM; Jan Claassen; Mikko O. Särkälä, PhD

Introduction: Electrographic seizures in critically-ill patients are often equivocal and uncertain. We sought to determine the degree of agreement and sources of disagreement between expert readers.

Methods: Inter-reader agreement (IRA) measures were derived from 5769 equivocal and 6263 equipoval seizure annotations by five experienced readers after reviewing 74 days of EEGs from 50 patients. Factors including seizure certainty (unequivocal vs. equivocal) and laterality (generalized, partial, bilaterally independent), special seizure patterns (cyclic seizures and status epilepticus), and patient consciousness level (coma vs non-coma) were also investigated for their influence on IRA measures.

Results: On average, 68.43% seizures marked by a reference reader overlapped, at least in part, with those marked by a test reader (any-overlap sensitivity, AO-
**POSTER ABSTRACTS**

Sn). Agreed seizure duration between reader pairs was 58.65% (overlap-integral sensitivity, 0.5Sn), while agreed non-seizure duration was 98.90% (overlap-integral specificity, 0.5Sp). A test reader would annotate one additional seizure not overlapping with a reference reader’s annotations in every 7.9 hours of EEG, i.e., the false positive rate (FPR) was 0.126/hr. Equivocal seizures compromised all IRA measures except 0.5Sp. Sensitivity was higher for patients with status epilepticus or in coma. Specificity was higher for partial than for generalized seizures, lower for cyclic ones.

Conclusion: Agreement between readers for diagnosing electrographic seizures in critically-ill patients is moderate at best. Improved criteria for diagnosing electrographic seizures in the ICU are needed.

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**F11**

**EEG Periodicity Checker II**

Fumisuke Matsuo, MD, FACNS; Michael Ball

Introduction: EEG periodic discharges (PD), occurring in bursts of varying duration, represent steady brain states, often associated with acute encephalopathy. PD are key part of many clinical EEG syndromes, varying in amplitude, space-domain attributes and more importantly, time-domain characteristics (periodic rate, burst duration, and burst recurrence rate), resulting in broad spectrum of epileptic symptoms. Examination of PD captured in prolonged EEG data typically requires expert review, and extraction of relevant EEG features and evaluation of their evolution (progression and regression) are subject to inter-rater variations. It was previously shown that simple digital EEG reformating (polygraphic channel overlay: PGCO) could help appreciate PD in subtle forms, and changing characteristics (J Clin Neurophysiol 2015: 32: 390). Even when supported by fast computing speed, PGCO taxes human operator.

Methods: Computerized automatic detection (CAD) of epileptiform transients (pre-release version of Persyst 13 set at default sensitivity) was applied to assorted clinical examples and compared against expert review of PGCO. Clinical EEG features included PD (6 cases, diffuse and multifocal), frontal intermittent rhythmic delta activity (FIRDA, 2 cases), diffuse spikes (1 case), and wicket spikes (1 case).

Results: This pilot study revealed: 1) PGCO viewer had higher sensitivity for PD detection, and was able to accommodate extreme wide variations of electrographic features. 2) CAD set at steady sensitivity performed better as indicator of PD abundance, reflecting either progression or regression. 3) CAD was indifferent to PD evolution (progression and regression) are subject to inter-rater variations. It was previously shown that simple digital EEG reformating (polygraphic channel overlay: PGCO) could help appreciate PD in subtle forms, and changing characteristics (J Clin Neurophysiol 2015: 32: 390). Even when supported by fast computing speed, PGCO taxes human operator.

Conclusion: In clinical settings of acute encephalopathy, CAD seems to offer distinct utility, because detection sensitivity can be set high and false positivity is of minor concern. CAD can effectively direct reviewer’s attention to changing features of PD for evaluation of clinical relevance.

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**F12**

**Clinico-Electroencephalographic Correlation for Consciousness in the Neuro-ICU.**

Gregory Kopinos, MD, MS; Heustein L. Sy, MD; Maryam Sheikh, DO; Michael Nissenbaum, MD; Willie Walker, REEGT; Sean T. Hwang, MD

Introduction: We report on our broader usage of qEEG in a neuro-ICU for 1) correlation to clinical assessment of level of consciousness (LOC), 2) reconciling clinical variance or clinical-radiologic discordance in terms of LOC ascertainment and 3) fine detection of subclinical hypoperfusion with increase LOC in response to mild hemodynamic augmentation (HDA).

Methods: In four patients, qEEG was used for traditional indications but hourly examinations yielded significant correlation between qEEG data and gradual improvements in arousability and LOC. In three patients, qEEG was used because of discordant clinical assessments of LOC or because of clinical-radiologic discrepancy. No patient was sedated but two patients had qEEG markers of LOC correlate with blood pressure (BP), further confirmed by LOC amelioration both clinically and electrologically with mild HDA.

Results: Background continuity and reactivity correspond to the clinical findings of gaze-attention and tracking. Furthermore, spontaneous variability and global alpha power seem to linearly correlate to LOC as per GCS & FOUR scores. qEEG with alpha power and reactivity can help demonstrate that clinical coma is not unequivocal when radiologic injury burden is disproportionately low. Two patients had exams suggestive of total loss of consciousness but complex cortical activity was easily detectable by reactive EEG. In 2 patients, positive correlation between alpha power and BP during spontaneous fluctuations predicted that subsequent active elevation of BP would eventually lead to easier arousability.

Conclusion: qEEG is valuable in the neuro-ICU for a more precise determination of LOC, as cortical activity can often be masked on clinical exam. It can also reveal an unrecognized pauci-clinical hypoperfusive status. qEEG should not be seen as a potential adjunct to clinical-radiologic monitoring in perplexing cases, but valued as a standard third equipotent modality.

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**F13**

**A Case of Ictal Burst Suppression**

Verena C. Haringer, MD; Pegah Akra, MD

Introduction: “Burst-suppression” (seen in severe cerebral damage, anesthesia, or prematurity) consists of complete attenuation of background between bursts of mixed frequencies.

Case Report: A 46 year-old-female with post-anoxic encephalopathy on cooling protocol was monitored with continuous video-EEG. Two electrographically similar patterns of burst-suppression were identified, one with clinical ictal correlate. On initial video-EEG monitoring the patient was in burst-suppression with 0.5-2 seconds’ bursts of sharp waves in the theta/low-alpha frequency range with a clinical correlate of slow eye opening followed by an eye blink. Therefore we use the term “ictal burst-suppression”. Occasionally an electro-clinical-seizure followed “ictal bursts” in the form of 20 seconds of ictal rhythmic delta intermixed with theta frequencies with a clinical correlate of eye blinking. After dose adjustments of intravenous propofol and midazolam the EEG transitioned to “therapeutic burst-suppression” with bursts of similar duration/frequency but lower amplitude and resolution of clinical correlates.

Conclusion: A case of “ictal burst-suppression” is presented in an adult with post-anoxic encephalopathy. "Ictal burst-suppression” has been reported in neonatal epilepsy syndromes and adult post-anoxic myoclonic-status epilepticus. Our patient’s ictal eye movements were not myoclonic in nature and to our knowledge non-myoclonic “ictal burst-suppression” has not been reported in adult post-anoxic encephalopathy.
F14
Postanoxic Myoclonus and Generalized Tonic-Clonic Seizures
Yara Mikhail-Derna, MD; Elizabeth Gerard, MD, FACS; Irina Bellinski, RN, MPH; Stephan Schuele, MD, MPH, FACS; Jay Govvala, MD

Introduction: Postanoxic myoclonus status (MS) is frequently thought of as a homogenous entity, associated with a poor outcome. While recent literature has called into question whether postanoxic MS is truly a poor prognostic factor, the prior literature has never described the clinical and electrographic presentations of MS. In this case report, we present 3 cases of postanoxic MS who had clinical semiology of myoclonus that progressed to generalized tonic-clonic seizures (GTCs).

Case Report: We present three patients, ages 80, 57 and 34 who had anoxic injury. All three patients had postanoxic MS that progressed to GTCs. Patients 1 and 2 had GTCs within 2 days of their anoxic/hypoxic event; patient 3 had GTC in the setting of persistent myoclonic status that acutely worsened 3 months after her cardiac arrest. In case 1, the clinical presentation led to withdrawal of life-support by the family. Patients 2 and 3 ultimately improved and were following commands.

Conclusion: We describe three unique cases of MS with progression to GTCs, two of whom had good outcomes. The variations in clinical semiology of MS have not been described before. Further studies characterizing the clinical semiology and distribution of MS and its association with prognosis are necessary for a better understanding of this complex clinical syndrome.

F15
Seizure Recurrence Risk in Neonates Treated with Hypothermia for HIE
Mark Fitzgerald; Shavonne L. Massey; Sudha Kilaru Kessler; John Filbottte; Dennis Dlugos, MD; Nicholas S. Abend, MD, FACS

Introduction: We aimed to characterize the risk of future seizures in neonates with hypoxic ischemic encephalopathy (HIE) who had electrographic seizures during or after therapeutic hypothermia (TH).

Methods: We conducted a retrospective cohort study in prospectively enrolled neonates who underwent TH for HIE between April 2010 and December 2014 using our institution’s neonatal database. All neonates underwent full-array continuous EEG during TH, warming, and 24 hours of normothermia. Survival analysis methods were used to determine risk of seizure recurrence.

Results: Follow-up data was available for 59 of 81 neonates (73%), with a median follow-up duration of 19 months (range 2-60 months). Electrographic seizures were present duration admission in 20 neonates (34%). Electrographic seizures were associated with increased risk of developmental delays in the gross motor skills (OR 2.01, 95%CI 1.07-3.79, p=0.029), fine motor skills (OR 20.46, 95%CI 2.29-182.44, p=0.004), language (OR 3.87, 95%CI 1.2-12.5, p=0.023), and social skills (OR 6.46, 95%CI 1.45-28.77, p=0.014).

Conclusion: Electrographic seizures in the neonatal period were associated with subsequent developmental impairments in preliminary analyses without adjustment for HIE severity.

F16
Seizures and Developmental Outcomes in Neonates with HIE
Mark Fitzgerald; Shavonne L. Massey; Sudha Kilaru Kessler; John Filbottte; Dennis Dlugos, MD; Nicholas S. Abend, MD, FACS

Introduction: We aimed to determine whether electrographic seizures are associated with unfavorable neurodevelopmental outcomes in neonates with hypoxic-ischemic encephalopathy (HIE) managed with therapeutic hypothermia (TH).

Methods: We conducted a retrospective cohort study in prospectively enrolled neonates who underwent TH for HIE between April 2010 and December 2014 using our institution’s neonatal database. All neonates underwent full-array continuous EEG during TH, warming, and 24 hours of normothermia. Outcomes were obtained from neonatal follow-up, neurology, and developmental pediatrics clinic visit notes. The association between electrographic seizures and outcome was evaluated using univariable logistic regression.

Results: Follow-up data was available for 59 of 81 neonates (73%), with a median follow-up duration of 19 months (range 2-60 months). Electrographic seizures were present duration admission in 20 neonates (34%). Electrographic seizures were associated with increased risk of developmental delays in the gross motor skills (OR 2.01, 95%CI 1.07-3.79, p=0.029), fine motor skills (OR 20.46, 95%CI 2.29-182.44, p=0.004), language (OR 3.87, 95%CI 1.2-12.5, p=0.023), and social skills (OR 6.46, 95%CI 1.45-28.77, p=0.014).

Conclusion: Electrographic seizures in the neonatal period were associated with subsequent developmental impairments in preliminary analyses without adjustment for HIE severity.

F17
Burst Suppression Patterns in Epileptics Vs Non-Epileptics
Abuhuziefa Abubakr, MD, FACS; Kareem Gadelmola

Introduction: The presence of burst suppression patterns during the use of anti-epileptic medications has not been well studied, nor is it known whether the pattern can predict seizures. Therefore we attempted to evaluate the existence of burst suppression patterns in epileptic vs. non-epileptic patients.

Methods: We conducted a retrospective review of all EEGs with burst suppression obtained in 2014 of patients in the intensive care unit at the University of Mississippi Medical Center. A total of 20 patients were reviewed. Patients were divided into 2 groups based on a history of epilepsy (non-epileptic vs. epileptic). The epileptic group consisted of 7 patients (5 females and 2 males) with a mean age of 43 years old. The non-epileptic group had 13 patients (7 females and 6 males) with a mean age of 55 years old. Bursts were reviewed on the basis of length of bursts, length of suppression and morphology of the bursts.

Results: In the epileptic group the mean duration of the bursts was 2498 msec and the mean duration of the suppression was 3371 msec. In the non-epileptic group the mean duration of the bursts were 9606 msec and the mean duration of the suppression 8478 msec are approaching significance (at p=.08, given that the data is not normally distributed) As regards to the morphology, it was highly variable in the 2 groups including sharp waves, theta, delta and spikes.

Conclusion: When comparing non-epileptic patients and epileptic patients with burst suppression it was found that there is a significant difference between the 2 groups in the length of the bursts and of the suppression. In the non-epileptic patients with
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burst suppression it was found that the bursts and the suppressions were longer in duration. However, there was no significant difference as the morphology was highly variable in the 2 groups.

F18
Subthreshold Cortical Stimulation Reduces Epileptic Activity in Patients with an Epileptic Focus in Eloquent Cortex
Brian Lundstrom; Greg Worrell, MD; Matthew Stead

Introduction: Epilepsy surgery remains the most effective treatment for medically refractory focal epilepsy. However, patients with an epileptic focus that includes eloquent cortex are often not viable surgical candidates. Here, we report on 13 cases in which chronic subdural electrodes were implanted and used for chronic subthreshold cortical stimulation in an effort to reduce seizure burden.

Methods: Patients with medically refractory seizures who had an epileptic focus including eloquent cortex underwent therapeutic focal subthreshold cortical stimulation (SCS) via a subdural grid. Retrospectively, seizure frequency was assessed before and after SCS. The quantity of epileptic discharges from the most active electrodes was assessed before and during SCS.

Results: Clinically 12/13 patients improved with SCS. Of 9 patients suitable for analysis, in the month prior they reported 133 (SD 194) disabling seizures and 4 (SD 10) in the month after. 8/9 experienced improvement. For 3 patients suitable for analysis, the fraction of the record with epileptic activity (1 sec bins) decreased from [0.87, 0.42, 0.48] to [0.56, 0.20, 0.20] in the most active electrode.

Conclusion: This initial data suggests SCS reduces seizure burden for patients with refractory epilepsy and is a suitable treatment option for patients with foci involving eloquent cortex.

F19
Multi-Modal Targeting in DBS for Depression.
Jonathan A. Norton, PhD; Marla Mickleborough; Eric Lorentz; Ron Borowsky; Ivar Mendez

Introduction: Treatment-resistant depression is a growing chronic health issue in North America. Non-pharmacological approaches to management include deep brain stimulation (DBS). The results have been mixed, as have the targets and approaches to stimulation parameters. In this case study, we used functional Magnetic Resonance Imaging (fMRI) to identify areas of abnormal activity, with scans performed on a 3T Siemens scanner and analyzed using Brain Voyager software.

Case Report: Prior to surgery, we employed an emotional word reading task in fMRI. When depressive words were read by the patient there was a decrease in the BOLD signal in the subgenual anterior prefrontal cortex (sACC). A critical hub in the network of depression and the target of the DBS in this case. Intraoperative microelectrode recordings were used to confirm target location. Recordings were obtained using a Leadpoint system (Medtronic). When the same stroop-like task was applied there was a decrease in firing rates of the cells in the target region. Post-hoc analysis was performed in Matlab (The Mathworks). The decrease in firing rate averaged 30 percent. Intraoperative and post-surgical stimulation produced sustained subjective benefits.

Conclusion: This is the first report of the combined use of fMRI and microelectrode recordings in deep brain stimulation paradigms.

F20
Is There an Optimum Time for Obtaining a Sleep-Deprived EEG in Adults and Children?
Katherine Nickels, MD; Suresh Kotagal, MD

Introduction: Routine wake and sleep electroencephalogram (EEG) is performed in patients with neurologic disorders. As sleep activates epileptiform discharges, it is important to sample sleep during the EEG study, but most EEG labs are operational only during daytime. Partial sleep deprivation is recommended. Sleep recording may not be successful due to individual preferences in the circadian drive. We address whether it might be advantageous to synchronize the sleep EEG with periods of increased physiologic sleepiness through a retrospective review of adult and pediatric EEG studies at our institution.

Methods: The Mayo Clinic EEG report database identified all patients who underwent routine sleep before or after EEG January 1, 2015 to April 1, 2015. EEG reports were reviewed to determine: sleep successful, epileptiform discharges recorded in sleep only, time of EEG recording (0700-1159 AM versus 1200-1800 PM), and age of patient.

Results: 1008 EEG reports were reviewed, 290 pediatric and 718 adults; 55% of EEGs were recorded in AM. Sleep was recorded in 82% and was equally successful in adults and children, regardless of time of recording (82% AM pediatric, 85% PM pediatric, 81% AM adult, and 75% PM adult). In 10% of all patients epileptiform discharges or the full extent of cortical areas involved in epileptiform discharges were present only in sleep.

Conclusion: Sleep recordings were successful in the majority of records, with sleep achieved in the morning equally as well as afternoon. For the purpose of EEG, regardless of time of day, partial sleep deprivation appears to adequately overcome the wakefulness drive mediated by the circadian system. EEG recordings do not need to be synchronized with periods of increased physiologic sleepiness. Furthermore, the sleep recording provided unique information (epileptiform discharges) in 10% of patients, emphasizing the importance of the sleep recording.

F21
An EEG Study of Smartphone Text Messaging
William O. Tabum, DO, FACNS; Benedetto S. DiCiaccio, BS.; Joseph A. Kipta, MD; Kirsten Yelvington; Michael Stein, MD

Introduction: The objective was to report the features of a unique EEG rhythm identified during text messaging.

Methods: 131 patients at 2 centers analyzed a texting rhythm (TR); a reproducible, stimulus-evoked, time-synchronized, generalized frontotemporal 5-6 Hz theta rhythm on EEG produced by active text messaging. Independent prospective and retrospective cohorts of epileptic and non-epileptic seizures were assessed and compared. Demographics, MRI and EEG were compared. Analysis used Pearson’s chi-square and Fisher’s Exact Test. Significance judged at p < 0.05.

Results: 24/98 had a TR at 1 site prospectively and 7/31 at another retrospectively (overall prevalence 31/129; 24.0%). The EEG features were identical independent of location. A TR was highly specific to active texting (p< 0.0001). Cognition, speech/language, motor activation and audio cellular telephone similar evoked rhythm was absent in 98 consecutive texters (p<0.0001). Age, gender, epilepsy type, MRI results, and EEG lateralization (in
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epilepsy) did not bear a relationship to the presence of a TR in either arm of the study (p= NS).

Conclusion: The TR is a novel technology-specific evoked rhythm on EEG synched to active text messaging during smartphone use. It provides objective evidence of altered neurophysiologic function potentially interfering with tasks requiring full attention (e.g. driving).

F22
Focal Characteristics in Childhood Absence Epilepsy
Jena Krueger, MD; Anne Berg; Cynthia V. Stack, MD

Introduction: The 3 Hz spike and wave EEG pattern is diagnostic in Childhood Absence Epilepsy (CAE). However, other electrographic features have been described, including focal and multifocal abnormalities and discharge fragments. We sought to determine the frequency of focal patterns and describe them in a consecutive series of children with CAE evaluated at our institution.

Methods: Patients were identified by a keyword search in the central EEG database at our center from 2007-2011. Each eligible EEG was reviewed for discharges, discharge duration, focal lead and fragments.

Results: 98 EEGs with 1615 bursts and fragments were analyzed. 96 patients had bursts and 69/96 (71.8%) had a focal lead in. 58/96 (60.4%) of the patients had frontal focal lead ins. 57/98 (58.2%) of patients had fragments. Treated records had more fragments (62% vs 37%, p=0.04), medication type and age had no effect. Records without fragments had longer discharges (8.66 seconds vs 5.01 seconds, p=0.001), duration did not vary with treatment (6.8 vs 5.1 sec, p=0.25). Sleep deprivation had no effect on number or duration of bursts (p=0.62, p=0.13).

Conclusion: The EEG in CAE is characterized by minor focal features. These findings are still consistent with a diagnosis of a generalized genetic epilepsy and should not weigh against the diagnosis.

F23
Diagnostic Yield of Ambulatory EEGs in the Elderly
Benjamin Tolchin; Jong Woo Lee, MD, PhD, FACNS; Milena Pavlova; Barbara Dworetzky; Rani Sarkis, MD, MSc

Introduction: The diagnostic yield of ambulatory EEGs has been studied in the general adult population, but not in the elderly. We conducted a retrospective review of elderly patients to determine the diagnostic yield of ambulatory studies in this population, and to identify factors predicting a positive study.

Methods: We reviewed 156 consecutive patients aged 60 or older who underwent ambulatory EEG lasting 24-72 hours at a tertiary referral center and two community hospitals.

Results: Patient age ranged from 59.6 to 94.0, with a mean of 72.8. Of the 156 studies, 107 (69%) yielded positive data (either an EEG abnormality or a typical event). Forty-two (27%) revealed epileptiform abnormalities and 4 (3%) captured an epileptic seizure. Twenty-five (16%) captured a typical non-epileptic event. One hundred and twenty-six studies (81%) were felt by the ordering physician to have contributed to diagnosis or management. Thirty-three (21%) resulted in a change in management. Focal findings on prior routine EEGs predicted epileptiform abnormalities on ambulatory EEG (p-value: 0.002, odds ratio: 4.0). Focal findings on prior MRI did not predict epileptiform abnormalities to a statistically significant extent (p-value 0.4, odds ratio: 1.4). Duration of ambulatory EEG did not correlate with yield of epileptiform abnormalities but did correlate with the capture of typical non-epileptic events (p-value: 0.02).

Conclusion: These findings offer guidance in the use of ambulatory EEGs in the elderly. In particular, focal findings on routine EEGs justify the need for an ambulatory EEG in the setting of diagnostic uncertainty. In addition, longer ambulatory EEGs have a higher yield in capturing patients’ typical non-epileptic events, and should be considered in patients where non-epileptic events are on the differential diagnosis.

F24
Predictors of Mortality In Patients With Generalized Periodic Discharges
Neville Jadeja, MD, MPH; Reza Zaregar, DO, FACNS; Alan D. Legatt, MD, PhD, FACNS

Introduction: Generalized periodic discharges (GPDs) are frequently identified in the EEGs of hospitalized patients but their prognostic significance is unclear. We retrospectively reviewed clinical data in patients with GPDs to elucidate factors that are associated with inpatient mortality.

Methods: We reviewed data from inpatients at three different hospital sites affiliated with our institution in whom GPDs were reported by fellowship-trained electroencephalographers during the years 2010-2012. Cox regression was used to determine statistical associations between in-hospital death and demographics, medical comorbidities, metabolic dysfunction, neurological comorbidities, neuroimaging abnormalities, and antibiotic and antiepileptic drug use.

Results: We identified 114 patients with GPDs. The mean age was 69.9 (± 14.1 SD) years, and 71 (62.3%) were women. There were 56 inpatient deaths (49%). The variables that were significantly associated with in-hospital death in the multivariate analysis are shown in the Table.

<table>
<thead>
<tr>
<th>Predictor Hazard Ratio Confidence Interval P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Neuroimaging Abnormalities 2.51 1.15-5.46 0.020</td>
</tr>
<tr>
<td>Cardiac Arrest 4.67 1.96-11.11 0.001</td>
</tr>
<tr>
<td>Creatinine 1.43 1.05-1.95 0.020</td>
</tr>
<tr>
<td>COPD 6.99 2.25-21.76 0.001</td>
</tr>
<tr>
<td>HIV 12.01 2.44-59.90 0.002</td>
</tr>
<tr>
<td>Thyroid dysfunction 6.08 1.39-26.62 0.016</td>
</tr>
</tbody>
</table>

Conclusion: Neuroimaging abnormalities, cardiac arrest, increased creatinine, COPD, HIV, and thyroid dysfunction are significantly associated with increased in-hospital mortality in patients with GPDs.

F25
The Rate of False Lateralization from Scalp EEG in Epilepsy Patients with Bilaterally Implanted Intracranial EEG
Michael Young; Mona Sazgar, MD; Indranil Sen-Gupta, MD; Travis Losey, MD; Firas Bannout; Jane Hwang, MD; Erika Pietzsch; Sumeet Vadera; Frank Hsu; Jack J. Lin; Lili Mnatsakanyan, MD

Introduction: Intracranial EEG (iEEG) recording is needed to define ictal onset zone (IOZ) when noninvasive studies failed to delineate epileptogenic zone. The proposed locations of intracranial electrodes are based on hypotheses derived from scalp
EEG (scEEG) and neuroimaging. The rate of false lateralization from scEEG in patients with bilaterally implanted iEEG is unknown.

Methods: A retrospective study identified 18 patients with medically refractory focal epilepsy having bilateral stereotactic depth electrodes implanted between 2014-2015. Primary aim was to examine concordance of ictal lateralization between scalp and intracranial EEG. Secondary aim was whether IOZ, determined by iEEG, was co-localized to lesion on MRI.

Results: In 7/18 patients, the scEEG lateralization was concordant with iEEG. Of the 11 patients with discordant lateralization, iEEG localized single IOZ in 10. Of these patients, 5 scEEGs suggested bilateral independent ictal onset and 5 scEEGs demonstrated ictal onset in the hemisphere contralateral to IOZ. In the remaining 1, scEEG suggested one IOZ, but bilateral independent seizures were captured on iEEG. Of 10 patients with single IOZ on iEEG, 7 were seizure free postoperatively, and 2 have >50% seizure reduction. 11/18 patients had MRI lesion. In 7/11 of those MRIs, lesion was co-localized to IOZ in all 5 with mesiotemporal sclerosis (MTS) and 2 with asymmetric atrophy.

Conclusion: In this small study, 11/18 (60%) scEEG were discordant with iEEG: 27% of scEEG falsely lateralized, or incorrectly suggested bilateral independent or single IOZ in 33%. Select patients with potentially bilateral seizures on scEEG should not automatically be excluded from epilepsy surgery evaluation. Implantation of bilateral intracranial electrodes should be considered, especially in absence of a lesion on MRI, or lesions other than MTS. Larger prospective studies are needed to confirm our findings.

F26 Pseudo Petit Mal Variant (Drowsy Bursts Pattern)
Osama Muthaffar, MD, SBPN, CSCP; Roy Sharma, RET, CBET; Miguel Cortez, MD, CSCP

Introduction: Benign EEG variants (bEEGv) are patterns with epileptiform appearance and are clearly distinguished from the ongoing background EEG activity and are of doubtful clinical significance. These bEEGv can be complex superimposition of wave-forms that can be challenging and confuse the reader. There are well described bEEGv however, the pseudo petit mal variant is one of the least studied and described EEG variants in current literature of pediatric electroencephalography.

Methods: We reviewed a random cohort of 650 pediatric EEGs (0 to 18 years) at The Hospital for Sick Children, in Toronto, from April 2013 to September 2015. All EEG recordings were performed with the 10-20 system of electrode placement. The prevalence of the pseudo petit mal variant was compared with the reported literature.

Results: Six (0.92%) out of 650 EEGs fulfilled the criteria for pseudo petit mal variant. Gibbs and Gibbs (1952) found it in (0.1%) in their cohort. Eeg-Olofsson (1971) reported pseudo petit mal variants in (7.9%) of 599 normal children aged 1 to 16 years. Alvarez (1982) reported paroxysmal spike and wave in (23%) of 375 children with febrile convulsions and (0%) of 92 normal children. Jeong et al (2013) reported (2%) of 123 children with febrile seizures had pseudo petit mal variant.

Criteria:
- Occurs only in drowsiness
- Seen only in children
- Paroxysmal bursts 3-4 Hz slow waves
- 100-300 µV in amplitude

-Poorly developed spikes (sometimes notched, rudimentary spike-like or polyspikes) in the positive trough of the slow waves

-Fronto-parietal-central areas distribution

Conclusion: The pseudo petit mal variant is still a rare pediatric bEEG that neurophysiologists should be aware of, to avoid overreading of these EEG benign variants and prevent misdiagnosis and unnecessary pharmacological treatments.

F27 Epileptiform Activity Mimics Electrode Artifacts due to Cranietomy
Pedro Oliveira, MD; Frank J. Williams, MD; Christopher Edwards, MD; Zhora Oganisyan, MD; Piotr Olejniczak, MD, FACNS; Edward C. Mader, MD

Introduction: The accentuation of high-frequency scalp EEG components due to skull defect is a familiar finding (breach rhythm). However, volume conduction through a cranietomy site may give rise to confusing EEG patterns.

Case Report: A 23-year-old man with a right frontotral cranietomy was admitted in convulsive status epilepticus. Levetiracetam, lacosamide, and propofol were administered and he was intubated. EEG monitoring showed rare F4 focal seizures, but the interictal EEG showed F4-restricted slow potentials and high-voltage apiculate waves with an FP2-F4 and F4-C4 “mirror image” appearance indicating an F4 electrode artifact. The “artifact” would not disappear despite repeatedly cleaning the scalp, replacing the F4 electrode, and keeping all electrode impedances between 2 to 5 kohms. Extending the montage by surrounding F4 with extra 10-10 electrodes (AF4, FC4, F2, and F6) proved that what appeared to be an F4 electrode artifact was really an exceptionally focal epileptiform discharge at F4 with a steep voltage drop-off.

Conclusion: Skull resistivity impacts the volume conduction of cortical potentials. Removing the skull (e.g. cranietomy) enhances high-frequency potentials. In extreme situations, epileptiform activity may be greatly accentuated and mimic an electrode artifact. Extending the montage with additional 10-10 electrodes can clarify the issue.

F28 Case of Mowat-Wilson Syndrome: Electroclinical Phenotype and MRI findings
Rosario Maria S. Riel-Romero, MD, FACNS; Jayson D. Rodriguez, MD; Susonne Ursin, MD

Introduction: Mowat-Wilson Syndrome (MWS), formerly Hirschsprung Disease-Mental Retardation Syndrome, is a rare syndrome resulting from the ZEB2 gene mutations which functions in neuronal development. We present this case to increase awareness of MWS and demonstrate new EEG and MRI findings.

Case Report: Mowat-Wilson Syndrome (MWS) is a rare autosomal dominant disorder arising from defects in the ZEB2 gene located in chromosome 2q22 region. Affected individuals have distinct facial features, intellectual disability and additional variable clinical features including Hirschsprung disease, microcephaly and seizures. Seizures in MWS have been described as variable in nature and thought to arise from cerebral malformations. Cordelli et al. have described an electroclinical phenotype of epilepsy in MWS. We present an adolescent male with typical MWS features from deletion of the ZEB2 gene highlighting the electroencephalographic (EEG) findings at seizure onset and on follow-up and serial magnetic resonance images (MRI) showing pachygryria, delayed opercular closure and left hippocampal atrophy. Cortical segmentation and reconstruction demonstrated right frontal
Localization and EEG Patterns of Electrical Status Epilepticus of Sleep in Selected Childhood Epilepsies

Abeer J. Hani, MD; Youssef Comair, MD

Introduction: The aim of this case report is to contrast the EEG patterns and localization of electrical status epilepticus of sleep (ESES) in benign childhood epilepsy with centrotemporal spikes versus symptomatic epilepsy. To achieve that, the charts of 2 patients in a new clinical practice were reviewed. At least 2 hours of sleep EEG recording were reviewed to determine localization of ESES and EEG pattern observed.

Case Report: The first patient was a 10-year-old girl with suspected epilepsy with centrotemporal spikes and normal brain imaging. Her seizures were well-controlled using levetiracetam but she presented with worsening learning difficulties. A 2-hour EEG was done that showed evidence of left temporal electrical status epilepticus of sleep occupying about 90% of the sleep record. EEG pattern consisted of organized periodic monomorphic left temporal spikes at 2 Hz seen throughout the sleep record. The second patient was a 5-year-old girl with symptomatic epilepsy due to left hemispheric encephalomalacia attributed to presumed perinatal stroke. She was fairly controlled using levetiracetam and carbamazepine. A 2-hour then a 24-hour EEG showed evidence of left frontocentral ESES occupying more than 90% of the sleep record. The EEG pattern consisted of poorly organized polymorphic spikes, sharp and spike waves occurring at 1-3 Hz seen throughout the recording. The EEG patterns of ESES seen responded better to the use of nocturnal diazepam in the first patient compared to the second patient.

Conclusion: Focal ESES was seen in both cases of benign childhood epilepsy and symptomatic epilepsy. However, the EEG pattern was more polymorphic, and poorly organized in the symptomatic epilepsy case and responded less favorably to nocturnal diazepam. Whether this EEG finding can be used as a biomarker to predict responsiveness to therapy remains to be investigated further.

MRI Abnormalities in Patients with Psychogenic Non-Epileptic Events

Robert D. Bolen, MD; Elizabeth Koonz; Paul B. Pritchard, MD

Introduction: Psychogenic non-epileptic events (PNEEs) are common, especially in epilepsy monitoring units (EMUs) at academic centers [1]. Studies have shown neuroimaging abnormalities in 27-86.4% of patients with PNEEs [2-3]. Limitations of these studies include; small sample sizes, inclusion of non-significant abnormalities, and unclear criteria for diagnosis of PNEEs. Despite limitations, these studies suggest neuroimaging abnormalities are more common in patients with PNEEs than the normal population, where abnormalities range from 4.8-13.6% [4-5].

Methods: We retrospectively identified all patients discharged from our EMU from July 1, 2010 to June 30, 2012. Brain MRI results were collected for analysis only from patients diagnosed with epileptic seizures (ES) and PNEEs. For the purpose of our study, non-significant findings such as developmental venous anomalies, arachnoid cysts, and chronic microvascular ischemic changes were not included.

Results: We identified 339 patients by the above criteria; 256 of whom had brain MRI imaging for analysis, including 111 patients diagnosed with ESs and 68 patients diagnosed with PNEEs. Significant imaging abnormalities were seen in 57.7% of patients with ESs and 33.8% of patients with PNEEs. Abnormalities were identified in the temporal lobes in 57.8% of ES patients and only 21.7% of PEE patients (p=0.003), while multifocal abnormalities were seen in 47.8% of PNEE patients and only 21.9% of ES patients (p=0.018).

Conclusion: We found significant MRI brain abnormalities in 33.8% of patients with video EEG confirmed PNEEs and identified statistically significant differences in the locations of these abnormalities compared with ES patients. We improved on previous studies by collecting a large sample size, only including significant abnormalities, and confirming diagnosis by video EEG. Our study is unique as it investigated the differences in neuroimaging abnormalities in ES versus PNEEs by location.

Normal MRI and Intracranial EEG-guided Laser Interstitial Thermal Therapy in TLE

Bryan Dredla, MD; John Lucas, MD; Robert Wharen, MD; William O. Tatsum, DO, FACNS

Introduction: Treatment for mTLE when patients are drug-resistant is surgical resection. Neurocognitive sequelae may occur after anterior left temporal lobectomy. Selective amygdalohippocampectomy has resulted in more favorable neurocognitive outcomes when compared to standard anterior temporal lobectomy. MRI-guided interstitial laser thermal ablation (MRg-LITT) uses a super-selective stereotactic amygdalohippocampectomy that has been reported to be devoid of neuropsychological deficits. We describe two patients with drug-resistant left mTLE and a normal high-resolution 3T brain MRI who underwent successful left temporal MRg-LITT with post procedure neurocognitive deficits.

Case Report: A 59-year-old right-handed man and a 39 year old right-handed man both had drug-resistant left mTLE verified by intracranial video-EEG. 3T high-resolution MRI of the brain were normal. FDG-PET scans displayed left temporal hypometabolism in both cases. Each patient underwent MRg-LITT of the left amygdala and hippocampus. Postprocedure neuropsychological evaluation revealed significant interval decline in verbal memory (SD, -1.4) and semantic verbal fluency (SD, -1.3) in patient 1. At two-years post-MRG-LITT he continued with stable complaints of memory difficulties despite being seizure free. Patient 2 underwent MRg-LITT of the left amygdala and hippocampus. Postprocedure neuropsychological testing demonstrated mild decline in verbal memory (SD, -0.6) within the context of improved visual memory (SD, +1.5) and otherwise stable cognition. Following several post-operative seizures he has remained seizure free.

Conclusion: We conclude that MRg-LITT may produce neurocognitive decline in adult patients with drug-resistant MRI- . PET + mTLE. Well-localized intracranial EEG and appropriate case selection may identify some patients who could benefit from MRg-LITT as an initial procedure when the high-resolution MRI of the brain is normal.
**F32**

Asystole During a Seizure; Treatment with Pacemaker

Christopher Hassett, DO; Imran I. Ali, MD, FACNS; Noor Pirzada, MD; Mark Buehler, MD

Introduction: Ictal asystole with partial onset seizures is uncommon. Previous reviews have suggested the location of these types of seizures in the frontal or temporal lobes. These seizures are thought to cause autonomic dysfunction leading to asystole. Such episodes of asystole may be of relevance in patients presenting with ictal falls and may also be a factor in causing SUDEP (Sudden Unexplained Death in Epilepsy Patients). However, there are no clear guidelines for the management of patients with ictal arrhythmias.

Case Report: We report a case of a 63 year old male who presented for video EEG monitoring. He had a history of MVA in 1992 and seizures beginning in the year 2000. His typical seizures were described as generalized tonic-clonic but his wife described atypical episodes of confusion and altered awareness at night, for which he was being evaluated. An MRI brain revealed T2 FLAIR hyperintensity involving the left frontal lobe and anterior insular cortex consistent with gliosis and encephalomalacia from his previous traumatic injury. On the second night of his admission, he had an ictal event which localized by EEG to the left temporal lobe and led to a 25 second period of asystole, after which he returned to normal sinus rhythm. Cardiology evaluated the patient, and based on concern for future asystolic events, inserted a pacemaker.

Conclusion: Our patient had a partial seizure arising from the left temporal lobe associated with asystole. Previous reports of ictal cardiac arrest have shown its association with both frontal and temporal lobe foci. Our patient subsequently underwent pacemaker implantation. Although ictal bradycardia and asystole is uncommon, identification of this phenomena can lead to prophylactic treatment with pacemaker implantation and possibly prevent the occurrence of complications such as SUDEP and ictal falls. However, there is a need to establish guidelines for treatment of patients with ictal arrhythmias.

**F33**

LPDs Fragments during Anesthetic-Induced Burst-Suppression

Frank J. Williams, MD; Saurabh Lalan, MD; Louis A. Cannizzaro, MD; Piotr Olejniczak, MD, FACNS; Edward C. Mader, MD

Introduction: Lateralized periodic discharges (LPDs), formerly known as PLEDs, are resistant to antiepileptic drugs (AEDs). Anesthetic-induced burst-suppression is often equated with elimination of epileptic activity—including LPDs. However, this assumption may not always be correct.

Case Report: A 66-year-old woman with right temporal lobe contusion and recurrent convulsive seizures underwent five days of EEG monitoring. Because of breakthrough seizures, several AEDs were administered at various points in time—all intravenously, except topiramate. Lorazepam 4-mg and levetiracetam 1500-mg load/750-mg q12 were given first. The pre-anesthesia EEG showed continuous right posterior temporal LPDs. With propofol induction of burst-suppression, the LPDs started to fragment and seemingly disappeared at an infusion rate of 60 mcg/kg/min. However, close inspection of the EEG showed persistent burst-embedded LPDs fragments. Phenytoin 1300-mg load/100-mg q8 and lacosamide 200-mg load/100-mg q12 did not alter the EEG. Ketamine 65 mcg/kg/min did not eliminate the LPDs fragments either. The LPDs fragments also survived midazolam 1.2 mcg/kg/min and topiramate 200-mg q12. Withdrawal from anesthesia resulted in reemergence of the continuous LPDs pattern.

Conclusion: LPDs fragments may survive propofol, ketamine, and midazolam anesthesia. The clinical significance of burst-embedded LPDs fragments and their tenacity to anesthetics deserve further investigation.

**F34**

Denovo Onset Mesial Temporal Sclerosis in Adult Patients

Joel Oster, MD

Introduction: BACKGROUND: Mesial temporal sclerosis and hippocampal atrophy are frequently associated with febrile convulsions in childhood or severe, frequent and recurrent seizures of the temporal lobe in the pediatric population. The exact mechanisms and factors, number and types of seizures involved, and the chronicity of this process are among the unknown variables. The observation of such cases developing de novo in the human adult clinical population has been rarely documented. This poster paper identifies 2 cases of adult onset mesial temporal sclerosis and describes the clinical features and MRI findings of this unusual condition.

Case Report: In both an urban and a suburban Neurology/Epilepsy Outpatient and Inpatient clinic and hospital setting we noted the onset of hippocampal atrophy and changes consistent with mesial temporal sclerosis (MTS) on 1.5 T MRI in a 48-year-old adult with adult onset epilepsy. The development of changes consistent with MTS on 1.5 T MRI occurred de novo within a 20 month period of seizure onset. This patient has frequent partial seizures, complex partial seizures, secondarily generalized convulsions, and a clinical history consistent with multiple episodes of probable status epilepticus after suffering an ipsilateral intracranial hemorrhage. We also note neuroimaging and clinical findings of a 27 year old patient who after an ipsilateral neocortical resection, develops a similar entity over several years.

Conclusion: De Novo Mesial Temporal Sclerosis (MTS) occurring in the adult patient population is a unique clinical entity and its occurrence and clinical features as suggested in this report indicate a unique retrospective observation having ramifications for future study.

**F35**

A Family with Jeavons Syndrome without Absences: A Study of Two Patients from Consecutive Generations

Ajay M. Tungatuni, MD; Danilo Vitorovic, MD

Introduction: Jeavons syndrome (JS) is characterized by childhood onset of eyelid myoclonia with or without absences, paroxysmal electroencephalographic (EEG) changes associated with eyelid closure and a photo paroxysmal EEG response. We present cases of a patient and her mother with clinical and electrographic features suggestive of JS. To the best of our knowledge, this is the first report of the two consecutive generations with presentation of JS without absences.

Case Report: A 32 year-old woman with a single generalized convulsion at the age of 11 provoked by sleep deprivation reported sensation of eyelid fluttering since early childhood but never experiencing staring spells. She was treated with divalproex sodium since age 11 and remained seizure free. An attempt to wean her off divalproex sodium was made which increased her subjective sensation of eyelid fluttering but no abnormal eyelid movements, convulsions, nor were changes in awareness observed. Her mother, who is 58 years-old, had eyelid myoclonia all her
F36
FIRES and Moyamoya: Refractory Seizures Status Post EDAS Surgery
Taylor Kaufman, R. EEG T., R.EP T.; Andrew White, MD, PhD

Introduction: FIRES (febrile infection-related epilepsy syndrome) is a condition with severe refractory epilepsy that presents in previously healthy school-aged children after significant febrile illness with concomitant rise in body temperature. Suspected causes include genetic or acquired channelopathies as well as mitochondrial disorders. In FIRES, the electroencephalography shows diffuse slowing and/or multifocal discharges. Seizures are present and resistant to treatment. Moyamoya disease is a separate entity, with progressive stenosis of branches of the internal carotid arteries and subsequent development of collateral circulation.

Case Report: We present here the case of a 6 year old with imaging evidence of right-sided moyamoya disease and a post-illness explosive seizure onset that was most consistent with FIRES. It is unknown how this patient’s moyamoya disease is related to FIRES. The patient underwent surgery to correct profusion insufficiency. The patient continues to suffer from intractable seizures and apparent mild encephalopathy. We describe the history, physical, laboratory, imaging, development and seizure control for a 6 year-old female with previously normal development.

Conclusion: Our patient demonstrated two rare diseases not described together previously in the literature. Although the prognosis of FIRES is typically poor, our patient has continued to develop, but with seizures. The impact of our interventions (surgery, AEDs) is unclear.

F37
A 68 Year Old Man With Temporal Lobe Seizures Associated With CASPR-2 Mediated Encephalitis.
Nada A. Alyousha, MD; Jaysingh Singh, MD; Imran I. Ali, MD, FACNS

Introduction: CASPR-2 mediated limbic encephalitis is rare entity with a classic presentation of progressive cognitive impairment and temporal lobe seizures. It is reported to be common in Asian population, may or may not be associated with an underlying malignancy but does respond to immunotherapy.

Case Report: A 68 year old male of Indian origin who was admitted to the hospital because he had a progressive memory loss for the last three months, with recurrent brief confusional episodes, frequent myoclonic jerks involving the jaw and upper body. On neurologic examination, he was noted to have short term memory impairment with intact language function. He also had bilateral arm rigidity, postural tremor and dysmetria on finger-nose testing. Occasionally upper limb myoclonus was observed without any impairment of consciousness. His EEG monitoring revealed electrographic seizures originating from left temporal lobe and MRI brain revealed non-specific white matter changes. His CSF analysis showed mild elevation in protein at 58 mg/dl but was otherwise normal. CT abdomen and pelvis showed diffuse hilar lymphadenopathy and possible liver metastasis but the biopsy of the lesion in the liver was inconclusive. His serum VGKC and CASPR-2 antibodies were positive. He received a course of IV-immunoglobulin and had marked clinical improvement with reduction in seizures, improved cognition and overall neurological status.

Conclusion: This case study highlights the important clinical and EEG aspects of limbic encephalitis. CASPR2 antibodies testing should be considered early in cases of unexplained seizures and encephalopathy. Early diagnosis may result in more effective treatment and better clinical outcome.

F38
A Case of Progressively Expanding Total Hemimegalencephaly
Jayson D. Rodriguez, MD; Arun A. Kalra, MD; Eduardo Gonzalez-Toledo, MD, PhD

Introduction: Hemimegalencephaly (HME) or enlargement of one cerebrum typically presents with triad of intractable epilepsy, psychomotor delay and hemiparesis. Total HME involving ipsilateral cerebellum and brainstem is the least frequent form. It is uncommon for cerebral dysgenesis like HME to continue to grow in size.

Case Report: A 2 year old male presented with seizures and right fronto temporal. Past history included cerebral palsy, developmental delay, left-hemiparesis, shunted hydrocephalus and seizures controlled on levetiracetam, which was tapered off 7 months prior. Present seizures are focal motor with left-sided Todd’s paralysis. Detailed history revealed unrecognized partial complex seizures. On examination, he had prominent hypotonia and no signs of increased intracranial pressure. EEG revealed multiple right hemisphere epileptogenic foci giving rise to 4 electroencephalographic seizures ranging from 3 to 12 seconds. We reviewed prior MRIs. MRI at 2 days showed prominent right ventriculomegaly compressing the rest of the intracranial structures. MRI at 2 months, after shunting, showed markedly decreased size of the right ventricle. Both hemispheres were about equal size but right cerebellum has prominently enlarged. Current MRI shows that the right cerebellum has grown tremendously squeezing the left cerebrum. The enlarged right cerebellum now shows abnormal folia. The right cerebrum also had a large frontal/parietal pachygyria, occipital polymicrogyria, and prominent cleft between fusiform and parahippocampal gyus. NRS of the right frontal lesion confirmed this to be Taylor-type focal cortical dysplasia. Levetiracetam was restarted with no further clinical seizures. He was referred to Neurosurgery for evaluation and possible hemispherectomy.

Conclusion: We presented a case of total HME with unique constitution of brain malformations and progressively expanding size that is rarely reported in literature.

F39
Could Normalization of the Electroencephalogram (EEG) Predict Developmental Outcome in Early Infantile Epileptic Encephalopathy?
Muhammad S. Zafor, MD; Arun Swaminathan; Zahra M. Haghighat, MD; Robert Baumann, MD; Gulam Khan, MD

Introduction: Ohtahara syndrome is an epileptic syndrome characterized by early onset tonic spasms and a burst suppression pattern on the electroencephalogram (EEG). Outcome is poor with drug resistant seizures and delayed development. We report the case of child whose seizures subsided and EEG normalized after Vigabatrin was initiated however this was not paralleled with developmental improvement.
**F40**

**Pygopagus Conjoined Twins: An Intraoperative Monitoring Schema**

Jennifer McKinney, MD; Judy Brown, BA; R. EEG T.; Christina Henry, BA; R. EEG T.; Barrett Cromeens; Monica Islam, MD

**Introduction:** The use of intraoperative neurophysiologic monitoring in the separation of pygopagus (rump-to-rump) conjoined twins has been described in the literature but the information on its utility is limited.

**Case Report:** We present a case of pygopagus conjoined twins who underwent separation surgery that included untethering of spinal cords and sectioning of an imperforate anus. Prior to surgery, the twins demonstrated normal motor and sensory function in their lower extremities. The neurophysiology team worked with a multidisciplinary surgical team to determine a monitoring protocol. Our goal was to provide each child with preserved sacral nerve root capacity and functional anal sphincter musculature. Free run and stimulated electromyography (EMG) of each child’s bilateral medial gastrocnemius, abductor hallucis brevis, and extensor digitorum brevis muscles were recorded. Additional needle electrodes were placed circumferentially to record from 6 regions of the external anal sphincter (EAS). During spinal cord untethering, EMG was used to delineate non-neural versus neural tissues as well as identify neural structures uniquely belonging to each twin. Consistent grouped firing between specific EAS and limb channels guided the division of the anal sphincter. Postoperatively, their lower limb function remains intact.

**Conclusion:** In summary, we present a unique approach to neurophysiologic monitoring that significantly impacted surgical decision-making and hopefully affects neurologic outcome and ultimate fecal continence.

**F41**

**Effect on UPDRS with Intra-Op Testing in DBS Surgery**

Anh Thu Tran, MD; Parasit Shihan, DO; Andres A. Gonzalez, MD, MMM, FACNS

**Introduction:** The Unified Parkinson’s Disease Rating Scale (UPDRS) is used to clinically assess patients with Parkinson’s Disease (PD). Deep brain stimulation (DBS) has been shown to be efficacious in the treatment of PD. Intraoperative testing using combined awake microelectrode recording (MER) and macrostimulation with neurologic exam assists proper localization of DBS targets as well as clinical assessment of DBS. UPDRS scores performed before and after DBS surgery can provide information on efficacy of DBS surgery.

**Methods:** A retrospective chart review of DBS surgeries after 1/10/2014 was performed. Data collected included patient demographics and intraoperative parameters such as repositioning based on MER and/macrostimulation. UPDRS was obtained in the “on” state; before DBS and again during initial DBS programming. First group included patients for whom DBS was repositioned based on MER and/macrostimulation. Second group included patients who did not require repositioning based on adequate MER and macrostimulation. We compared the difference of UPDRS before and after DBS surgery for both groups.

**Results:** In total, there were 27 PD patients who received DBS. 48% (13/27) required repositioning. 52% (14/27) did not require repositioning. In the repositioned group, the mean improvement of UPDRS was 5.92. In the non-repositioned group, the mean improvement of UPDRS was 5.39. There was no statistically significant difference between the mean of both groups.

**Conclusion:** Intraoperative testing plays a valuable role in lead placement and clinical assessment of patients during DBS surgery as shown in this study. Nearly half of patients required repositioning of DBS leads, either because MER was unable to confirm imaging-predicted targets or due to side effects during macrostimulation. Lack of statistically significant difference between the groups demonstrates that they have similarly good outcomes.
F43
The Use of Multipulse Stimulation for Somatosensory Evoked Potentials (SSEP) as an Alternative Method in Poorly Reproducing Signals
Herniberto Guillen; Benjamin Nye, DC; Melissa Ross, CNIM; Indranil Sen-Gupta, MD; Lilit Mnatsakanyan, MD

Introduction: Despite growing research and experience, acquisition of SSEP may still be challenging in the operating room. The operating room environment, anesthetics and the inherent condition of the patient affect the recordings. There is not much knowledge about alternative methods to successfully record adequate SSEP in preexisting nerve injury. A single pulse stimulation is the traditional accepted method for posterior tibial nerve (PTN) SSEP. We describe the use of multipulse stimulation as an alternative method to acquire SSEP signals based on the principle of temporal summation.

Methods: Patients undergoing spine surgery with unreliable pre-incision PTN SSEPs were included in the study. The SSEP were stimulated posterior to the medial malleolus and recorded on the scalp according to ACNS standards. After inability to baseline PTN SSEP by standard recording using a single pulse, maximum suitable stimulation intensity and repetition rate, we changed the stimulation parameter to a multipulse method with a subsequent train of 3, 5, 7 and 9 pulses until reproducible cortical signals (P37) were obtained. The multipulse frequency was set at 2000 Hz, a interpulse latency of 0.5 milliseconds was used.

Results: 12 patients with poorly reproducible posterior tibial nerve SSEP were included in the study. We recorded reliable SSEPs for baseline and throughout the entire surgery in 9/12 after using a train of 5 for multipulse stimulation. In 1 patient a reliable posterior tibial SSEP was recorded after using a train of 7 and 2/12 failed to reproduce reliable SSEP after using a train of 9.

Conclusion: Multipulse stimulation is an alternative option to record reliable PTN SSEPs in patients with poorly reproducible SSEP signals using the traditional singular pulse method. Larger studies are needed to confirm these findings and utility of multipulse stimulation in intraoperative SSEP monitoring.

F44
Role of SSEP and MEP During Clip Ligation of MCA Aneurysm
Anh Thu Tran, MD; Leila Darki, MD; Parastou Shilian, DO; Andres A. Gonzalez, MD, MMM, FACNS

Introduction: The goal of aneurysm ligation is to clip the aneurysmal neck while preventing surgery related ischemia. Neurologic intraoperative monitoring (NIOM) with SSEP and MEP is utilized for early detection of intracranial hypoperfusion. Routine use of NIOM is limited by availability especially after hours. Urgency of surgery may preclude the use of NIOM in ruptured aneurysms, where earlier treatment is preferred. We explore a situation in which NIOM played an integral role in early detection of surgery related hypoperfusion.

Case Report: 57 yo M with ruptured R-MCA aneurysm presented to OR for clip ligation of a 2.5mm right M2 aneurysm. During surgery, there were diminished MEP and SSEP corresponding to placement of temporary clips, which recovered with removal of clips. When a permanent clip was placed, there was complete flattening of MEP and SSEP. The surgeon was alerted of all changes in real time allowing for removal of clips. When a permanent clip was placed, there was complete flattening of MEP and SSEP corresponding to placement of temporary clips, which recovered with removal of clips. The surgeon was alerted of all changes in real time allowing for removal of clips.

Conclusion: This case illustrated the usefulness of NIOM in early detection of hypoperfusion. Although argument can be made for indocyanine green video angiography (ICG-VA) to evaluate patency of vessels after clip ligation, the technique has several limitations. Unlike, NIOM, which can detect hypoperfusion nearly instantaneously, ICG is injected after placement of the clip which may delay detection of an improperly placed clip by several minutes. More importantly, ICG-VA may not be able to adequately visualize all perforating arteries. We propose that every effort should be made to involve NIOM in aneurysm surgery. The decision to delay surgery until NIOM becomes available should be made on an individual case basis weighing risks and benefits of such a delay.

F45
Neurophysiologic Intraoperative Monitoring prompts Evacuation of Previously Asymptomatic Spinal Hematoma
E. M. Hoffman, DO, PhD; Jeremy L. Fogelson, MD; Jamie J. Van Gompel, MD; Jeffrey A. Strommen, MD

Introduction: Traumatic spinal epidural hematomas do not invariably require clot evacuation, especially when asymptomatic. Surgical correction of spine malalignment may cause a hematoma to compress the spinal cord. If identified by neurophysiologic intraoperative monitoring (NIOM), the clot could be evacuated.

Case Report: A 79-year-old female was in a motor vehicle collision and suffered cervical spine fracture with anterolisthesis of C5 on C6 and C6 on C7. A circumferential spinal epidural hematoma was also noted, but she was neurologically intact (asymptomatic). She underwent instrumented fusion and realignment from C3 to T2 with no intention to remove the clot. Immediately after rod placement, bilateral lower limb motor evoked potentials (MEP’s) and lower cervical MEP’s abruptly decreased in amplitude while somatosensory evoked potentials remained stable. Rods were removed, a C4-T1 laminectomy was performed, and the epidural clot was removed. MEP’s recovered and remained stable even after rods were replaced with the same degree of pre-laminectomy alignment correction. The patient woke from surgery neurologically intact and remained so at hospital discharge and at three month follow up.

Conclusion: A traumatic spinal epidural hematoma caused asymptomatic central canal stenosis which, after surgically correcting spinal alignment, caused spinal cord compression. NIOM detected this impeding neural injury, prompting evacuation of a hematoma that would have otherwise been left to naturally resorb.

F46
The Methohexital Challenge: Delayed Loss of SSEPs and tcMEPs
Orhan Bican, MD; Viet Nguyen, MD; S Charles Cho; Leslie H. Lee, MD, FACNS, FAAN; Scheherazade Le; Jaime R. Lopez, MD, FACNS

Introduction: Methohexital is an ultrashort acting barbiturate used for provocative testing in the functional evaluation of brain tissue during embolization of arteriovenous malformations. Its rapid onset of action allows transient functional symptoms within one minute of administration in the awake patient. However in the anesthetized patient, its dosing and the onset of critical changes in the neurophysiologic studies are not well defined. We describe a case that demonstrated changes in the SSEP beyond the conventional 5 minute analysis timeframe.

Case Report: A 24 year old man with right subcortical arteriovenous malformation measuring 8.7 x 2.8 x 7.2 cm with ACA, MCA and PCA arterial supply, and
superficial and deep venous drainage underwent four successful staged endovascular embolization procedures and presented for his fifth session. Provocative testing was performed with 5 mg intraarterial methohexital without any other anesthetic or BP changes. EEG burst suppression was recognized within 5 seconds of infusion. SSEPs remained within normal baseline range within the first five minutes of infusion; but, had a significant decline at 20 minutes postinfusion. A corresponding diffuse loss of tcmEPs, persisted for 25 minutes postinfusion. Patient did not have new postoperative deficits.

Conclusion: This case demonstrates sequential changes in a positive methohexital provocative study, not appreciated within the conventional 5 minute analysis time. The authors suspect a true isolated methohexital effect due to its self-resolution within 25 minutes. This suggests increased but delayed methohexital effect on SSEPs and tcmEPs during provocative testing, requiring a longer testing period for accurate assessment. This also suggest that methohexital effects can be more generalized and longer lasting than expected for superselective testing, and that a single dose (5mg or 10mg) may have more generalized cerebral effects and not fulfill its intended role.

F47
Fentanyl-Induced Suppression of Transcranial Motor Evoked Potentials (tcmEPs)
Orhan Bican, MD; Jaime R. Lopez, MD, FACSNS; S Charles Cho; Viet Nguyen, MD; Scheherazade Le; Leslie H. Lee, MD, FACSNS, FAAN

Introduction: Volatile anesthetic agents affect transcranial motor evoked potentials. Intraoperatively, opioids are commonly used to supplement volatile anesthetic agents as part of a balanced anesthesia regimen or as an analgesic drug during total intravenous anesthesia (TIVA). Prior animal studies reported concerns regarding fentanyl-induced suppression of motor evoked potentials. Clinically, however, this association has not been described in the literature. We report the critical reduction of tcmEP amplitudes following intravenous fentanyl administration during pediatric spine surgery.

Case Report: A 5 year-old girl with Hurler syndrome and severe kyphosis underwent a kyphectomy with a removal of the L1 vertebral column and posterior spinal fusion with instrumentation from T9 to L3 under TIVA with propofol and remifentanil. She was given boluses of intravenous fentanyl (25 mcg) at three separate times during the course of surgery, each resulting in suppression of the tcmEP amplitudes by greater than 50% lasting for approximately 30 minutes. One bolus resulted in near loss of tcmEP responses during screw placement. In contrast, somatosensory evoked potentials (SSEPs) and EEG remained stable throughout the surgery.

Conclusion: This case highlights the previously undescribed potential of fentanyl to result in isolated critical suppression of tcmEP amplitudes. Remifentanil and fentanyl both may influence latency and amplitude of SSEPs. However, no literature has clearly suggested a clinical association between these agents and tcmEPs. Since a TIVA regimen for intraoperative neurophysiologic monitoring (IONM) is routinely employed, further studies are required to elucidate the direct effects of opiate dosing on tcmEPs, as the medication may directly pose challenges to reliable interpretation of IONM changes.
F50
Spinal Stimulator Artifact Contaminating Evoked Potential Signals in Intraoperative Neuromonitoring (IONM)
Gowri Lakshminarayan, MD; Viet Nguyen, MD; Leslie H. Lee, MD, FACNS, FAAN; Scheherazade Lo; S Charles Cho; Jaime R. Lopez, MD, FACNS

Introduction: A major challenge for the intraoperative neurophysiologist is to isolate the live and evoked signals of interest from various external (electronic) and internal (physiologic) sources of noise. Here we describe a previously unreported external artifact created by a neural spinal stimulator distorting signals and its resolution by turning off the stimulator intraoperatively.

Case Report: A 71 year old male presented with back pain, especially with leaning forward, and difficulty with ambulation secondary to pain. Radiologic studies revealed positive sagittal balance and degenerative scoliosis. Past medical history was significant for chronic low back pain managed with opioids and a spinal cord stimulator placed in 2013 that was reportedly turned off prior to surgery. Proposed surgical intervention included L5/S1 transfemoral laminar interbody fusion with L2-L4 laminotomy and T10-S1 posterior spinal fusion. IONM included 2 channel EEG, upper and lower extremity SSEPs, free and triggered EMG, and MEPs of bilateral T7-9, T10-12, psoas, vastus lateralis, tibialis anterior, abductor hallucis and ankle spindlers. The patient was placed prone under sterile drape. At baseline, all monitored modalities were diffusely contaminated by a regular, sinusoidal artifact. All bedside operating room equipment was investigated in an attempt to isolate the noise source; none were identified. The spinal stimulator patient control device was obtained from a family member. Underneath the operating table, the stimulator was interrogated and found to be still on. It was then turned off, with immediate resolution of the sinusoidal artifact.

Conclusion: This emphasizes the importance of the role of the clinical neurophysiologist in identifying external noise sources that can contaminate recordings, and advocate for interventions that can result in more reliable IONM, with the goal of improving patient outcomes.

F52
Think Outside the Box! Immune Mediated Neuropathy in Neurofibromatosis Type 1 (NF1)
Thandar Aung; Suraj Muley, MD

Introduction: NF1 is a common, autosomal dominant, neurocutaneous disease and neurofibromatous neuropathy has been regarded as an unusual and unexplained complication of NF1.

Case Report: 19 year old male with PMH of NF1, epilepsy and chronic pain syndrome presented with progressive lower back pain and bilateral (BL) mild foot drop without bowel or bladder involvement for 1 year. His exam showed mild BL ankle and toe dorsiflexion weakness with muscle atrophy; symmetric reflexes in patella(3+) and ankle(2+); decreased BL pinprick and loss of vibration sense up to mid-shin. MRI spine revealed enhancing C2-3 neurofibroma with mild Rt ventral cord flattening. EMG revealed absent bilateral sural response. Rt peroneal CAMP was absent at EDB but normal at TA. Rt tibial, median and ulnar motor nerve distal latencies were prolonged at 6.9ms, 5.4ms and 4ms respectively. Decreased Rt tibial CAMP was observed (0.3mV). Rt tibial, median, ulnar and Lt median nerves motor CVs were decreased at 25m/s, 42m/s 32m/s and 36m/s respectively. There was focal ulnar conduction block in the forearm. Rt median F wave minimal latency was prolonged with absence Rt tibial. Needle EMG showed neurogenic motor units in the Rt TA and medial gastrocnemius without abnormal insertion activity. Findings could be explained by inherited neuropathy with superimposed entrapment neuropathies by multiple peripheral neurofibromas as well as by acquired immune mediated neuropathy. MRI neurography showed bilateral sural nerves were splayed by neurofibromas. Spinal fluid showed protein of 300mg/dl. He was started on IVIG 0.5mg/kg/day for 2 days every 3 weeks. After 3 courses, he noticed improvement in the strength as well as back pain.

Conclusion: Patients with neurofibromatosis can present with multifocal conduction slowing that may be related to an immune mediated neuropathy that is responsive to treatment rather than the conduction slowing being related to the neurofibromas.

F53
Acute Intermittent Porphyria (AIP) presenting as Guillain Barre’ Syndrome (GBS)
Vishal Shah; Deborah Y. Bradshaw, MD; Sameer Sharma

Introduction: Acute intermittent porphyria is an acute neurovisceral porphyria caused by a partial deficiency of the heme biosynthetic enzyme porphobilinogen deaminase (PBGD) resulting in accumulation of neurotoxic porphyrins and their precursors. Abdominal pain, peripheral neuropathy and changes in mental status are the classic triad of an acute attack and all are due to effects on the nervous system. Sensory and motor neuropathy is common during acute AIP attacks. However, symptoms can simulate more common disorders such as GBS, resulting in diagnostic delay and avoidable complications. Our case highlights this diagnostic dilemma.

Case Report: 35-year-old female presented with fever, cough and severe abdominal pain for a week followed by slowly progressive arm and leg weakness with tingling and numbness over 1-2 weeks. Neurologic exam was consistent with proximal more than distal weakness in the limbs along with decreased pin prick and light touch sensation on the trunk more than the limbs. Hyporeflexia was present apart from preserved Achilles reflexes. The CSF profile was normal. Initial EMG NCS reported areas of decreased recruitment with signs of early denervation and possible conduction block. IVIG was started for suspected GBS. However, the patient showed no signs of improvement and delirium ensued. At this point, AIP was considered and the urine was noted to turn red with light exposure. Urine porphyrins tested positive. A number of medications known to exacerbate AIP were discontinued and hemin and carbohydrate were administered.. The patient improved with aggressive rehabilitation.

Conclusion: While GBS is the most common cause of acute flaccid weakness, it is not the only cause. Here, proximal weakness and numbness, preservation of Achilles reflexes and the evolution of a delirium suggested the diagnosis of AIP.
F54
Association of Immunologically Mediated Peripheral Neurological Disorders with Systemic Autoimmune Disease.
JaySingh Singh, MD; Sadik Khuder; Assad Amin; Noor Pizzada, MD
Introduction: Autoimmune neurologic and systemic disease may coexist. The pathogenesis of such disorders may involve both environmental triggers and genetic susceptibility.
Methods: A cross-sectional study was conducted utilizing the national inpatient sample data (NIS). Selected autoimmune diseases which included systemic lupus erythematosus, rheumatoid arthritis, Wegener granulomatosis, polyarteritis nodosa, Graves’ disease, Hashimoto’s disease, pachydermoperiostosis, idiopathic thrombocytopenic purpura, hemolytic anemia and autoimmune hepatitis were identified by ICD –9 codes and a series of chi-square analyses were performed to study their relationship with selected peripheral neurological disorders including Guillain-Barre syndrome (GBS), multifocal motor neuropathy, dermatomyositis, polymyositis and myasthenia gravis.
Results: Analysis revealed statistically significant association of dermatomyositis with SLE (2 642, p< .001) Scleroderma (2 1991, p<.001), RA (2 556, p<.001) ITP (2 81.1, p<.004), autoimmune hepatitis (2 24.7, p<.0001), and statistically significant association of GBS was seen with Wegener granulomatosis (2 11.2, p<.0006) and autoimmune hemolytic anemia (2 34.5, p<.0001). No statistically significant association was found between myasthenia gravis, polymyositis, multifocal motor neuropathy and systemic autoimmune diseases.
Conclusion: Statistically significant association of dermatomyositis and GBS with systemic autoimmune disease was noted. It indicates the importance of search for systemic autoimmune disease in patients with immunologically mediated neurologic disorders. Further study of such associations may be important in understanding disease pathogenesis and determining whether such associations influence the prognosis of neurologic disease.

F55
Somatosensory stimulation for induction of cortical plasticity-mechanical versus magnetic stimulation
Monica Christova
Introduction: Peripheral somatosensory stimulation facilitates corticomoar excitability and induces outlasting neuromodulatory changes within the human sensorimotor cortex. Electrical, mechanical or magnetic stimulation can be implemented as intervention protocols.
Methods: Using transcranial magnetic stimulation (TMS) and functional magnetic resonance imaging (fMRI) the study compares the effects of repetitive peripheral magnetic stimulation (rPMS) and mechanical stimulation (MSTIM) on associated motor controlling regions in healthy volunteers. Both interventions were applied on the upper extremity for 20min at 25 Hz frequency and compared to control groups. TMS single and paired pulse motor evoked potentials (MEP) from the hand muscles were recorded at baseline and up to 2h post intervention. Changes in BOLD contrasts were examined at baseline and 1h post intervention.
Results: TMS revealed increased MEP recruitment curves and decreased intracortical inhibition overlasting rPMS with 1h and MSTIM with 2h. FMRI showed increased activation within the contralateral sensorimotor area for 1h after rPMS. The BOLD responses after MSTIM increased within the contralateral S1 (+20%) and M1 (+25%) for 2h.
Conclusion: Whole-hand mechanical stimulation induces more prominent and longer-lasting neuroplasticity changes. These findings are of clinical relevance, for example, when choosing stimulation protocols in rehabilitation of stroke patients.

F56
Rapid Review of Long-Term EEG Recordings from Epilepsy Monitoring
Manfred M. Hartmann; Franz Fuebla; Gerhard Gitsch; Ana Skupch; Johannes Koen; Johannes Herta; Andreas Gruber; Christoph Baumgartner; Tilmann Kluge, PhD
Introduction: A quantitative EEG trending tool called NeuroTrend was used for rapid review of EEG recordings from patients in an epilepsy monitoring unit (EMU) in order to identify ictal events. NeuroTrend identifies periodic discharges and rhythmic EEG activity and shows trends of their localization, frequency and amplitude. It also shows trends of aEEG and background EEG frequency and allows viewing the corresponding EEG. The goal of our study was to assess the review quality that can be achieved in rapid, software-assisted review of long-term EEG.
Methods: Two reviewers used NeuroTrend to review long-term EEGs from 14 randomly selected patients from the EMU at General Hospital Hietzing in Vienna. Only patients who had electrographic seizures were included. Patients with ictal EEGs characterized solely by paroxysmal fast activity were excluded. The reviewers were asked to compile lists of time instances with suspicious patterns that potentially correspond to seizures within a short time. They were not asked to do a detailed assessment of the patterns. The lists of events were compared with the ictal events from the clinical reports from standard clinical review procedures. Sensitivity and specificity were assessed.
Results: Mean recording duration per patient was 3 days and 20 hours. The mean review time spent by the two reviewers was 3 minutes per 24 hours of EEG data. In 57% of patients (8 patients, n=14) the two reviewers found 100 % of all seizures. In 79% of patients (8 patients, n=14) more than 2/3 of seizures could be identified. In 3 patients they found less than 2/3 of the seizures but at least one.
Conclusion: We could demonstrate the suitability of NeuroTrend for rapid review of long-term EEG recordings for EMUs. In our study NeuroTrend allowed identifying ictal events with a high mean sensitivity of 81% and a high mean specificity of 63%, while spending only 3 minutes for reviewing 24 hours of EEG recordings on average.

F57
Choosing Continuous Pulse Oximetry Thresholds in the EMU
Daniel Goldenholz, MD, PhD; Amanda Kuhn; Sara Inati, MD; William H. Theodore, MD
Introduction: Cardiopulmonary dysregulation precedes sudden unexplained death in epilepsy. Some propose using continuous pulse-oximetry for epilepsy monitoring unit safety. However, no optimized oximeter thresholds exist.
Methods: We observed seizure onset, offset and generalized tonic-clonic (GTC) seizure onset for 7104 hours (46 patients: 42 video-EEG/7 intracranial-EEG recordings), assessing SaO2 thresholds for true (ictal) and false (interictal) alarms. Any threshold-crossing interictal oximeter reading was considered “false.” Hourly interictal false detections were averaged for each subject and used to compute mean between-alarm time.
Results: Twenty-six recordings from 23 unique patients contained seizures. For false alarms, the medians (across patients) of the mean (within patients) number of minutes between alarms for $\text{SaO}_2\%$ thresholds of 90, 88, 86, 84, 82, 80, and 78 were 3, 11, 25, 62, 96, 146 and 223, respectively. GTC detections for those thresholds were: 96%, 94%, 94%, 84%, 84%, 81%, and 78% while non-GTCs detections were: 57%, 40%, 36%, 32%, 27%, 25%, and 22%.

Conclusion: $\text{SaO}_2\%$ threshold of 80-86% results in one false alarm per 0.5-2 hours, detecting 81-94% of GTCs and 22-32% non-GTCs. Higher thresholds detect additional seizures but increase false alarms unacceptably (1 per <11 minutes). Continuous pulse oximetry is a low-cost, reliable safety tool.

F59
Making the Diagnosis in Frontal Supplementary Motor Area Seizures with In-Home Video Capture of Seizure Semiology
David J. Sinclair, MD; Mecheri Sundaram, MD, MBBS, FACNS

Introduction: The frontal lobes are the second most common location for seizure onset. Many seizure types have been described but general categorizations include focal clonic, asymmetric tonic, hyperkinetic, absence, opercular, and hyperkinetic; many of these types are misdiagnosed as psychogenic attacks.

Case Report: Our patient was a 41 year old who was referred to me for paroxysmal episodes of unsponsiveness without loss of awareness, body tightening, and then occasionally loss of postural tone or abnormal movements. These had been present off and on for 5 years. “Pseudoseizure” was suspected upon referral to me. Routine electroencephalography (EEG) and sleep deprived EEG were both normal. Differential diagnosis was restricted to epilepsy, convulsive syncope, hypoglycemic or hyperglycemia induced seizures, hypocalcemia induced spasm or psychogenic seizure. A 72 hour in-home ambulatory video-EEG was performed. The event monitor was actuated six times. In all instances, surface EEG activity was normal other than EMG artifact. During the second night of recording the event pictured occurs. This patient demonstrated paroxysmal asymmetric tonic posturing that is pathognomonic for left frontal supplementary motor area involvement (M2e).

Conclusion: This case demonstrates the capability of in-home video-EEG to capture and adequately describe a frontal supplementary motor area seizure. This particular seizure type presents a unique diagnostic dilemma. Since the characterization of the M2e sign and its naming by Ajmone-Marsan and Ralston in 1957, identification of this seizure focus continues to rely on serendipitous though rare cortical EEG changes, or adequate characterization of seizure semiology; intracranial EEG recording can be useful in selected cases. Ambulatory video-EEG monitoring of sufficient duration will capture an event in a patient capable of actuating the event monitor.

F60
Prolonged Seizure Remission after Intracerebral Depth Electrode Placement
Angelica Lee, DO; Vinita J. Acharya, MD; Michael Sather; Jayant Acharya, MD

Introduction: Invasive monitoring with bilateral intracerebral depth electrodes is used to identify the side of seizure onset and to decide whether or not surgery can be performed in patients with scalp EEG findings suggesting bilateral temporal lobe epilepsy. Electrode insertion itself is usually not therapeutic. We report a patient with pharmacoresistant focal epilepsy who had prolonged seizure remission after invasive monitoring with bitemporal depth electrodes.

Case Report: A 29 year old woman presented with focal dyscognitive and generalized tonic-clonic seizures since age 16. She tried and failed multiple antiseizure drugs. On noninvasive video-EEG monitoring, there were no interictal epileptiform discharges, but 10 seizures were recorded. 6 showed right temporal EEG onset with concordant clinical lateralizing signs (right eyelid blinking, left clonic jerking); 1 showed left temporal onset without clinical lateralizing signs and, in 3 seizures, ictal onset could not be lateralized. Brain MRI showed subtly increased signal, but normal volume, in the left hippocampus. FDG-PET was normal. Neuropsychology indicated left temporal dysfunction. Invasive monitoring with bitemporal depth electrodes showed bitemporal interictal spikes. 7 seizures were recorded, including 3 focal seizures with left eye blinking, right clonic activity and left temporal ictal EEG onset, and 4 seizures with no EEG changes. Due to conflicting findings and lack of a clear unilateral focus on presurgical evaluation, surgery was not performed. However, after discharge from the unit, she reported complete resolution of seizures for 22 months, followed by recurrence.

Conclusion: Intracerebral depth electrode placement can rarely result in prolonged remission of seizures even if surgical removal of the focus is not performed. We speculate that the mechanism is disruption of a critical portion of the epileptic network by the electrodes.
S1

Resting-State EEG Connectivity in Critically Ill Patients

Manuel B. Garcia, MD; Valia Rodriguez, MD, PhD; Lester Mele, PhD; Adonisbel Valero

Introduction: In this study as part of a protocol that aims to standardize the cognitive assessment of critically ill patients we carried out a resting-state EEG functional connectivity analysis.

Methods: We evaluated changes in synchronization features and graph theoretical properties quantified by synchronization likelihood in patients with different scores in the Glasgow Coma Scale- GWS. EEG segments were manually selected from continuous EEG. Segments included at least 3min in closed eyes resting state. In all segments synchronization likelihood (SL) and graph parameters in delta (1-4Hz), theta (4-7), alpha (8-13) and beta (13-30) frequency bands were calculated.

Results: Results showed that strength of the overall spatial synchronization in delta, theta, alpha and beta frequency bands increased with the depth of coma. Specifically synchronization in conscious patients was higher in frontal, temporal, occipital and frontotemporal regions but long distance synchronization was weaker than in comatose patients. Network properties as clustering coefficient -C- and local efficiency -Le- showed a similar pattern: both features in all frequency bands were higher in comatose than in conscious patients. However only in the theta band the change in C and Le with the score of GWS was gradual. Properties as characteristic path length and global efficiency did not show a consistent relationship with the GWS score.

Conclusion: Overall spatial synchronization differed between comatose and conscious patients. The increment in the overall spatial synchronization observed in comatose patients could be a sign of the altered interaction between the thalamocortical system and the forebrain systems that is restored with awareness. Changes in the topology of the neural networks from coma to awareness could be a potential functional marker for the assessment of consciousness recovery in ICU.

S2

Quantitative Detection of Seizure Onset

Kumar Sannagowdara, MD; Kurt Hecox, MD, Phd

Introduction: This is a study on direct comparison of the accuracy of detecting seizure onsets using quantitative analysis of EEG signals versus the performance of trained epileptologists. One motive for this study is to better separate apparently generalized seizure onsets from onsets, which are focal in origin with secondary generalization.

Methods: Seizures were chosen from a larger set based upon the availability of simultaneous artifact free segments of intra and extra cranial data, focal onset seizures (based upon the intracranial data) distributed in different pediatric age groups. There were ten samples of ten second each, intracranial EEG segments with seizures were chosen and along with ten segments which did not contain seizures. The simultaneous surface EEG recordings of the same duration with and without seizures were obtained. Four-experienced epileptologist's were asked to label each of the segments as containing or not containing a seizure onset. None of the judging epileptologist’s was involved in the selection of the EEG segments. The quantitative analysis applied Kolmogorov entropy, correlation dimension, tests using surrogates and eigenvalues. Since this data was categorical, chi-square analyses were applied

Results: There were a total of 80 judgments made by the epileptologist’s, of which 42 were correct. There were 20 categorizations by the quantitative metrics, of which 19 were correct. This difference in performance was significant at the P<.01 levels

Conclusion: It is possible for quantitative measures to out-perform trained human observers in the detection of seizure onset in blinded studies of surface EEGs. This raises the opportunity for the earlier detection of seizure onset, prior to secondary generalization.

S3

Functional Network Organization Derived from EEG Reveals Postsurgical Evolution in Temporal Lobe Epilepsy Patients.

Lilia Maria Morales Chacon, PhD

Introduction: Objective: To evaluate functional network organization derived from postsurgical Resting-state Electroencephalogram (EEG) in temporal lobe epilepsy patients.

Methods: 30 adult patients with intractable temporal lobe epilepsy (TLE) submitted to temporal lobectomy were evaluated. Resting-state EEG recordings were obtained at 24 months after resection. We investigated the characteristics of the functional brain network of TLE patients that were seizure-free (SF) and non-seizure free (NSF) after surgery for right or left temporal lobe (RTLE and LTLE respectively). Epileptogenic zone lateralization and postsurgical evolution were taken into account. Synchronization likelihood (SL) was used to characterize synchronization in different frequency bands of the EEG recorded under resting-state eyes-closed condition. Graph theoretical properties were reconstructed from the synchronization matrix and characterized by a clustering coefficient (a measure of local connectedness) and shortest path length (a measure of overall network integration).

Results: Results showed that differences between surgical groups were more prominent in the alpha band (8-13Hz). In this band, SF and NSF RTLE patients significantly differed in the synchronization strength between occipitotemporal and frontotemporal leads; no significant difference was found however in the synchronization of LTLE patients. Network property analysis showed that shortest path length and clustering coefficient were smaller in SF than in NSF.

Conclusion: These results suggest that seizure-free patients have a less organized functional brain network (lower clustering coefficient and smallest path length) than non-seizure-free patients, a type of network organization that probably hampers seizure generation and propagation. Our results even when preliminary increase the insight into functional network reorganization related to surgical outcome.
**S4**  
Utilization of Quantitative EEG Trends for Critical Care Continuous EEG Monitoring: A Survey of Neurophysiologists  
**Christa B. Swisher, MD; Saurabh R. Sinha, MD, PhD, FACSNS**

Introduction: Objective: Quantitative EEG (QEEG) can be used to assist with review of the large amounts of data generated by critical care continuous EEG (CCEEG) monitoring. This study aimed to identify current practices regarding the use of QEEG in monitoring of critical care patients.

Methods: Methods: An online survey was sent to 796 members of the American Clinical Neurophysiology Society (ACNS). The survey invitation instructed only neurophysiologists to participate.

Results: The survey was completed by 97 neurophysiologists. Survey respondents reported that neurophysiologists and neurophysiology fellows are most likely to serve as QEEG readers (97% and 52%, respectively). However, 21% of respondents reported non-neurophysiologists are also involved with QEEG interpretation. The majority of non-neurophysiologist QEEG data review is aimed to alert neurophysiologists to periods of concern, but 22% reported that non-neurophysiologists use QEEG to directly guide clinical care. QEEG was utilized most frequently for seizure detection (92%) and burst suppression monitoring (59%). A smaller number of respondents utilize QEEG for monitoring depth of sedation (29%), ischemia detection (28%), vasospasm detection (28%) and prognosis after cardiac arrest (21%). About half of respondents do not review every page of the raw CCEEG record when using QEEG. Respondents prefer a panel of QEEG trends displayed as hemispheric data, when applicable. There is substantial variability regarding QEEG trend preferences for seizure detection and ischemia detection.

Conclusion: Conclusions: QEEG is being used by neurophysiologists and non-neurophysiologists for applications beyond seizure detection, but practice patterns vary widely. There is a need for standardization of QEEG methods and practices.

**S5**  
Effect of Electrode Reduction on Detection of EEG Patterns in Intensive Care Patients  
**Franz Fürbass; Johannes Herta; Johannes Koren; Andreas Gruber; Christoph Baumgartner; Tilmann Kluge, PhD**

Introduction: We investigated the influence of the number of electrodes on automatic detection of EEG patterns in intensive care patients.

Methods: Long-term EEGs of 83 patients (total 6733h, mean 73h) with 19 electrodes were recorded in two intensive care units. Two EEG experts independently annotated periodic discharges (PD), slow rhythmic activity (SRA, <4Hz), fast rhythmic activity (FRA, >=4Hz), and burst suppression patterns (BSP) in the first minute of each recording hour. A fully automatic computer algorithm was used to detect the EEG patterns. We then reduced the number of electrodes available for the automatic detection. These detection results were compared to the consensus annotations of the reviewers to quantify sensitivity and the number of electrodes for which sensitivity dropped more than 15% (D15%).

Results: PD were detected with a sensitivity of 87% in the EEGs including 19 electrodes and reached a D15% already at 14 electrodes. SRA patterns were detected with sensitivity=95% and D15%=11, FRA with sensitivity=94% and D15%=12, and BSP with sensitivity=90% and D15%=7.

Conclusion: PDs are most susceptible to electrode reduction; BSPs can be detected adequately even with 7 electrodes. This study shows that the number of required EEG electrodes strongly depends on the EEG pattern of interest.

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**S6**  
Combining Inverse Solutions and EEG-Functional Connectivity to Assess for Preserved Cognitive Processing in ICU.  
**Valia Rodriguez, MD, PhD; Adianes Herrera; Adonisbel Valero**

Introduction: Deciding whether a patient with a disorder of consciousness -DOC- is in a vegetative or a minimal conscious state is challenging. Lack of sensitive and objective diagnostic techniques determines that about 40% of cases are misdiagnosed. The situation is more complicated in intensive care environment where a wrong diagnosis could affect the quality of treatment. In this study as part of a protocol that aim to standardize the cognitive assessment of critically ill patients we carried out a combined inverse solution and EEG functional connectivity analyses to explore differences between patients with and without DOC in intensive care unit -ICU-

Methods: Data was obtained from EEG of critical ill patients with different degree of awareness (ie: conscious, stupor, LIS, coma and UWS). For analysis EEG segments were obtained from different auditory stimulation conditions.

Results: Results showed that main differences between conscious and coma or UWS were: 1- distinctive activations of frontoparietal areas, including precuneus and cuneus in conscious, stupor and LIS patients, especially during the presentation of structured stimuli (ie: music, message); 2- A pattern of synchronization at the level of electrodes characterized by an increase in the clustering and strength of connections during the presentation of structured stimuli in conscious, stupor and LIS patients.

Conclusion: Even when our results are preliminary they suggest that the combination of these analysis techniques could have enough sensitivity to detect preserved cognitive processing in ICU.

**S7**  
Quantitative Measurement of EEG Reactivity  
**Michael Clark; Emily Johnson, MD; Eva K. Ritzl, MD, FACSNS**

Introduction: EEG reactivity is an important predictor of outcome in brain injury, including anoxic injury and traumatic brain injury. However, inter-rater reliability of EEG reactivity can be low. Hermans et al published an algorithm to detect EEG reactivity using quantitative measures in comatose patients undergoing EEG. The temporal brain symmetry index (tBSI) was found to be the best predictor of reactivity. We aimed to test this method using patients undergoing continuous EEG monitoring for altered mental status.

Methods: We identified 15 consecutive patients undergoing continuous EEG monitoring for altered mental status with documented stimulation for reactivity testing. Sixty-second periods before and after stimulation were preprocessed with a sixty hertz notch filter and decomposed into independent components for EKG and artifact removal. Five qEEG statistics (the temporal brain symmetry index, a peak comparison classification) were calculated over various frequency bands for each channel in the EEG patient file.

Results: Replication of the quantitative analysis described by Hermans et al was feasible. Statistical analysis is ongoing, and results will be presented.
POSTER ABSTRACTS

Conclusion: Validation of the new quantitative EEG algorithms is important for future research and has implications for clinical care.

S8
Choosing Wisely: Top Five List for Pediatric Outpatient EEG
Brian Grabert, MD

Introduction: Choosing Wisely initiated in 2012 by ABIM and consumer oriented organizations to encourage “physicians, patients, & others to think and talk about tests and procedures that may be unnecessary.” The AAN is the only neurological society out of 65 Societies to publish a list. I propose a Choosing Wisely Top Five list for Pediatric Outpatient EEG to include: Behavior/Mood Disorders; ADHD; Syncope; Developmental Delay/ Autism; Headache.

Methods: 474 consecutive, outpatient EEGs, ages 4 to 18 yr., were read by one Pediatric Neurologist and correlated with presenting diagnosis and the specialty of the ordering Practitioner. The referral diagnosis was confirmed by EEG technician’s history and any mention of seizures, the EEG was placed in this category. Ordering diagnoses were collapsed into 6 categories: Seizures; Developmental Delay/Autism; ADHD; Syncope; Headache; Behavioral/ Mood disorders. Four Practitioner Categories were formed: Pediatricians; Pediatric Neurologists; Pediatric Psychiatrists; Family Practitioner/PAs/NPs. EEGs were dichotomized into Normal or Epileptiform

Results: Most epileptiform EEGs were recorded in patients with a diagnosis of seizures (76%). Of all EEGs ordered for seizures 26% were epileptiform. Epileptiform EEGs were seen in 3.5% of EEGs ordered for Behavior disorders; 4.9% for ADHD; 9% for Developmental Delay/Autism; 0% for Syncope, & Headache Child Psychiatrists ordered the most outpatient EEGs (43.5%); 4.4% epileptiform. Pediatricians 27.6% of EEGs; 16.8% epileptiform. FP/NPs ordered 19.4% EEGs; 5.4% epileptiform. Pediatric Neurologists 6.8% EEGs; 37.5% epileptiform.

Conclusion: The major reason to order an EEG in an outpatient pediatric population is to rule out seizures. This study shows that epileptiform EEGs ordered for 5 common diagnoses other than seizures are very infrequent. EEGs should not be routinely ordered for these five diagnoses. Consideration should be given to limiting which practitioner can order an EEG.

S9
Automatic Interpretation of EEGs for Clinical Support
Amir Harati, PhD; Meyyam Golmohammadi; Mercedes P. Jacobson, MD; Silvia Lopez; Iyad Obeid, PhD; Joseph Picone; Steven Tobochnik, MD

Introduction: Manual review of an EEG by a neurologist is time-consuming and tedious. Inter-rater agreement is low for annotation of low-level events such as spikes and sharp waves. A clinical decision support tool that automatically interprets EEGs can reduce time to diagnosis, reduce error and enhance real-time applications such as ICU monitoring. We present a high performance classification system based on principles of big data and machine learning.

Methods: A hybrid machine learning system was developed using a combination of hidden Markov models (HMMs) for sequential decoding and deep learning for postprocessing. The system detects three events of clinical interest: (1) spike and/ or sharp waves, (2) periodic lateralized epileptiform discharges, and (3) generalized periodic epileptiform discharges. The system also detects three events used to model background noise: (1) artifacts, (2) eye movement and (3) background.

Results: A baseline system, originally developed to give high performance on the MIT-CHB Corpus, was evaluated on this task. This system, which consists of heuristics based on waveform properties, delivered a 99% detection rate with a 37% false alarm rate. It is not uncommon for such research systems to fail on clinical data. Clinicians have long complained about the high false alarm rates of such systems, and indicated a detection rate of 95% with a false alarm rate below 5% was required for clinical use of this technology. Our system produced a detection rate of 89% while maintaining a false alarm rate of 4%. The postprocessing also improved accuracy on spike detection from 25% to 55%.

Conclusion: Clinical use of such systems is limited due to poor classification performance — specifically a high false alarm rate. The existence of the TUH EEG Corpus provides for the first time a sufficient amount of data to apply powerful machine learning algorithms. As a result, performance is now approaching that required for clinical acceptance.

S10
Is 100 Seconds of NREM Sleep Sufficient to Diagnose ESES?
Amanda R. Weber, DO; Dara Albert; Anup Patel

Introduction: Strategies for diagnosing ESES vary widely among interpreting neurologists. Standard methods can be time consuming and require a child to be able to tolerate an overnight EEG. Our aim was to evaluate if the spike-wave index (SWI) for the first 100 seconds of sleep is reflective of the SWI based on a conventional method.

Methods: We reviewed EEGs from 2005-2011 that were considered diagnostic of ESES by the original interpreting neurophysiologist based on unspecified methods. The SWI for the first and last sleep cycles (long method) was calculated by two independent neurophysiologists; two different neurophysiologists then calculated SWI for the first 100 seconds of sleep (short method). ESES was defined based on a SWI of > 85%. The two SWI scores were compared.

Results: Fourteen EEGs were reviewed. Despite being considered by the initial interpreter as diagnostic of ESES, only 4 of the studies had a SWI of >85% based on the long method. The short method identified 5 of those studies that had a SWI >85% in the first 100 seconds of sleep. For a diagnosis of ESES, the sensitivity of the short method is 100%, and the specificity is 90%. The Spearman’s correlation coefficient is 0.5815 (p-value=0.0292), indicating the two methods are moderately correlated.

Conclusion: The SWI for the first 100 seconds of NREM sleep is predictive of the SWI for the entire first and last sleep cycle with a good sensitivity and specificity in our cohort. This suggests an alternative method for diagnosing ESES without requiring analysis of a full night of sleep.

S11
Delay in Diagnosis of Hashimoto Encephalopathy presenting with De Novo Seizures and Status Epilepticus: A Rare Entity or An Underdiagnosed Condition?
Arun Swaminathan; Muhammad S. Zafar, MD; Gulam Khan, MD; Meriem Bensalem-Owen, MD, FACNS

Introduction: Hashimoto encephalopathy (HE) also known as steroid-responsive encephalopathy associated with autoimmune thyroiditis (SREAT) is considered a rare and underdiagnosed cause of seizures. Delay of treatment of HE is not uncommon due to the relapsing-remitting course and diversity of symptoms. We
describe electroencephalographic (EEG) features of 5 patients with HE whose initial presentation was either seizures or status epilepticus.

Methods: Retrospective review of clinical and EEG features of patients diagnosed with HE presenting with seizures or status epilepticus.

Results: Over a 2 year period, 5 patients, all women, were identified. Mean age was 48 years. Diagnosis was made approximately 6 months after initial presentation (range 3 weeks to 18 months). On continuous video-EEG monitoring and standard EEG, all patients had slowing and interictal epileptiform discharges. Two patients presented with sub-clinical status epilepticus.

Conclusion: In this short series, there was considerable delay in diagnosis of HE (up to 18 months in one case). All patients presented with either seizures or status epilepticus. Unlike other encephalitides such as NMDAr encephalitis, where ictal extreme delta brushes have been described, there is no specific ictal or inter-ictal EEG pattern in HE that may guide to an early diagnosis and treatment.

S12
Reliability of Scalp EEG in the Epilepsy Monitoring Unit
Erik Sass, MD; Mackenzie C. Cervenka, MD; Dana Boatman, PhD

Introduction: Epilepsy patients are assessed for surgical candidacy with scalp video EEG collected in Epilepsy Monitoring Units (EMU). Reliable data are essential for seizure localization. Our aim was to assess the reliability of scalp EEG recordings with the auditory N100 event-related potential (ERP) as it is an obligatory and robust signal and is reliable in intracranial recordings.

Methods: Patients age 18-65 years admitted to the Johns Hopkins EMU from August 2014-June 2015 were included. Patients with intellectual disability, abnormal hearing, and those non-fluent in English were excluded. Auditory ERPs were generated with pure tone stimuli (passive oddball paradigm; 1000/1200Hz; 300 trials). Noisy channels/trials were excluded. Remaining trials were averaged and N100 was identified. Within patients response latencies and amplitudes were compared between test and retest with paired t-tests and Spearman’s rank correlation coefficient given small sample sizes.

Results: Recordings from 14 patients were analyzed from those with interpretable midline (11/14) and lateral N100 (10/14). There was no significant difference between midline latencies or amplitudes (latency p=0.78; amplitude p=0.92). Midline latencies (r=0.92, p=0.0001) were more correlative than amplitudes (r=0.05, p=0.87). Right latencies were more correlative than left (right r=0.84, p=0.003; left r=0.54, p=0.11). In patients with focal epilepsy, latencies on the side contralateral to a known seizure focus were more correlative than ipsilateral (contra r=0.88, p=0.02; ipsi r=0.44, p=0.38).

Conclusion: Auditory N100 latencies are reproducible across multiple days of EEG recording for individual patients. There was a stronger correlation between response latencies contralateral to a patient’s seizure focus than ipsilateral. There was poor correlation between amplitudes within subjects.

S13
Yield of ambulatory EEG
Pedro Balaguera, MD; Anna Serafini, MD; Stephan Schuele, MD, MPH, FACNS

Introduction: Ambulatory EEG (AmbEEG) is frequently used in patients with new onset epilepsy and normal routine EEG to capture interictal epileptiform abnormalities. AmbEEG can also be used to record events in patients with frequent clinical seizures to confirm the diagnosis or to capture subclinical seizures in patients with epilepsy who may not be aware of all their events. The yield of AmbEEG in answering the clinical question for these three indications is unclear.

Methods: Complete AmbEEG tracings of 155 patients (290 recording days) were reviewed by a certified neurophysiologist. AmbEEG were ordered for the three indications outlined above.

Results: The most frequent indication was for detecting subclinical seizures (73 patients, 47%). Events were recorded in 15 patients (20.5%); 3 had paroxysmal events (PE) and 12 had epileptic seizures (9 focal epilepsy and 3 generalized epilepsy). The second most frequent indication was for capturing clinical events (47 ambulatory). Events were recorded in 16 patients (34%); 12 were PE and 4 were epileptic seizures (2 focal epilepsy and 2 generalized epilepsy). The third indication was diagnostic, for determining the epilepsy syndrome, in the remaining 35 studies. Epileptiform discharges were found in 7 of them (20%); 2 patients were diagnosed with focal epilepsy and 5 with generalized epilepsy. In 3 out of those 35 patients, PE were captured.

Conclusion: In patients with clinically controlled epilepsy, AmbEEG can capture unrecognized subclinical seizures in about 20% and are an important tool to optimize management and direct safety precautions. Clinical events were recorded in about one third of patients which is approximately half of the yield reported in EMU permitting medication taper if necessary. The incremental value of detecting epileptiform activity in patients with a normal initial EEG was only 20% which is slightly lower than historical data showing an additional 35% of epileptiform abnormalities by performing up to 4 serial EEGs.

S14
14- and 6 Hz Positive Spikes in Pediatric Population
Osama Muthaffar, MD, SBPN, CSCN; Roy Sharma, RET, CBET; Miguel Cortez, MD, CSCN

Introduction: Benign EEG variants (bEEGv) are patterns with epileptiform appearance and are clearly distinguished from the ongoing background EEG activity and are of doubtful clinical significance. These bEEGv can be complex superimposition of wave forms that can be challenging and confuse the reader. There are well described bEEGv however, the pattern of 14 and 6 Hz positive spikes is under reported in the current literature of pediatric electroencephalography.

Methods: We reviewed a random cohort of 650 pediatric EEGs (0 to 18 years) at the Hospital for Sick Children in Toronto, from April 2013 to September 2015. All digital EEG recordings were performed with the 10-20 system of electrode placement. The prevalence of 14 and 6 Hz positive spikes variant was compared with the reported literature.

Results: Four (0.62%) out of 650 EEGs fulfilled the criteria for 14 and 6 Hz positive spikes. All four patients had history of seizures. 14 & 6 per second positive spiking (ctenoids) were reported by Schwartz and Lombroso (1968) in (55%) of 118 normal students. Metcalf (1963) reported (28%); Gibbs and Gibbs (1964) (2%); Lombroso et al., (1966) (58%); Demerdash et al. (1968) reviewed 472 normal children’s EEGs and found (7%) and (16.2%) of 599 by Eeg-Olofsson aged 1-15 years (1971). Charles and Zeigler in 1963 reported it in (6%) Of 2200 adults and children EEGs from (1956-to-1958). Domenici et al (1991) reported it in (17%) of 109 children.
Conclusion: The 14 and 6 Hz positive spikes are of rare occurrence in digital EEG recordings compared to the classical years of analog EEG recordings. Awareness of the occurrence of these EEGs will continue to prevent misdiagnosis and unnecessary pharmacological treatments.

S15
A Micro-Subdermal Wire Electrode (μSWE): a Chronic, 40-Gauge Ag-Ag/Cl EEG Electrode
John R. Ives, B.Sc.

Introduction: Traditional cup EEG electrodes are not designed for nor are they well suited for very long-term (days, weeks, or months) EEG recordings.

Methods: We have developed an extremely fine, subdermal, pure silver wire EEG electrode that is only 40-gauge (0.004" or 0.11mm) in diameter. The μSWE is similar in design to the original Subdermal Wire Electrode (SWE), as it is a pure silver wire with a Teflon insulation and bored about 3mm at the tip to form a high quality, low noise Ag-Ag/Cl biopotential (EEG) recording electrode. However, now the μSWE can be inserted into the subdermal space using a 32-gauge (0.010", or 0.25mm diameter) hypodermic needle instead of a 25-gauge (0.019" or 0.50mm diameter) needle. The μSWE insertion needle is now the same size as standard acupuncture needles which are routinely placed in awake patients without significant discomfort. As with the standard SWE, the μSWE once placed and fixed is capable of recording for months without maintenance or adjustment. The μSWE is very small and thus compatible with CT, MR, X-ray, SPEC, MEG and TMS. The μSWE has a higher impedance (<25kΩ compared to <5kΩ); however, modern, very high input impedance EEG amplifiers can readily accommodate this impedance without any degradation in EEG signal quality.

Results: Limited clinical experience has been obtained at the U of Lausanne Clinics on selected chronic, comatose patients. They were recorded with 11 μSWEs each. We “did not find any significant difference in the recordings quality” compared to regular SWE or standard EEG electrodes; per Dr. Andrea Rossetti.

Conclusion: The μSWE may have an application in very long-term EEG recordings (cEEG). The μSWE may also be used in other EEG situations where it’s extremely small size, high quality, and maintenance-free EEG recording characteristics are an asset (cEEG, ITM, AEEG) and superior to traditional cup electrodes in specific applications.

S16
EEG Spikes are Associated with Sleep Architecture in Children with Epilepsy
Sejal V. Jain, MD; Jennifer Vannest; Jeffrey Tenney, MD, PhD; Thomas Maloney; Caroline Spencer; Melodie Dixon

Introduction: Benign epilepsy with centro-temporal spikes (BECTS) is one of the most common epilepsy in children and is associated with location-specific spikes that are amplified during sleep. Sleep-related co-morbidities also are common in children with BECTS. Despite these, limited data evaluate the impact of sleep on epilepsy parameters. The purpose of this study was to identify the association of sleep architecture with EEG spikes.

Methods: Six to 12 years old children with BRE were enrolled and overnight electroencephalograms (EEGs) were performed at the baseline time-point, prior to medication onset. The EEGs were scored for spikes during sleep and wakefulness. The EEGs were scored for spikes as left sided, right sided and if present in both sides—bilateral, and for sleep architecture as wakefulness after sleep onset (WASO: awake period after sleep onset, measured in minutes), total sleep time (TST: total duration of sleep during the study, measured in minutes) and arousal index (AI: numbers of arousals per hour of sleep) were calculated. Regression analysis was performed to establish association between these sleep parameters and EEG spikes.

Results: Thirty three children, 18 male, 15 female, aged 5.1-12.7 were included. Sleep architecture showed WASO mean 28.2, standard deviation (SD) 35.1, AI 6.7 SD 1.6 and TST 515.3 SD 51.2. On regression analysis, increase in WASO was significantly associated with increase in spikes during wakefulness on the right side (R2= 0.5, p=0.03) and AI with awake discharges on the left side (R2=1.3, p=0.01). No significant association was seen with TST.

Conclusion: The results show that increased arousals and increased wake time in sleep are associated with worsening spikes during daytime in children with BECTS. Hence, poor sleep may be associated with increased spikes in children with BECTS. Further studies are needed to understand the impact of sleep and spikes on neuropsychological outcomes.

S17
Automated EEG Interpretation: A Clinical Evaluation and Validation
Justin Cheongstannoy, MD, MBA; Ning Zhong, MD, PhD; Hiroshi Shibasaki, MD, PhD, FACNS; Masatoshi Nakamura; Marc R. Nuwer, MD, PhD, FACNS

Introduction: Due to its complexity and variability, EEG is conventionally interpreted by neurophysiologists through visual analysis. However, the accuracy of EEG reports is dependent upon the experience of the reader. If EEG can be automatically interpreted, it is hypothesized to be more quantitative and more objective than visual inspection by different reviewers. Drs. Hiroshi Shibasaki and Masatoshi Nakamura have developed a computer-assisted system (QP-270) for automatic, systematic and comprehensive interpretation of the adult EEG.

Methods: To test inter-reader reliability, we collected 90 routine 30-min EEGs (60 outpatient and 30 inpatient) and ran QP-270 software to obtain EEG interpretation and results. We then compared these automated results to those made by attending level neurophysiologist through visual analysis.

Results: 1) Of the 90 EEGs analyzed, 56 were normal and 34 abnormal studies according to visual inspection; QP-270 recognized 18 normal studies, and labeled the remaining 72 as abnormal. 2) Inter-reader (Inter-rater) agreement Kappa between QP-270 vs visual analysis to determine normal vs abnormal study was 0.225; QP-270 had high false positive rate mis-interpreting normal studies as abnormal EEGs. 3) QP-270 showed high sensitivity detecting the abnormal EEG. 4) When analyzing abnormal EEG, QP-270 did well by detecting slow EEG rhythms which were either focal or diffuse; it also demonstrated fairly accurate detection of epileptiform discharges. Inter-reader agreement Kappa was close to 0.3 when analyzing abnormal studies.

Conclusion: QP-270 may serve as an educational tool to help neurology trainees recognize abnormal EEG findings including spike and sharp waves as well as focal findings in the record. In this study, QP-270 sometimes labeled episodes of drowsiness as abnormal. Therefore, QP-270 may be optimal in assessing only those portions of the record which demonstrate maximal wakefulness.
S18 Neuroimaging Abnormalities That Predict Mortality In Patients With Generalized Periodic Discharges (GPDs)
Neville Jadeja, MD, MPH; Reza Zarnegar, DO, FACNS; Alan D. Legatt, MD, PhD, FACNS

Introduction: Neuroimaging abnormalities have been described in association with generalized periodic discharges (GPDs) but their prognostic significance is unclear. We investigated the association of specific neuroimaging abnormalities with mortality in hospitalized patients with GPDs.

Methods: We reviewed neuroimaging data from inpatients at three different hospital sites affiliated with our institution in whom GPDs were reported by fellowship-trained electroencephalographers during the years 2010-2012. Neuroimaging abnormalities were classified as acute and chronic infarctions, leukoaraiosis, intracranial hemorrhage, diffuse axonal brain injury and mass lesions. Cox regression was used to determine statistical associations. Other clinical data was included in the multivariate analysis.

Results: We identified 114 patients with GPDs. Their mean age was 69.9 ± 14.1 SD years, and 71 (62.3%) were women. There were 56 inpatient deaths (49%). In the multivariate analysis, leukoaraiosis was found to be significantly associated with increased in-hospital mortality as shown in the Table.

<table>
<thead>
<tr>
<th>Neuroimaging/Abnormalities</th>
<th>Number</th>
<th>Hazard Ratio</th>
<th>P-Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Acute Infarction</td>
<td>17</td>
<td>1.178</td>
<td>0.768</td>
</tr>
<tr>
<td>Chronic Infarction</td>
<td>9</td>
<td>1.945</td>
<td>0.291</td>
</tr>
<tr>
<td>Leukoaraiosis</td>
<td>24</td>
<td>2.755</td>
<td>0.040</td>
</tr>
<tr>
<td>Intracranial Hemorrhage</td>
<td>5</td>
<td>1.070</td>
<td>0.912</td>
</tr>
<tr>
<td>Diffuse Anoxic Brain Injury</td>
<td>7</td>
<td>1.352</td>
<td>0.663</td>
</tr>
<tr>
<td>Mass lesions</td>
<td>7</td>
<td>3.854</td>
<td>0.056</td>
</tr>
</tbody>
</table>

Conclusion: Unexpectedly, cerebral leukoaraiosis was the only neuroimaging abnormality that was significantly associated with in-hospital mortality in patients with GPDs. The smaller sample size for the other neuroimaging abnormalities may have contributed to their failure to reach statistical significance. Further studies are needed to confirm these results and to determine if the severity of the leukoaraiosis affects mortality.

S19 Perception of HD EEG Studies Among HD EEG Clinical Users
Yi-Jou Tsai, MD; Robert D. Bolan, MD; Leonardo Bonilha, MD, PhD; Jonathan J. Halford, MD, FACNS

Introduction: During high-density electroencephalogram (HD EEG), a recording is made from a larger numbers of electrodes (64, 128, or 256) than conventional EEG, theoretically improving localization of intracranial electrical signals. This higher EEG spatial resolution, as well as lower cost compared to magnetoencephalography (MEG), could make it a useful tool for epilepsy pre-surgical evaluation. However, HD EEG continues to be used infrequently for clinical purposes.

Methods: We developed a 17 question questionnaire to assess perception of HD EEG studies among HD EEG providers. E-mail addresses of HD EEG users were obtained from Electrical Geodesic (EGI) and the questionnaire was sent as an attachment. Responses were obtained from 14 institutions and results were tabulated with percentages calculated for multiple choice questions.

Results: Results suggested that the most common usage of HD EEG was a one-hour 256 lead EEG in adult patients undergoing pre-surgical evaluation. Interestingly, 79% of respondents thought that “all patients undergoing pre-surgical evaluation should have an HD study”, but only 21% of responders indicated that HD EEG data was “crucial for the diagnosis and management of patients with epilepsy”. Though most institutions reported only performing 1-10 HD EEGs per month, 43% thought that HD EEG will become standard of care for selected patients with difficult medication-refractory epilepsy, and 43% thought that HD EEG will become standard of care for all patients with epilepsy. Most respondents used Netstation (EGI software) to review the HD EEG and the most common features employed were (1) scalp maps and (2) source localization with LAURA.

Conclusion: Overall, most respondents thought that HD EEG was an important, but not crucial, component of epilepsy pre-surgical evaluation and that it would eventually be used more broadly.

S20 Bilateral Needle EMG Changes in A Unilateral Hypoglossal Neuropathy From A Skull Based Cyst
Julia Whitlock, MD; Kennelly Kathleen, MD, PhD; Jay Van Gerpen, MD; Devon Robin, MD

Introduction: Unilateral hypoglossal neuropathy is rare but can be seen with various pathologies. Needle EMG and neuroimaging can be helpful in establishing the diagnosis and determining a potential etiology. Tongue innervation has traditionally been regarded as unilateral; however, crossed motor innervation to the tongue has been demonstrated via EMG.

Case Report: A 67 year old man presented with a 7 month history of dysarthria and “swollen tongue” after a prodrome of headaches, myalgias, neck pain, nausea, and vomiting. He was treated for strep throat but did not improve. At the time of neurologic evaluation, his exam was remarkable for flaccid dysarthria, left greater than right tongue weakness and atrophy but no other bulbar weakness. The rest of the neurologic exam was normal. Lingual needle EMG showed increased insertion activity with fibrillation potentials on the left and high amplitude, long duration motor unit potentials with moderately reduced recruitment bilaterally. Brain MRI w/wo contrast showed a peripherally enhancing 14 x 7 mm T2 extradural hypointense focus in the left premedullary cistern at the level of the sella and cephalad to the left hypoglossal canal, consistent with a skull based ganglion or synovial cyst.

Conclusion: Unilateral hypoglossal neuropathy from a skull based cyst is a rare cause of dysarthria. Needle EMG is helpful in confirming hypoglossal neuropathy. Crossed motor innervation to the tongue has previously been demonstrated via EMG. The electromyographer should be aware of crossed innervation to the tongue.

S21 Prolonged CMAP Duration In Early Acute Inflammatory Demyelinating Polyradiculoneuropathy
Priya Dhawan, MD; Brent Goodman, MD

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Conclusion: Unilateral hypoglossal neuropathy from a skull based cyst is a rare cause of dysarthria. Needle EMG is helpful in confirming hypoglossal neuropathy. Crossed motor innervation to the tongue has previously been demonstrated via EMG. The electromyographer should be aware of crossed innervation to the tongue.

S22
Mirror Movements(MM) as an Objective Marker of Upper Motor Neuron Involvement in Egyptian Patients with Subclinical Amyotrophic Lateral Sclerosis(ALS)
Mohamed H. Hassan, MD; Marwa Hassan, MD; Reinhard Dengler, MD

Introduction: Mirror movements (MM) is a useful method to detect early UMN involvement in ALS which is sometimes often difficult to assess in early stage of the disease or in patients in whom LAMN lesion is severe.

Methods: 30 patients with subclinical ALS had been examined for MM phenomena, needle EMG study and transcranial magnetic stimulation in both upper and lower limbs muscles at both sides

Results: MM had been recorded by using magnetic stimulation in the lower and upper limbs (27%,46%) and by using EMG from the lower and upper limbs (46%,46%).Correlations had been detected with the weakness.

Conclusion: MM can be detected by using electrophysiological procedures like the MEPs and the EMG . It is good specific test but less sensitive with high positive predictive value in diagnosing the ALS.

S23
Pathophysiologic Approach by Blink Reflex and EMG Studies in Essential Blepharospasm
Doo E. Kim, MD, PhD; Jeong H. Han, MD, PhD

Introduction: Blepharospasm (BS) is best categorized as focal dystonia, but the biochemical and neuroanatomical mechanisms are poorly understood. We performed this study in order to postulate the pathophysiologic mechanism of essential BS, using blink reflex test and EMG studies.

Methods: We studied 24 patients with essential BS and 51 normal adults. Blink reflex tests and EMG on orbicularis oculi muscle were performed in all patients. We evaluated our electrophysiological data, comparing with those obtained from other studies, in which bulbocavernous reflex, H-reflex, and T-reflex tests were done. We also compared our EMG data with those of hemifacial spasm and facial myokymia in other studies.

Results: 1. RL response latency of blink reflex test in 24 patients with essential BS was not changed, but R2 latency in the patient group was significantly shortened, comparing with those of normal control group. These results can be explained by overexcitability of the interneuron with polysynaptic pathway. Another supporting evidence is the fact that the latency of polysynaptic bulbocavernous reflex test in upper motor neuron lesion is shorter than normal control, although the latency of monosynaptic H-reflex and T-reflex test are not changed. 2. EMG on orbicularis oculi muscle in patients with essential BS showed spontaneous MUPs at irregular intervals at rest, while those in patients with hemifacial spasm and facial myokymia, known to be caused by overexcitability of facial nucleus, showed spontaneous MUPs at regular intervals.

Conclusion: It is suggested that the pathophysiologey of essential BS is overexcitability of interneuron due to disinhibition in inhibitory interneuron, and that EMG findings of essential BS are remarkably different from those of hemifacial spasm and facial myokymia.
**POSTER ABSTRACTS**

**S25**

**Status Epilepticus in Children: First Line Medication and Dosing**

Marina Gaínza Lein, MD; Ivan Sanchez Fernandez; Michele Jackson; Nicholas S. Abend, MD, FACNS; Ravindra Arya; J. Nicholas Brenton; Jessica L. Carpenter; Kevin Chapman, MD; William D. Guillard; Tracy Glauer; Joshua Goldstein; Howard P. Goodkin; Ashley Helseth; Kush Kapur; Mahamad Mikati; Katrina Pearso; Robert C. Tasker; Alexis Topjian; Mark Wainwright; Angus Wilfong; Koryyn Williams; Tobias Loddenkemper, MD, FACNS; And the Pediatric Status Epilepticus Research Group (pSERG)

Introduction: Objective: To describe first line medication dosing in refractory convulsive status epilepticus (RSE) and to compare actual doses with minimum recommended doses.

Methods: Multi-center prospective observational cohort study including pediatric patients with RSE, admitted from 2011-2015, who failed at least two anti-seizure medications. Multiple benzodiazepine (BZD) doses within the first 5 minutes of treatment initiation were counted as one dose.

Results: We included 184 patients with a median (p25-p75) age of 4.3 (1.4-9.7) years. RSE onset was in-hospital and pre-hospital in 122 and 62 patients, respectively. BZD was the first drug in 96% of patients. The most common BZD were lorazepam 106 (57.6%), diazepam 51 (27.7%) and midazolam 19 (10.3%). We compared the BZD doses to minimum recommended doses (Loddenkemper & Goodkin 2011). The median (p25-p75) administered dose vs recommended dose were: lorazepam 0.09 mg/kg (0.05-0.11) vs 0.05 mg/kg (p<0.0001), diazepam 0.42 mg/kg (0.31-0.55) vs 0.5 mg/kg (p=0.04), and midazolam 0.1 mg/kg (0.06-0.18) vs 0.2 mg/kg (p<0.001). A lower than the minimum recommended dose was given for lorazepam in 25 (24.3%) (p<0.000001), diazepam in 33 (66%) (p=0.03), and midazolam in 16 (88.9%) (p<0.000001). Lower than recommended doses were given in the in-hospital and pre-hospital settings to patients receiving lorazepam (in-hospital:24%; pre-hospital:28%), diazepam (in-hospital:80%; pre-hospital:57%), and midazolam (in-hospital:86%; pre-hospital:91%).

Conclusion: Approximately 75% of patients received appropriate doses of lorazepam. The majority of midazolam and diazepam doses were lower than minimum recommended for SE treatment. Lower than recommended BZD doses were present in both the pre-hospital and in-hospital settings. Further study is needed to determine whether BZD dose optimization during the early treatment stages has the potential to terminate seizures sooner.

**S28**

**Patients with Electrical Status Epilepticus in Sleep (ESES) Evaluated by New Method, Sleep Wakefulness Ratio**

Ahmet Tanitaniar, MD; Michele Jackson; Kush Kapur; Jack Connolly; Tobias Loddenkemper, MD, FACNS

Introduction: Intertical epileptiform activity in ESES has been quantified with spike wave index (SWI) and spike frequency (SF). We aim to describe a novel quantification tool, sleep wakefulness ratio (SWR) and evaluate its clinical application in patients with ESES treatment.

Methods: This is a retrospective descriptive study of consecutive patients with abnormal overnight EEG and suspected ESES from 2002-2014. SWI was defined as percentage of 1-second bins containing at least one spike. SF was defined as spike number. SWR was defined as sleep-SF divided by wakefulness-SF. Measures were calculated during a random 5 minute awake and sleep period. We compared patients with SWI <50% to patients with SWI ≥50%.

Results: 119 (67%) patients had SWI <50% (69 males; median age 9.1yrs.;p25-p75 6.2-12.1 yrs.). 57 (33%) patients had SWI ≥50% (42 males; median age 9.6yrs.;p25-p75 5.9-13.5 yrs.). Patients with seizures had more frequent spikes (sleep-SF:202; SWI:68%; awake-SF:12 vs sleep-SF:105; SWI:35%; awake-SF:4;p<0.001) and lower SWR (17.8 vs 21.6;p<0.05). Patients with abnormal MRI had more frequent spikes (sleep-SF:232; SWI:76%; awake-SF:18 vs sleep-SF:152; SWI:52%; awake-SF:6;p<0.001) and lower SWR (10.3 vs 21.5;p<0.001). Patients with cerebral palsy had more frequent spikes (sleep-SF:245; SWI:73%; awake-SF:26.5 vs sleep-SF:167;SWI:61%; awake-SF:8.5;p<0.001) and lower SWR (8.2 vs 19.2;p<0.001). SWR and SWI correlated during sleep (Spearman R=0.247;p<0.001) and wakefulness (R=0.878;p<0.001). SWR and SF correlated during sleep (R=0.235;p<0.001) and wakefulness (R=0.883;p<0.001).

Conclusion: ESES patients with seizures, cerebral palsy, and abnormal MRI demonstrated higher SF and SWI, and lower SWR. SWR has shown a good correlation with SWI and SF. SWR may assist in capturing the sleep potentiation component of spiking in ESES patients.

**S27**

**Hand Postures in Primary and Secondary Generalized Tonic-Clonic Seizures**

Jason Siegel, MD; Jake McKay, MD; William O. Tatum, DO, FACNS

Introduction: Classifying epilepsy is largely based on history, MRI, and ictal and interictal EEG. However, even video-EEG occasionally fails to provide a definitive diagnosis. Reliable semiologic lateralizing signs have been described, but hand and finger posturing has received little attention. We aim to describe hand postures during generalized convulsions in patients with genetic generalized epilepsy (GGE), localization-related epilepsy (LRE), and nonepileptic attacks (NEA).

Methods: We retrospectively analyzed 98 consecutive videos of generalized convulsions in 64 patients admitted for diagnostic video-EEG monitoring (VEM). Demographics and EEG were recorded, and hand postures were divided into fanning, fishing, index-finger pointing (IFP), clawing, and flaccid hand. Hand postures were compared between LRE, GGE, and NEA for each stage of the seizure (onset, tonic phase, and clonic phase). A p = < 0.05 was significant.

Results: The most common posture in LRE was IFP (96.0%), higher than GGE (56.5%, p = <0.001) and NEA (12.0%, p = <0.001). The most common posture in GGE was fanning (91.3%) which only occurred during the electrographic seizure onset and was more common than LRE onset (42.0%, p = <0.001). All GGE fanning evolved into either fisting (69.6%) or IFP (30.4%) during the tonic phase on EEG. The most common hand posture in NEA was the flaccid hand posture (56.0%), higher than GGE (4.3%, p = <0.001) and LRE (20.0%, p = 0.003). The clawing posture occurred exclusively in NEA without EEG changes (12.0%).

Conclusion: Distinct ictal hand and finger posturing is present in GGE, LRE, and NEA, based on VEM. We found a high prevalence of IFP in patients with LRE and flaccid and claw postures are highly suggestive of NEA. Hand postures also commonly changed over the course of a seizure. Hand posturing during seizures provides unique information and aids in the differential diagnosis and classification of epilepsy during VEM.

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S29
Chronic Ambulatory Human ECoG as a Tool to Assess Response to Antiepileptic Medications
Tara L. Crowder, PhD; Sharanya Arcot Desai, PhD; Rosana Esteller, PhD; Felice Sun, PhD; Martha J. Morrell, MD

Introduction: Responsive neurostimulation with the RNS® System (NeuroPace) provides a treatment option for adults with medically intractable partial onset seizures. Analysis of chronic ambulatory electrocorticographic (ECoG) activity recorded at the seizure focus may provide quantitative insights into the effects of antiepileptic medications (AEDs).

Methods: Subjects implanted with the RNS® Neurostimulator and NeuroPace leads that started clobazam during the open label periods of the RNS® System clinical trials and remained on a stable dose for ≥3 months were identified. Analysis was limited to subjects receiving responsive stimulation who had sufficient “scheduled ECoGs” (~90 secs. of ECoG activity stored at scheduled times of day). Spike rate, overall power and normalized power within delta, theta, alpha, beta, and gamma frequency bands were measured on each ECoG channel (up to 4 per ECoG). Clinical response to the initiation of the adjunctive AED was also assessed.

Results: Thirteen subjects initiated and maintained treatment with clobazam and met the ECoG data analysis criteria. Seizure onsets were neocortical (n=6), mesial temporal (n=6), MTL plus non-MTL (n=1). On average 199 scheduled ECoGs were analyzed per subject (total of 2589 ECoGs). There were significant reductions in spike rate and overall power with clobazam (p<0.001). These changes were evident in subjects that also had a clinical response to the AED.

Conclusion: Data obtained from a responsive neurostimulator provided a quantitative assessment of the electrocorticographic response to a new AED. Adjunctive clobazam treatment was associated with significant reductions in clinical seizure rates, spike rate and overall ECoG power. These results suggest that quantitative ECoG metrics such as spike rate and power may provide data that are useful to assess a patient’s clinical response to adjunctive pharmacological treatment.

S30
Changes in Electrographic Activity after Depth and Strip Lead Implantation
Thomas Tcheng, PhD; Felice Sun, PhD; Sharanya Arcot Desai, PhD; Martha J. Morrell, MD

Introduction: Electrocorticographic records (ECoGs) collected from chronically implanted NeuroPace® depth and/or cortical strip leads in 126 adults with epilepsy were analyzed to understand the ‘implant effect’ (change or reduction in seizures seen in the first few months after electrode implantation).

Methods: All subjects were participating in a double-blind, randomized, sham-stimulation controlled trial of a responsive neurostimulator (RNS® System, NeuroPace®, Inc.) as an adjunctive treatment for medically intractable partial onset seizures. The analysis included an average of 763 scheduled ECoGs per subject (n = 126), for a total of 96,162 ECoGs. Spike rate, overall power and normalized power were measured on each channel of the interictal ECoGs. Month-to-month differences were assessed for each patient ECoG channel, and group statistics were calculated by averaging the within-channel results.

Results: There were significant month-to-month changes in spike rate, overall power, and normalized power within frequency bands that were most pronounced in the first 3 to 5 months after implant. There was a significant change in over half (55%) of all the ECoG channels from the first to the second month (p<0.05, t-test), including 68% of the channels recorded from strip lead electrodes and 47% of the channels recorded from depth lead electrodes. After 5 months, the overall power became more stable, with significant month-to-month changes seen on average in less than 20% of the channels recorded from depth lead electrodes and less than 25% from strip lead electrodes. Similar patterns of changes were observed with normalized power within frequency bands as well as for spike rate.

Conclusion: ECoG data collected in the 3 to 5 months after implantation of depth or subdural electrodes are not stable and may not be representative of the chronic state.

S31
Effectiveness and Safety of Lacosamide as Adjunctive Therapy in Drug-Resistant Epilepsy: A Non-Interventional Study in Daily Clinical Practice
Hernan N. Lemus, MD; Luis C. Mayor, MD; Saul Reyes, MD; Claudia M. Guio, MD

Introduction: Drug resistant epilepsy occurs in 30-40% of patients despite adequate treatment and adherence to medications. Even though efficacy and safety of lacosamide was proved in placebo-controlled clinical trials for the treatment of partial-onset seizures, there is a lack of observational studies involving patients with drug resistant epilepsy in real world clinical practice settings.

Methods: Prospective non-interventional study in one hospital in Bogota Colombia. Adult patients with drug resistant epilepsy were screened between 2011 and 2014. Patients included in the study had medical appointments at six and twelve months, where seizure frequency and lacosamide related adverse events were assessed. Basal seizures (three months prior to the beginning of the study) were compared to the seizure frequency during the last three months of the study.

Results: A total of 79 adult patients with drug resistant epilepsy were included in the study. Seizure frequency after 12 month of continuous oral lacosamide was: >50% reduction (N=29), <50% reduction in seizure frequency or no change (N=18), worsening or increase in seizure (N=12), and seizure free (no seizures during the last three months, N=12). 23% of the patients (N=17) stopped lacosamide before the twelve months. After lacosamide introduction, 10% of the patients (N=8) had drug-related adverse effects with dizziness being the most common.

Conclusion: Lacosamide is a safe and effective drug when given as adjunctive therapy in drug resistant epilepsy. Lacosamide treatment reduced the number of seizure by more than half in patients with drug-resistant epilepsy. Lacosamide in this study was generally well tolerated and with a safe pharmacokinetic profile.

S32
Application of RNS in Refractory Epilepsy Originating from Insular
Hai Chen, MD, PhD; Patricia Dugan; Derek J. Chang, MD; Daniel Friedman

Introduction: Surgical resection treatment of insular cortex originated refractory epilepsy is usually difficult. RNS (responsive nerve stimulator) has been approved for treatment of resistant focal epilepsy. We report experience of RNS treatment of insular epilepsy in our center.

Methods: We identified patients who were implanted with RNS with electrodes targeting the insula between 4/2014 and 8/2015. We analyzed the preoperative
Clinical, image, video-EEG (vEEG) and intraoperative electrocorticography (ECoG) findings. Postoperative outcome and seizure frequency were examined in patients who had > 6 months of follow up.

Results: Seven patients had insular RNS implantation (Age 18-45). Ictal localization was inconclusive with either MRI or scalp vEEG tests. On intracranial EEG monitoring, five demonstrated an insular onset; one had broad hemispheric onset that included the insula. All patients underwent intraoperative ECog that demonstrated epileptiform discharges in the insular region. One patient developed worsening of hydrocephalus following RNS implantation which resolved after ventriculoperitoneal shunt placement. RNS stimulation was activated in 6 patients. Therapeutic settings range from 0.5-3 mA (current), 160 ms (pulse width), 100 ms (burst duration), 0.5-1.5 mc/cm2 (charge density) and 100-200 Hz (frequency). Among patients with > 6 month follow up (n = 4), one patient showed reduction of seizure frequency by 75% at the last visit (15 months). Two patients demonstrated an overall 50% reduction of seizures (9 and 13 months). One patient had significant seizure frequency reduction 4 months after implantation, which also coincided with addition of a new AED, and had been completely seizure free for 5 months at the time of last visit (13 months).

Conclusion: Insular RNS electrode placement in selected patients is relatively safe without permanent neurological deficit. Insular RNS may provide meaningful seizure relief at early follow up.

**S33**

**EEG Characteristics of EMU Patients with Psychogenic Nonepileptic Seizures with and Without Comorbid Epilepsy**

Samuel Lapalme-Remis, MD; Yeyao Joe Yu; Seyed Misattari; Richard McLachlan; Jorge G. Burneo; David Diosy

Introduction: Psychogenic non-epileptic seizures (PNES) are frequently comorbid in patients with epileptic seizures (ES). Patients with PNES without ES have a rate of interictal abnormalities nearly twice as high as normal controls. Our study characterized a cohort of PNES patients to assess the EEG differences between patients with PNES-only and PNES+ES.

Methods: Consecutive patients admitted to the Epilepsy Monitoring Unit (EMU) at London Health Sciences Centre (LHSC) between 2000 and 2008 were prospectively enrolled. Retrospective chart analysis included patients with 1) diagnosis of PNES or PNES+ES, 2) two or more captured habitual spells or single episode of psychogenic status epilepticus, 3) spells confirmed to have EEG and semiology consistent with PNES. Comorbid ES was diagnosed in cases featuring spells with semiology distinct from the PNES and either 1) EEG seizure or 2) combination of clinical, neuroimaging, and EEG evidence of ES. All LHSC neurology notes were reviewed to 2015 to ensure diagnosis remained accurate. Epileptiform EEG abnormality rates of the PNES and PNES + ES groups were compared.

Results: Of 217 patients included, 151 (69.6%) had PNES only, 48 (22.1%) had probable or definite comorbid ES, and 18 (8.3%) had possible comorbid ES. Patients with possible ES were excluded. Within the PNES-only group, 9.9% had epileptiform abnormalities; within the PNES + ES group, the figure was 83.3%.

Conclusion: In our EMU population, PNES-only patients had a proportion of epileptiform abnormalities higher than previously reported normal controls or PNES-only patients. This was driven by female PNES-only patients, who had a much higher rate of abnormal EEG than males. Although referral bias or prolonged EEG monitoring may play a role in these findings, they reinforce that in patients with clinical suspicion of PNES, interictal epileptiform EEG changes alone should not prompt a diagnosis of ES.

**S35**

**Use of Vigabatrin in Refractory Status Epilepticus — An Update**

R E. Ramsay, MD; Vivek Sabharwal; Mugilan Poongkunran, MD; Rachel Shumate, MD; Fawad A. Khan, MD; Megan Irlan

Introduction: Animal studies have shown very high glutamate levels in status epilepticus (SE). Vigabatrin (VGB) irreversibly inhibits GABA-T, increases GABA. Theoretically, this effect could reduce excitatory states and help control SE. We report the control rate of refractory status epilepticus (RSE) associated with adjunct VGB administration to other AEDs.

Methods: After IRB approval, we reviewed charts of patients admitted to the Neuro ICU for management of RSE between 2012 - 2015 at Ochsner Medical Center. This is a retrospective analysis of cases of RSE in which VGB was used adjunctively in the treatment of RSE.

Results: We identified 36 pts with RSE treated with VGB. Gender was 25 females & 11 males. Age ranged 3 mo to 80 years. Etiologies were cerebral anoxia (5), genetic/developmental (5), structural (4), cerebrovascular accident (5), toxic (5), autoimmune disease (2), sepsis (1), anti-epileptic drug non-compliance (3) and unclear (9). SE resolved with the addition of VGB in 26 (72%) patients. SE resolved in 3 pts before starting VGB but its addition prevented relapse when anesthetics were stopped. In 4 pts though suppressed, SE relapsed when anesthetics were reduced. 13 (36%) suffered in hospital mortality due to multiple organ failure (4), cardiac arrest (4) and withdrawal of care 24/35 received both propofol (PRO) and ketamine (KET) infusions and 11 received PRO only. AEDs used prior to initiation of VGB were levetiracetum 36, lacosamide 36, clobazam (CLZ) 15, depakote 16, & lorazepam 12. VGB was the last agent added prior to control in 24/29. The time from diagnosis of SE to VGB initiation averaged 7.18 days. The duration of VGB treatment before SE was controlled ranged from 0.19 to 28.77 days (average= 5.17 days).

Conclusion: The use of VGB has shown to be extremely important in achieving and maintaining control in the management of many patients with refractory and super-refractory SE.
the 439 patients admitted, 29 patients were excluded because their pre-admission seizure frequency could not be found in their medical record, and therefore 410 patients were included in the analysis. In 38 cases, precise pre-admission seizure frequency could not be found in the medical record but some indication of seizure frequency (such as “several seizures per month”) was reported. In these 38 cases, the seizure frequency was extrapolated.

Results: During their VEM, 149 patients had epileptic seizures, 189 had non-epileptic events and 101 patients had no seizure events. The average pre-admission seizure frequency was 0.72 events/day. The average event frequency in the EMU was 1.95 events/day. The Pearson correlation between pre-admission event frequency and EMU event frequency was 0.24.

Conclusion: Our study shows that there is no correlation between self-reported pre-admission seizure frequency and seizure frequency during video-EEG monitoring. This suggests that the decision to admit patients for video-EEG monitoring for seizure characterization should not be affected by patient’s self-reported seizure frequency.

S37 Regression Patterns and ESES Localization in CSWS and LKS
Anthony L. Fine, MD; Elaine Wirtrell; Lily C. Wong-Kisiel, MD; Eric T. Payne, MD, MPH; Katherine Nickels, MD

Introduction: We sought to assess whether developmental regression differences in children with continuous spike and wave in sleep (CSWS) and Landau-Kleffner syndrome (LKS) were associated with differences in their pattern of electrical status epilepticus in sleep (ESES).

Methods: We retrospectively identified children (<18 years) with CSWS or LKS and concomitant ESES (spike-wave index >50%), from our comprehensive electronic EEG report database (2007-2014). All children underwent overnight evaluation in our epilepsy monitoring unit and were evaluated by a pediatric epileptologist. Patterns of developmental regression were identified from clinical history and their EEG was carefully evaluated.

Results: Thirty-two children with CSWS (24 male; median age 7y) and 8 with LKS (5 male; median age 6y) were included. ESES was fronto-centrotemporal (77%) and bihemispheric (72%) in those with CSWS vs. centrottemporal (64%) and focal hemispheric (75%) in those with LKS. The mean spike-wave index was similar in CSWS (77%) vs. LKS (69%) (p=0.4). All children with CSWS developed regression in multiple developmental domains (34% global; 34% language; 22% motor). All children with LKS presented with language regression.

Conclusion: In this pediatric CSWS and LKS cohort, electro-spatial differences in ESES patterns were associated with, and plausibly explain, differences in clinical regression patterns.

S39 Focal Neurologic Deficits due to Hyponatremia in a Case Series of Pediatric Patients with Subdural Grids and Strips for Resective Epilepsy Surgery.
Andrea Lowden, MD; Deepa Sirs; Angelo Price; Rana Said, MD

Introduction: Multiple complications with subdural grid electrode implantation have been reported including infection, cerebral hematoma & edema. Hyponatremia alone or in combination with transient neurological deficit have not been described as a complication of this procedure in the pediatric or adult population.

Methods: We present a retrospective case series of pediatric patients who developed hyponatremia and transient focal neurologic deficits in the setting of subdural grid/strips implantation for epilepsy surgery.

Results: We present 4 children (3 F, 1 M), ages 11-17 yo with focal onset refractory epilepsy. The pathologic etiology, cortical dysplasia (2), tuberous sclerosis (1) & astrocytosis (1). 3 patients had subdural grids & strips (128 electrodes in all cases) over the left hemisphere & 1 over the right. 3/4 patients developed hyponatremia with serum Na levels between 123-130mEq/L. 2 patients developed acute aphasia & 1 a dense left arm monoplegia. The 4th patient did not develop a true hyponatremia however did have a sudden drop in serum Na from 144 to 135mEq/L over 3.5 hours. This child developed dysarthria & right arm weakness & was found to have cerebral edema upon prompt grid removal. All patients experienced the hyponatremia and the focal deficits between days 2-4 postoperatively. Despite the perioperative complications all patients proceeded to resective surgery of the seizure focus and are currently doing well from an epilepsy standpoint.

Conclusion: Typically hyponatremia results in diffuse cerebral manifestation such as altered mental status. In our patients the focal manifestations of low Na were likely due to compression of the underlying swollen cortex by the subdural electrode itself. This is the first pediatric case series reported to describe the focal manifestation of hyponatremia and the ultimate complete recovery in this setting.
**S40**

**Outcome in Children with Newly Diagnosed Partial Onset Epilepsy Treated with Carbamazepine**

**Gewalin Aungaroon, MD; Katherine Holland, MD, PhD; Shannon Standridge, MD**

**Introduction:** This study objective is to evaluate outcome in children with newly diagnosed partial onset epilepsy treated with carbamazepine (CBZ).

**Methods:** A retrospective medical records review and telephone questionnaire was undertaken on a total of 100 subjects.

**Results:** Long-term follow up was obtained on 79 children with a mean duration of 7.1 years from CBZ initiation. A total of 66 subjects achieved 2-year seizure remission and 7 of these subjects had seizure recurrence. Drug-resistant epilepsy (DRE) was diagnosed in 7 subjects and 5 subjects required epilepsy surgery. The mean duration from seizure onset to the diagnosis of DRE was 3.0 years (SD ± 1.8), and the mean duration from the diagnosis of DRE to epilepsy surgery was 2.0 years (SD ± 1.4). The mean duration from seizure onset to epilepsy surgery was 5.3 years (SD ± 2.1). The logistic regression analysis showed no association between sex, age of seizure onset, age at AED initiation, epilepsy etiologies and DRE.

**Conclusions:** More than 70% of children with newly diagnosed partial onset epilepsy achieved 2-year seizure remission with CBZ monotherapy and the majority maintained seizure remission overtime. However, almost 10% of this population met the criteria for DRE and the majority of this group required epilepsy surgery. The duration from seizure onset to epilepsy surgery is an important potential area of improvement in DRE patient care.

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**S41**

**Hemodynamic Changes Associating with Intercital Epileptiform Activities using Simultaneous video Electro-encephalography (EEG)/Near Infrared Spectroscopy (NIRS) in patient Self Control Study**

**Kumar Sanugowadara, MD; Priya Manrod, MD; Kurt Hecox, MD, PhD; Michael Schwabe, MD; Michael Meyer; Jenna Prigge; Harry T. Whelan, MD**

**Introduction:** NIRS is a non-invasive method for continuous measuring of cerebral oxygenation (S(o)2), which has been used to monitor cerebral oxygenation and blood flow in various conditions. Different types of hemodynamic changes during seizures have been described: cerebral blood flow and volume may increase or decrease in relation to hyper or hypometabolism.

**Methods:** This study included 4 patients age from 5 to 17 years with medically refractory epilepsy - partial complex seizures, undergoing presurgical evaluation. Video electroencephalography recordings and data from a near infrared spectroscopy cerebral/somatic oximeter were gathered and related to electrographic seizure onset and offset as determined by paediatric epileptologist.

**Results:** Two patients had hypometabolic area, which confirmed by PET scan, showed clear low hemodynamic changes (20% decreases) in NIRS compared to the contra lateral hemisphere. Two patients had baseline hemispheric hyper-perfusion on NIRS, which were correlated with baseline more epileptiform activity compared to contralateral hemisphere. All four patients showed haemodynamic changes associated with epileptiform activities. The increased blood flow clearly coincided with epileptiform activity and continued to increase as the epileptiform activity built up.

**Conclusion:** This study showed clear hemodynamic response associated with interictal epileptiform discharges and ictal events. The hemodynamic changes observed in NIRS study could potentially provide further insight into the pathophysiology of the evolution of seizures. NIRS could become useful adjunct to EEG. **ACKNOWLEDGEMENTS:** This study was supported by the Bleser Endowed Chair in Neurology (to Dr. Whelan), the Baumann Research Endowment (to Dr. Whelan), and a Covidien NIRS Grant (to Dr. Whelan).

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**S42**

**Could Lateralized Periodic Discharges in a Child with Human Herpes Virus 6 be an Indicator of Neuro-invasion in Febrile Status Epilepticus?**

**Muhammad S. Zahar, MD; Zahra M. Haghighat, MD; Arun Swaminathan; Robert Baumann, MD; Gulam Khan, MD**

**Introduction:** Human Herpes Virus 6 (HHV-6) Viremia is the most common cause of febrile status epilepticus (FSE). There is a speculation about neuro-invasion from HHV- however no definite conclusion can be made from prior studies. We reported lateralized periodic discharges (LPDs) in a child with FSE in the setting of systemic HHV-6; hypothesizing the direct role of HHV-6, as LPDs usually suggest structural abnormalities or direct neuro-invasion.

**Case Report:** A previously healthy 3-year-old boy was admitted for FSE. Clinical seizures were stopped with Phosphenoin. EEG showed electrographic seizures and LPDs, which were treated with intravenous Lacosamide. MRI brain and spinal fluid analysis were normal. Serum showed HHV-6 via polymerase chain reaction. EEG continued to improve over 24 hours with resolution of LPDs and no recurrence of seizures.

**Conclusion:** FSE is a severe form of complex febrile seizures with an increased risk of subsequent temporal lobe epilepsy. Although HHV-6 was only found in the serum of this child and not in the spinal fluid, the presence of LPDs in setting of FSE could suggest a direct effect of the virus on the brain rather than an effect of an inflammatory process and high fever caused by the viral infection.
**POSTER ABSTRACTS**

frequency responses and lateral and anterior sources for low frequency ASSR). The modulation frequency was obtained a significant effect in ASSR.

Conclusion: The multichannel recordings and the use of BMA models are suitable to obtain the current primary distribution of ASSR and for estimations of brain sources of the auditory response. Throughout the methodology presented here we could pinpoints the main sources of ASSR at the superior and medial cortical temporal lobes, with some extra temporal areas lobes. The carrier and modulation frequencies of AMT have a significant effect in location and in the magnitude of estimated sources.

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**S44**

**The Effect of Anti-Epileptic Drugs on Visual Evoked Potential in Patients with Generalized Tonic-Clonic Seizures: A Prospective Case-Controlled Study**

Aysha A. Alshareef, MD

Introduction: The main purpose of the present study was to determine if anti-epileptic drugs induce any abnormal changes in the visual evoked potential patterns.

Methods: This prospective experimental study was done at the Neurology Department at King Abdulaziz University Hospital, Jeddah, Saudi Arabia (between January 2013 and December 2014). in our study we made A comparison between visual evoked potential waves in the control group and case group. The case group consisted of the patients taking anti-epileptic drugs, and was further divided into 3 groups according to number of anti-epileptic drugs taken. Using the Visual Evoked Potentials (VEPs), control and case subjects were compared regarding values of: Latency N75, Latency P100, and Amplitude P100.

Results: The study showed a statistically significant variation between control and the group of patients taking double drug therapy, as well as the group of patients taking 3 or more drugs therapy, in terms of Latency P100. The analysis of variances (ANOVA) test, used to compare between the mean scores of the control group and the group of patients on mono-therapy, discovered that there is a statistically significant variation between the mean scores of Latency N75 and the mean scores of Amplitude P100 in relation to age.

Conclusion: This study showed that anti-epileptic drugs do in fact induce abnormalities in the Visual Evoked Potentials patterns. These abnormalities occur for certain age groups and gender, in relation to the number of anti-epileptic drugs taken by the patients. More studies are required to evaluate other predisposing factors in relation to epilepsy types and duration.

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**S45**

**PESST and H Reflex in Patients with UI Before Lumbosacra Trauma: Comparative Study at Three Months.**

Teresa M. Montes de Oca Daminquez, MD; Juan Manuel Rojas de Dios, MD; Gladys Maya Morales, Tec; Olga Gonzalez Perez, Tec

Introduction: Urinary incontinence (UI) is defined as a condition in which involuntary loss of urine is a social or hygienic problem and is objectively demonstrable. Spinal cord lesions can alter sympathetic and parasympathetic tone resulting in urinary incontinence. If the sacral cord is involved, like S2-S5 nerves can causes bladder dysfunction and urinary incontinence or retention can be with an unfavourable prognostic. Objectives: Analysis of H Reflex and PESST in patients with urinary incontinence posttraumatic of column lumbosacra in initial stages and study comparative at three months.

Methods: Study of H Reflex and PESST with analysis of central conduction in 27 patients, between 24-45 years of age with urinary incontinence transitory posttraumatic of column lumbosacra in initial stages (21 days of trauma).

Results: H Reflex was absent bilateral in 19 patients (70.37%) and slowed down in the rest. PESST and TCC was absent in 22 patients (81.48%). The comparative study in three months, demonstrates H Reflex absent in 14 patients (51.85%) and the PESST in 16 patients (59.25%).

Conclusion: The alterations in initial stages of this pathology could evaluate the susceptibility of urodynamic in the incontinence urinary in the course of affections moduler. The PESST and H Reflex will be effective in diagnostic of IC.

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**S46**

**Scalp-recorded Somatosensory Evoked Potentials in Swine are Reliable for Studying Cerebral Ischemic Injury and Recovery Following Cardiac Arrest**

Joshua Margos, MS, CNIM; Nicole L. Werner, MD, MS; Nick Chulski; William C. Stacey, MD, PhD; Alvaro Rojas Pena; Robert Neumar

Introduction: Swine have been employed as a model for studying cardiac arrest and resuscitation. Additionally, various techniques have been tested on swine to study traumatic brain and spinal cord injury. Here, we use somatosensory evoked potentials (SSEPs) to develop a swine model for monitoring neurologic function following cardiac arrest (CA).

Methods: SSEPs were recorded from 15 Yorkshire pigs following supramaximal stimulation of the right forelimb using standard stimulation parameters. Subdermal needle electrodes were placed for stimulation and recording at the distal forelimb and brachial plexus, respectively. Corkscrew electrodes were placed on the scalp and behind the neck to record cortical and subcortical potentials. Inhalational anesthetics were discontinued after induction and total intravenous anesthesia was used to obtain optimal SSEP recordings. CA was induced at least once in each pig, with a second CA induced if return of spontaneous circulation (ROSC) was achieved and accompanied by a return of the cortical signal to >50% of baseline.

Results: Cortical and subcortical SSEPs were successfully recorded in all pigs. Cortical responses had an average latency and amplitude of 19.94 ± 1.28msec and 1.35 ± 0.56uV, respectively, and were recorded from the lateral aspect of the skull contralateral to the site of stimulation. The cortical response was completely lost within three minutes of CA though subcortical and peripheral responses varied in rate and degree of loss. ROSC was achieved in 6/18 studies. Cortical responses returned to at least 39% of baseline amplitude following ROSC though the rate and degree of return varied.

Conclusion: SSEPs appear to be a reliable biomarker of neurological function in this swine model of CA. This study demonstrates the utility of SSEPs in swine as a reliable model for studying neurological damage and recovery following CA.

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**S47**

**Concordance of Multifocal PET in Patients with Epilepsy**

Zeke Campbell; Leonardo Bonilha, MD, PhD

Introduction: Interictal positron emission tomography (PET) has been shown to be a valuable clinical tool in the presurgical evaluation of patients with refractory epilepsy. Several studies have examined the concordance of PET with other localizing
Axonal Excitability Increased by a Train of Subthreshold Stimuli: The Role of Stimulus Duration

Paulo A. Kimaid, MD and PhD; Marcondes Franca, MD, PhD

Introduction: An under-recognized phenomenon of increased axonal membrane excitability of the peripheral nerves caused by a train of subthreshold stimuli was recently described as the explanation for some peculiar findings observed during intraoperative monitoring of transcranial motor evoked potential (TCMEEP) recorded from cranial nerve-innervated muscles (CNIM). We investigated how stimulus duration influenced this phenomenon in an attempt to identify a protocol that avoids peripheral stimulation during the intraoperative monitoring of TCMEEP recorded from CNIM.

Methods: A constant-current stimulation with trains of subthreshold stimuli was applied to the median nerve at the wrist of 5 normal volunteers and 10 patients. The stimulation protocols for each group were: 1) Duration 500µs, Interstimulus interval (ISI) 2ms; 2) Duration 500µs, ISI 4ms; 3) Duration 50µs, ISI 2ms; 4) Duration 50µs, ISI 4ms. We recorded the compound muscle action potential (CMAP) from the abductor pollicis brevis using surface electrodes in normal volunteers and needle electrodes in patients who underwent lower spinal surgery under total intravenous anesthesia without neuromuscular blockade.

Results: A single near-threshold stimulus didn’t generate CMAP in any subject in the 4 studied groups. When the number of near-threshold stimuli was increased from 2 to 10, a CMAP of varying latency and amplitude was recorded in the normal volunteers and patients from groups 1 and 2, but not from the groups 3 and 4.

Conclusion: We hypothesize that the capacitance of the subcutaneous tissue is the cause for the observed phenomenon. Our results prove that reducing stimulus duration, we reduce the capacitance and avoid the increase of peripheral nerve excitability. In our opinion, to avoid the peripheral stimulation of cranial nerves during TCMEEP one should use short duration stimuli such as that used for upper and lower limb TCMEEP (between 50 and 80µs).

Clinical Utility and Yield of Pharmacologic Provocative Testing During “High” and “Low” Risk Spinal Endovascular Procedures

Joshua Mergos, MS, CNIM; Vikas Singh, MBBS, MHSA; Daniela N. Minecan, MD; Aditya Pandey

Introduction: Neuroendovascular embolization is increasingly performed for treatment of arteriovenous malformations or fistulae (AVM/AVF) of the spinal cord and to treat hyper-vascular metastatic lesions to the spine. Pharmacological provocative testing (PPT) using continuous intraoperative neurophysiological monitoring (IONM) in patients under general anesthesia for the treatment of spinal AVM/AVF has been reported in the literature, but has not included its application to spinal conditions.

Methods: We conducted a retrospective chart review of all patients who had IONM-based PPT performed during spinal embolization for AVM/AVF or pre-surgical or palliative embolization of hyper-vascular lesions from December 2009 to May 2014. All procedures were performed under general anesthesia with transcranial electric motor evoked potentials (Tce-MEPs) and somatosensory evoked potentials (SSSEPs). PPT was performed using injection of Brevital and Lidocaine in a sequential manner through a micro-catheter superselectively placed in the feeding vessel. Standard SSEP alarm thresholds were used to consider a PPT as positive. Tce-MEP changes were considered positive if compound muscle action potential amplitudes in any significant limb decreased by more than 50%. Embolization was altered or foregone in the case of positive PPT.

Results: 37 procedures utilizing PPT were performed on 32 patients. 18 procedures were performed on patients as a pre-operative measure for surgical resection of metastatic spinal lesions, 15 for AVM/AVF embolization, and 4 as palliative treatment. 6 positive PPT results resulted in modification of surgical maneuvers. No new focal neurological deficits were reported on postoperative neurological examination and immediate follow-up clinic visit.

Conclusion: IONM-based PPT may prevent long term morbidity in patients with a variety of spinal vascular lesions not limited to AVM/AVF.

Preoperative Value of Intraoperative Auditory Brainstem Response Audiometry for Hearing Loss in Acoustic Neuroma Resection via Middle Fossa Craniotomy

Joshua Lucas, MD; Andres A. Gonzalez, MD, MMM, FACNS; Rick A. Friedman, MD, PhD; Steve L. Giannotta, MD

Introduction: Auditory brainstem response (ABR) audiometry is a valuable tool in monitoring functional integrity of the cochlear nerve during acoustic neuroma resections. We examined the predictive value of initial (prior to incision) and intraoperative ABR data in postoperative hearing preservation in resections performed via middle fossa craniotomy.

Methods: Twenty-five consecutive patients who underwent a standard infratemporal approach to the petrous apex (middle fossa craniotomy) for surgical resection of an acoustic neuroma were analyzed. Intraoperative ABR parameters, including contralateral controls, were measured prior to incision and throughout the procedure. Postoperative hearing was assessed via audiogram data and clinical exam upon routine follow-up. Receiver-operator curve (ROC) analysis was performed to determine the most appropriate predictive criteria for each parameter.
POSTER ABSTRACTS

SS1
Role of MER in DBS Surgery in an Era of High Resolution MRI
Anh Thu Tran, MD; Parastou Shilian, DO; Andres A. Gonzalez, MD, MMM, FACNS; Daniel M. Togasaki

Introduction: Deep brain stimulation (DBS) has been shown to be efficacious in the treatment of Parkinson’s disease (PD). Intraoperative testing with microelectrode recording (MER) and macrostimulation with neurologic exam play an essential role in target localization. Advancement in neuroimaging has brought the use of intraoperative testing into question. At our institution awake MER and neurologic testing are routinely performed. We evaluate the role of such testing in an age of high resolution imaging.

Methods: A retrospective chart review of DBS surgeries after 1/10/2014 was performed. Data collection included patient demographics and intraoperative parameters such as repositioning based on MER and/or macrostimulation. First group included implants in which imaging adequately targeted STN as confirmed by good quality first-pass MER (MER-image congruent). Second group included implants that required repositioning based on poorer quality first-pass MER (MER-image incongruent). We used intraoperative macrostimulation to assess the correct positioning of the electrode. Those requiring repositioning were identified for each group.

Results: In total, 54 STN DBS implants were performed. 78% (42/54) of implants comprised the MER-image congruent group. 22% (12/54) comprised the MER-image incongruent group. 7% (3/42) of congruent and 8% (1/12) of incongruent groups required repositioning based on macrostimulation. There was no statistically significant difference between the two groups.

Conclusion: This study demonstrates that MER continues to play an essential role in DBS surgery. MER localization is essential where imaging and MER were incongruent. Macrostimulation is useful in both groups as it assists in both localization and functional measures of DBS. Lack of a statistically significant difference between the groups suggest that DBS implants requiring repositioning based on MER fared just as well as first-pass MER.

SS2
Novel Intraoperative Neurophysiologic Monitoring (IONM) Techniques during Thoracic Endovascular Aortic Repair (TEVAR) to Rapidly Assess Central Spinal Cord versus Peripheral Limb Ischemia
Leslie H. Lee, MD, FACNS, FAAN; Jason T. Lee, MD; Eric Jones, Bachelor of Science; S Charles Cho; Viet Nguyen, MD; Scheharazade Le; Jaime R. Lopez, MD, FACNS

Introduction: Traditional IONM techniques including somatosensory evoked potentials (SSEPs) and/or transcranial motor evoked potentials (tcMEPs) may not adequately distinguish between evolving central spinal cord versus peripheral limb ischemia in TEVAR. We present a novel multimodality IONM approach to facilitate rapid assessment and prompt differentiation of contributing factors to suspected ischemia.

Methods: We present seven TEVAR cases for which a unique multimodality IONM protocol was employed that incorporates pudendal/perianal SSEPs and anal sphincter tcMEPs, along with upper/lower extremity SSEPs and tcMEPs, as well as EEG.

Results: In all seven cases reliable bilateral anal sphincter tcMEPs were obtained, and in four cases reproducible pudendal/perianal SSEPs were also elicited. In three cases, sudden loss of leg SSEPs and/or tcMEPs was observed during selective vessel catheterization. In contrast, anal sphincter tcMEPs and pudendal/perianal SSEPs remained unchanged. This pattern appeared consistent with transient peripheral limb ischemia rather than spinal cord ischemia (SCI). Following stent deployment, leg SSEPs and tcMEPs returned to baseline with no further changes. In all cases anal sphincter tcMEPs remained stable throughout TEVAR. All patients awoke postoperatively with no new neurologic deficits or symptoms to suggest recent or delayed onset SCI.

Conclusion: Spinal cord and limb ischemia are potential complications of TEVAR. The loss of extremity SSEPs/tcMEPs with preservation of pudendal/perianal SSEPs and anal sphincter tcMEPs suggests peripheral limb ischemia rather than SCI. We advocate the routine monitoring of anal sphincter tcMEPs and pudendal/perianal SSEPs in conjunction with traditional IONM techniques, to allow for rapid differentiation between central and peripheral causes of evolving ischemia.

SS3
Neuronomonitoring Changes and Neurologic Outcome by Approach and Number of Levels in Extradural Cervical Spine Surgery
Jeffrey Cohen, MD, PhD; Anthony Sestokas, PhD; Eric Tesdahl; Joseph Jares, MD; Raymond Harrow; Bradley Wallace, MD, PhD; Rachel Roberts, BS CNIM; Jessica Cherevko, CNIM, R.Ep.T.; Samuel Weinstein, MD, MBA

Introduction: Traditional IONM techniques including somatosensory evoked potentials (SSEPs) and/or transcranial motor evoked potentials (tcMEPs) may not adequately distinguish between evolving central spinal cord versus peripheral limb ischemia in TEVAR. We present a novel multimodality IONM approach to facilitate rapid assessment and prompt differentiation of contributing factors to suspected ischemia.

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Conclusion: Spinal cord and limb ischemia are potential complications of TEVAR. The loss of extremity SSEPs/tcMEPs with preservation of pudendal/perianal SSEPs and anal sphincter tcMEPs suggests peripheral limb ischemia rather than SCI. We advocate the routine monitoring of anal sphincter tcMEPs and pudendal/perianal SSEPs in conjunction with traditional IONM techniques, to allow for rapid differentiation between central and peripheral causes of evolving ischemia.

Results: Hearing was completely preserved in 16 of 25 patients, partially preserved in 4 patients, and lost in 5 patients. ROC analysis determined two that reached statistical significance. An initial ABR wave 5 amplitude <0.295 was 80% sensitive and 75% specific for complete postoperative hearing loss (p = 0.0383). Also, a decrease in the ABR wave 5 amplitude by greater than 55.5% was 80% sensitive and 90% specific for complete postoperative hearing loss (p = 0.007). The initial ABR wave 1 amplitude, initial ABR wave 1 latency, initial ABR wave 5 latency, and difference in initial ABR wave 5 latency and the contralateral ear were not significantly correlated to postoperative hearing outcome.

Conclusion: Intraoperative ABR data can be used to predict postoperative hearing loss. Initial ABR wave 5 amplitude and percentage change in ABR wave 5 amplitude from surgery start to surgery end were significant predictors of complete postoperative hearing loss.

SS5
Neuronomonitoring Changes and Neurologic Outcome by Approach and Number of Levels in Extradural Cervical Spine Surgery

Methods: NM changes and NDs were retrospectively reviewed for 20,861 consecutive cervical spine procedures (CSPs) from the SpecialtyCare Multi-Institutional IONM database between May, 2013 and August, 2015. Procedures were categorized according to approach and number of levels. NM changes and NDs in the immediate post-op period were recorded. Incidence rate differences were analyzed using binary logistic regression and post-hoc Tukey tests.

Results: Overall incidence of NM changes and NDs in CSPs was 13.0% and 0.77% respectively. NM changes were more common in combined anterior/posterior
Predictive Factors Determining Poor Baseline Lower-Extremity Somatosensory Evoked Potentials During Surgery.

Jonathan Chen; Andres A. Gonzalez, MD, MMM, FACNS; Parastou Shilian, DO; Anh Thu Tran, MD

Introduction: Intraoperative neuromonitoring (IONM) utilizes neurophysiological techniques for real-time monitoring during surgery to detect and warn of potentially reversible neurological injuries. By interpreting changes in SSEP signals that fall below threshold values, the neurologist can give live feedback to the surgeon. However, the ability to predict changes is reliant on acquiring adequate baseline signals for comparison. Therefore, elucidating predictive and potentially modifiable patient characteristics can assist in providing more robust monitoring.

Methods: Single-center, retrospective chart review of 632 patients undergoing spine or cranial surgeries between 2010 and 2011 for the presence of diabetes mellitus type 2 (DM2), hypertension, hyperlipidemia, height, weight, gender, smoking, pre-existing neurologic conditions, degree of lower extremity edema, neurologic exam findings in motor, sensory and reflexes. A multivariable logistic regression analysis was performed looking at the ability of these factors to predict poor baseline signals in lower extremity (LE) SSEPs.

Results: DM2, being a male, certain neurologic conditions, LE edema, motor, sensory and reflex abnormalities each individually is associated with poor baseline LE-SSEPs. Furthermore, in patients with DM2, deficits in sensory exam alone predicts poor baselines. In patients without known history of DM2, the combination of increased weight, lower extremity edema, and abnormalities in motor, sensory and reflex exams predicts poor baselines.

Conclusion: Having adequate baselines is paramount for IONM. DM2 and lower extremity edema are potentially modifiable risk factors strongly associated with absent lower extremity evoked responses. Further research (e.g., proximal stimulation at the popliteal fossa) should be sought out in anticipation of these conditions in order to improve our capacity to protect the nervous system during surgery.

Operative Motor Mapping

Functional Outcomes in Neuro-Oncology Surgery Cases Utilizing Intra-Operative Motor Mapping

Michelle M. Mora, DO; Shawn Hervey-Jemper, MD; Daniel Orringer, MD; Daniela N. Minecan, MD

Introduction: Several factors influence functional outcomes during neuro-oncology surgical cases utilizing intraoperative mapping (tumor location, type of anesthesia used). The state of the patient during the mapping procedure is less well known.

Methods: Intra-op neuromonitoring database from July 2014 - July 2015 was searched for all neuro-oncology surgical cases requesting motor mapping. These included awake and asleep cases, divided as such. We retrospectively reviewed corresponding op reports, anesthesia record, intra-op mapping report, pre-op H&P, and post-op progress reports.

Results: 45 neuro-oncology surgical cases utilizing intra-operative mapping were identified. 23/45 utilized motor-only cortical/subcortical mapping — 15 cases with the patient awake and 8 cases asleep. 6 awake patients (40%) had non-focal pre-surgical exam, 9 patients (60%) had focal/lateralized findings. Follow-up 4-6 weeks later, 3 patients (20%) had functional deficits, one (7%) motor (left upper extremity weakness worsened from baseline). The other 2 cases with functional deficits were non-motor (expressive aphasia, left homonymous hemianopia). Of 3 cases with post-operative functional deficits, positive responses during motor mapping were obtained in 2 cases (left upper extremity weakness worsened from baseline, expressive aphasia). 3 asleep patients (37%) had non-focal pre-surgical exam while the other 5 (63%) had focal/lateralized findings. Functional status at 4-6 weeks follow-up revealed 6 patients (75%) had an unchanged exam, and 2 patients (25%) had new focal findings. Of these 2 cases, no positive responses were identified during asleep motor mapping.

Conclusion: Awake cortical motor mapping during tumor resection in surgical neuro-oncology cases, while more demanding for both the patient and the surgeon, correlated with better post-surgical functional outcomes as compared with asleep cortical motor mapping.

Intraoperative Neurophysiologic Changes Observed with the Lateral Interhemispheric Surgical Approach to Midline Lesions

Leslie H. Lee, MD, FACNS, FAAN; Omar Choudhri, MD; Steven Chang, MD; Jaime R. Lopez, MD, FACNS

Introduction: The lateral interhemispheric approach is less routinely utilized in neurosurgical cases, but is an ideal technique for resection of ipsilateral midline lesions as well as contralateral cingulate or callosal lesions. At our institution multimodality intraoperative neurophysiologic monitoring (IONM) is standard for intracranial surgeries that may place neural structures or pathways at risk.

Methods: We present two cases in which the lateral interhemispheric approach was used for the resection of an ipsilateral tumor (one case with a left parafalcine meningioma, the second case with a metastatic melanoma involving the corpus callosum and medial left cerebral hemisphere). Multimodality IONM was employed in both cases, including transcranial motor evoked potentials (tMEPs), somatosensory evoked potentials (SSEPs), and electroencephalography.
Results: Following complete surgical resection of the midline lesion, the unexpected ipsilateral loss of SSEPs (in one case), and SSEPs along with tcMEPs (in the second case) was observed without recovery by the end of case, which was suggestive of potential compromise of the somatosensory and corticospinal pathways contralateral to the lesional hemisphere. Both patients awoke with no new clinical deficits that correlated with these IONM critical changes observed at the end of resection.

Conclusion: We hypothesize that these IONM changes ipsilateral to the lesional hemisphere reflect the unique positioning of the patient’s head for the lateral interhemispheric approach, with gravitational effects dynamically shifting the position of the brain relative to the static placement of scalp electrodes used for IONM. This pattern of unanticipated loss of evoked potentials is important to recognize, as rapid assessments are continually made to determine the etiology and potential reversibility of observed neurophysiologic changes.

S59
Comparison Between Cortical Subdural Grids vs Stereo-Encephalography Guided Surgical Intervention in Pediatric Population; the Colombian Experience.
Walter Gonzalez, MD; Cesar A. Buitrago, MD; Milton D. Herrera, MSc; Eugenia Espinosa, MD

Introduction: Subdural electrode (grids/strips) placement is widely used in Colombia as an epilepsy surgical tool in selected refractory epilepsy cases; even though, our center moved to stereo-electroencephalographic (SEEG) implantation as a standard technique since more than 3 years ago. Recently many centers in America now are implementing Stereoelectroencephalography intervention instead of classical subdural electrodes, little is known about the outcome of this procedure in South America.

Methods: A retrospective study of 29 patients admitted to our epilepsy surgery center with confirmed diagnosis of pharmacoresistant epilepsy, who underwent preoperative intracranial monitoring with SEEG or subdural electrodes where compare in terms of outcome, medical and surgical complications.

Results: Mean age of 11,4 years, 11 (38%) were female, mean duration of epilepsy before surgery was 8,2 years, 65% with a structural lesion preoperatory. 14 (48%) underwent subdural monitoring and 52% to SEEG. The mean time of follow up was 1,2 years. After surgery there were excellent results (Engel I) in 7 (46%) patients with SEEG and 5 (35.7%) with subdural grids, significant difference was not found in postoperative results (P = 0,5).

Conclusion: Our study showed that the use of intracranial monitoring either subdural electrodes or SEEG is safe and have good general outcome as has been reported by the literature. We find there is a better outcome with SEEG than with subdural electrodes, but without significant difference, probably by the size of the sample. Few complications were found in both comparative groups, although patients within SEEG group looks to have lower cognitive decline. We need more studies in our population with larger period of following after epilepsy surgery.

POSTER ABSTRACTS

S58
Learning Curve of New Intraoperative Neuromonitoring Service
Osama Shams

Introduction: Intraoperative neuromonitoring (IONM) is becoming a standard of care in progressively increasing number of surgeries. It is not always feasible to have well trained IONM team for the surgeons performing these types of surgeries. In that case, healthcare facilities may be left with a single option of investing in its clinical neurophysiology service to provide IONM.

Methods: This a descriptive study for the learning curve and challenges faced while establishing a new IONM service in our center where access to formal training programs in the field is limited.

Results: Sharing anesthetics effects on recorded responses with anesthesia team paid off gradually as increasing rate for using total intravenous anesthesia (TIVA). Ketamine was also introduced to anesthesia protocol with reduced tendency to use bolus dose of anesthetics. Surgical team gradually developed the routine of merging IONM into their procedures. They gradually paid more attention to IONM alerts and recognized how to best use different modalities like screw stimulation, trans cranial motor stimulation and root stimulation. IONM team arranged with surgical team to start by monitoring simple cases and gradually increase complexity of monitored cases. Frequency and type of technical troubleshooting were major issues affecting monitoring process. Mixing cables, 60Hz noise, and stimulation failure gradually vanished over time. Amplifier saturation remained an issue due to technical limitation in our IONM system. Setup time from the first needle to baselines gradually decreased with using better team dynamics and technical tips. Setup in parallel with anesthesia after intubation significantly improve set up time.

Conclusion: New IONM services can be established when collaboration and shared vision among the managing team is available even with poor access to local formal training programs.
S60
Defining Networks During Generalized Seizures: A Combined MEG and fMRI Approach
Jeffrey Tenney, MD, PhD; William Agler, BS; Claudio Toro-Serey; Darren Kadis

Introduction: The aim was to identify childhood absence seizure network activity using fMRI-informed MEG effective connectivity analysis.

Methods: Magnetoencephalography (MEG) and combined EEG-functional magnetic resonance imaging (EEG-fMRI) were recorded in 7 subjects with untreated childhood absence seizures. EEG-fMRI data were pre-processed and EEG was used to identify timing of absence seizures during fMRI. An event related independent component analysis (elICA) method was used to define fMRI activity correlating with the seizures. These regions were then parcellated, to express the network in terms of functional nodes. These nodes were used as virtual sensor locations for beamformer analyses of absence seizures recorded using MEG in the same 7 subjects (34 seizures). After extracting source waveforms, the effective connectivity was estimated using a phase slope index (PSI) metric.

Results: elICA of fMRI data identified regions similar to those previously reported (thalamus, frontoparietal, precuneus, biparietal). Effective connectivity analysis using PSI at lower frequencies (delta, theta, and alpha) showed connections within and between parietal and precuneus regions, and the thalamus. At higher frequencies (beta and gamma) connectivity tended to be within and between frontal regions. Some inter-subject variability was also noted.

Conclusion: fMRI-informed MEG analysis can be used to identify patterns of brain connectivity during generalized seizures, such as childhood absence seizures. In this way, maximal spatial and temporal information during these seizures can be extracted. We are hopeful that these types of connectivity patterns could be used in the future to “group” patients in a way that helps to predict their phenotype, such as treatment responsiveness.

S61
LSUHSC-Shreveport Neurology Resident Sleep Project
Jayson D. Rodriguez, MD; Jeannie McGee, DHEd, MSHE, CHES, CCRC; David McCarty, MD; Debra Davis

Introduction: This pilot study aims to determine sleep schedules among LSUHSC-Shreveport neurology residents using a two-day sleep log, correlating sleep with sense of well-being and education satisfaction.

Methods: PGY2 and 3 residents take “24+6” hour calls, every 3+days, with non-clinical staff for analysis. Information on resident sleep during the preceding two days, call status and PGY level were recorded in 7 subjects (34 seizures). After extracting source waveforms, the effective connectivity was estimated using a phase slope index (PSI) metric.

Results: Two hundred and fifty studies were identified in 146 boys and 104 girls with mean age of 9.2 years. 150 children either had epilepsy or were diagnosed with epilepsy after the evaluation. Polysomnography or sleep evaluation was abnormal in 94%. EEG in 54% with events or seizures recorded in 40% of the studies. Sleep related breathing problems were the most common presenting symptoms (30%) and obstructive sleep apnea and obstructive hypoventilation were the most common sleep disorders (28%). Additionally, significantly higher number of children with spells/parasomnia had normal sleep evaluation (37% Vs 9%, p=0.00003) as compared to children with epilepsy.

Conclusion: This study demonstrates that combined EEG and polysomnography may be a convenient, efficient and useful tool in evaluation of children with epilepsy and spells. Additionally, significantly fewer children with epilepsy had normal sleep study/evaluation as compared to children with other events/parasomnia. Hence, sleep disorders are more common in epilepsy and sleep evaluation is important in children with epilepsy. Further prospective study can help in establishing the prevalence of sleep disorders in children.
**EXHIBITS & PRODUCT THEATERS**

**Exhibit Hall Hours**
Friday, February 12, 2016
- 10:30AM – 4:00PM    Exhibit Hall Open
- 10:30 – 11:00AM    Coffee Break
- 12:45 – 1:00PM    Lunch
- 3:30 – 4:00PM    Coffee Break

Saturday, February 13, 2016
- 7:00AM – 2:00PM    Exhibit Hall Open
- 7:00 – 8:00AM    Continental Breakfast & Poster Tours
- 9:30 – 10:30AM    Coffee Break
- 12:45 – 2:00PM    Lunch

**Exhibit Hall Floorplan**

**Exhibitors**

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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.

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Marlborough, MA 01752
Phone: 508.481.6700
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Aptiom (eslicarbazepine acetate) indicated for the treatment of partial onset seizure for epilepsy.

Upsher-Smith Laboratories, Inc.
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Upsher-Smith Laboratories, Inc., founded in 1919, is a growing, fully integrated pharmaceutical company dedicated to its mission of delivering high-value, high-quality therapies and solutions which measurably improve individuals’ lives. Upsher-Smith has a particular focus on developing therapies for people living with central nervous system (CNS) conditions, such as seizure disorders. For more information, visit www.upsher-smith.com.
EXHIBITS & PRODUCT THEATERS

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PRODUCT THEATERS

IMPLEMENTING A ROSA™ ROBOTIC-ASSISTED NEUROSURGERY PROGRAM
Presented by: MedTech
Friday, February 12, 2016
1:00 – 2:00PM
Location: International Ballroom, Product Theater
Instructor: Sumeet Vadera, MD (Assistant Clinical Professor — Department of Neurological Surgery, UC Irvine Health)

Dr. Vadera earned his medical degree at Jefferson Medical College in Philadelphia, Penn., and completed a neurosurgery residency and a fellowship in epilepsy surgery at the Cleveland Clinic Foundation in Cleveland, Ohio.

This session, hosted by Medtech Surgical, will feature a discussion of the ROSA™ robotic program at UC Irvine, and a review of some of the cases performed utilizing ROSA to guide minimally invasive SEEG and surgical interventions.

Dr. Vadera is a fellowship-trained UC Irvine Health neurosurgeon who specializes in epilepsy surgery, with expertise in minimally invasive surgery for adult and pediatric patients with epilepsy. His clinical expertise is in neuromonitoring, specifically subdural grids and stereoelectroencephalography (SEEG), and the early treatment of patients with medically intractable epilepsy. He performs laser surgery to treat deep epileptic lesions in the brain, using minimally invasive techniques as well as implantation of the Neuropace RNS® (responsive neurostimulation) device.

REVELATIONS FROM CHRONIC AMBULATORY ECOG MONITORING: INSIGHTS FROM THE RNS® SYSTEM
Presented by: NeuroPace
Saturday, February 13, 2016
1:00 – 2:00PM
Location: International Ballroom, Product Theater
Instructors: Martha Morrell, MD
David Spencer, MD
Lawrence Hirsch, MD, FACNS

This session will provide an update on what chronic ambulatory ECOG data is teaching us about epilepsy in general and about individual patients. Discussions will cover ECOG biomarkers, circadian periodicities in epileptiform discharges and electrographic seizures and shifting laterality in mesial temporal lobe epilepsies.
ANNUAL MEETING OVERVIEW

Saturday, February 13, 2016

7:00 – 8:00AM  Continental Breakfast — Visit Exhibits and Poster Tours

8:00 — 9:30AM  Concurrent Sessions:
  - Montage Matters: Designing EEG Montages for Optimal Localization  Grand Salon 4 & 5
  - Stand-Alone Home Video: A New Tool for the Diagnosis of Seizures?  Grand Salon 2
  - Interpreting Challenging Electrodiagnostic Findings - An Interactive Case-Based Approach  Grand Salon 1
  - Sleep and Sudden Death  Grand Salon 3

9:30 – 10:30AM  Coffee Break — Visit Exhibits and Posters  International Ballroom

10:00 — 11:00AM  General Session: Travel Award Presentation & Jasper Award Lecture  Grand Salon 4

11:00 – 11:15AM  Walking Break

11:15AM – 12:45PM  Concurrent Sessions:
  - New Insights in the Mechanism of Sudden Unexpected Death in Epilepsy  Grand Salon 4 & 5
  - The New Landscape of Neurostimulation a Myriad of Choices  Grand Salon 1
  - Can Clinical Neurophysiology Makes Sports Healthier (and Better)?  Grand Salon 2
  - History and Future of Clinical Neurophysiology  Grand Salon 3

12:45 – 2:00PM  Lunch — Visit Exhibits and Posters  International Ballroom

2:00 – 3:30PM  Concurrent Sessions:
  - Clinical Neurophysiology Trials and Tribulations in Neurocritical Care  Grand Salon 4 & 5
  - Remote EEG Monitoring: Building a Network  Grand Salon 1
  - Toxic and Drug Induced Peripheral Neuropathies  Grand Salon 2
  - REM Sleep Behavior Disorder and REM Sleep without Atonia: Diagnosis and Treatment  Grand Salon 3

3:30 – 3:45PM  Walking Break

3:45 – 5:15PM  Concurrent Sessions: Special Interest Groups
  - The Wisdom and Vision from the ACMEGS Inaugural Decade  Grand Salon 2
  - Non-invasive Methods of Cortical Mapping  Grand Salon 4 & 5
  - Neonatal and Infantile EEG and Seizure Patterns: When do we Need an EEG?  Grand Salon 1
  - The EEG a Great Tool  Grand Salon 3

5:15 – 5:30PM  Walking Break

5:30 – 7:00PM  General Session: Research Highlights & Schwab Award Lecture  Grand Salon 4 & 5

7:00 – 7:30PM  Annual Business Meeting  Grand Salon 4 & 5

Sunday, February 14, 2016

7:00 – 8:00AM  Continental Breakfast  Grand Salon Foyer

8:00 – 9:30AM  Concurrent Sessions:
  - Functional Neurosurgery and NIOM — From Spine to Brain  Grand Salon 1
  - The Future of EMG in the Current Medical Environment  Grand Salon 2
  - Multimodal Strategies in Surgical Epilepsy Planning ( Estrategias Multimodales en la Planeación de Cirugía de Epilepsia (Joint ACNS/Latin American Chapter of IFCN)  Grand Salon 3
  - New Frontiers in Electrocorticography  Grand Salon 4 & 5

9:30 – 10:00AM  Coffee Break

10:00 – 11:30AM  Concurrent Sessions:
  - Clinical Neurophysiology of Insular Cortex Epilepsy  Grand Salon 1
  - Functional Brain Mapping: Overview and Future Directions  Grand Salon 2
  - What Can We Learn from the Postictal State?  Grand Salon 3

11:30AM  Adjourn
ANNUAL MEETING & COURSES

PHOENIX 2017

FEBRUARY 8-12, 2017
SHERATON GRAND PHOENIX