

ACNS Fall Courses  
SEPTEMBER 20-21, 2014  
**Boston**  
HYATT REGENCY BOSTON

# Final Program

[www.acns.org](http://www.acns.org)



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# President & Chair's Message

Dear Colleagues,

On behalf of the American Clinical Neurophysiology Society (ACNS), it is our pleasure to welcome you to Boston for the 2014 ACNS Fall Courses. The Fall Courses continue to provide an excellent opportunity for professional growth through education in rapidly-evolving areas of clinical neurophysiology led by experts in the field.

The ACNS Course Committee has developed an exceptional program, including parallel courses on Intensive Care Unit Electroencephalography (ICU EEG) monitoring and Neurophysiologic Intraoperative Monitoring (NIOM). We also hope you will find the new Epilepsy Surgery and Pediatric EEG morning courses to be beneficial additions to this weekend's program.

In addition to the courses, please do not hesitate to take advantage of the strong exhibitor presence and ample opportunities to interact with colleagues. Please be sure to stop by the Exhibit Hall during meals and breaks as well as attend the Welcome Reception on Saturday evening.

On behalf of the ACNS Council and Course Committee, we would like to thank everyone involved in the 2014 Fall Courses for ensuring their continued success. We hope that the courses provide a valuable educational opportunity for all in attendance and wish everyone a safe and enjoyable weekend in Boston.

Sincerely,



Atif M. Husain, MD, FACNS  
President



Tobias Loddenkemper, MD, FACNS  
Course Committee Chair



# Special Thanks to the Fall Course Faculty

Dr. Loddenkemper would like to recognize and thank the 2014 Fall Course Faculty:

## **Intensive Care Unit Electroencephalography (ICU EEG)**

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Nicholas S. Abend, MD  
Cecil D. Hahn, MD, MPH, FACNS  
Susan T. Herman, MD, FACNS  
Lawrence J. Hirsch, MD, FACNS  
Gregory L. Holmes, MD, FACNS  
Aatif M. Husain, MD, FACNS  
Suzette M. LaRoche, MD, FACNS  
Marc R. Nuwer, MD, PhD, FACNS  
Saurabh R. Sinha, MD, PhD  
Courtney J. Wusthoff, MD

## **Neurophysiologic Intraoperative Monitoring (NIOM)**

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Ronald Emerson, MD, FACNS  
Gloria Galloway, MD, FACNS  
Aatif M. Husain, MD, FACNS  
Alan D. Legatt, MD, PhD, FACNS  
Marc R. Nuwer, MD, PhD, FACNS  
Mirela V. Simon, MD, FACNS  
Stanley Skinner, MD, FACNS

## **Epilepsy Surgery Update**

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Lawrence J. Hirsch, MD, FACNS  
Stephan U. Schuele, MD, MPH, FACNS  
William O. Tatum, DO, FACNS

## **Pediatric EEG Update: Special Applications of EEG to Pediatric Epilepsy**

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Gregory L. Holmes, MD, FACNS  
Phillip L. Pearl, MD, FACNS  
Elizabeth A. Thiele, MD, PhD

# ACNS Information

## Officers and Council 2014-2015

### PRESIDENT

Aatif M. Husain, MD, FACNS  
Duke University Medical Center

### FIRST VICE PRESIDENT

William O. Tatum, DO, FACNS  
Mayo Clinic College of Medicine / Mayo Clinic Florida

### SECOND VICE PRESIDENT

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

### SECRETARY

Stephan Schuele, MD, MPH, FACNS  
Northwestern University

### TREASURER

Tobias Loddenkemper, MD, FACNS  
Children's Hospital Boston

### IMMEDIATE PAST PRESIDENT

Frank W. Drislane, MD, FACNS  
Beth Israel Deaconess Medical Center

### PAST PRESIDENT

Susan T. Herman, MD, FACNS  
Beth Israel Deaconess Medical Center

### COUNCILORS-AT-LARGE

Jeffrey Britton, MD, FACNS  
Mayo Clinic

Richard C. Burgess, MD, PhD, FACNS  
Cleveland Clinic Foundation

Gloria Galloway, MD, FACNS  
Ohio State University Medical Center

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

Suzette M. LaRoche, MD, FACNS  
Emory University School of Medicine

Jaime R. Lopez, MD, FACNS  
Stanford University

Raj D. Sheth, MD, FACNS  
Mayo Clinic / Nemours Clinic

### AMA OFFICER

Marc R. Nuwer, MD, PhD, FACNS  
UCLA Medical Center

### JOURNAL EDITOR

Aatif M. Husain, MD, FACNS  
Duke University Medical Center

## Course Committees 2014-2015

### COURSE COMMITTEE

Chair: Tobias Loddenkemper, MD

Nicholas S. Abend, MD  
Cecil D. Hahn, MD, MPH, FACNS  
Lawrence J. Hirsch, MD, FACNS  
Marc R. Nuwer, MD, PhD, FACNS  
Juan Ochoa, MD  
Saurabh R. Sinha, MD, PhD  
Jaime Lopez, MD, FACNS  
Ex Officio: Stephan U. Schuele, MD, MPH, FACNS  
William O. Tatum, DO, FACNS

### CONTINUING MEDICAL EDUCATION COMMITTEE

Chair: Stephan Schuele, MD, MPH, FACNS

Nicholas S. Abend, MD  
Jayant Acharya, MD  
Meriem Bensalem-Owen, MD, FACNS  
Rohit Das, MD, FACNS  
Susan T. Herman, MD, FACNS  
Jong Woo Lee, MD, PhD, FACNS  
Mirela V. Simon, MD, FACNS  
Saurabh R. Sinha, MD, PhD  
Christa Swisher, MD  
Greg Worrell, MD

## Executive Office

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

### EXECUTIVE DIRECTOR

Megan M. Kelley, CMP  
mkelley@acns.org



## Not an ACNS Member? Join Now!

The benefits of joining are endless but here are just a few:

- Reduced fees for the Annual Meeting & Courses and In-Service Examination.
- Reduced dues for members in training and first-year practitioners.
- Access to the *Journal of Clinical Neurophysiology*.
- Access to the Online Member Directory.

Please visit the ACNS website, [www.acns.org](http://www.acns.org), for more information and ways to join!

# General Meeting Information

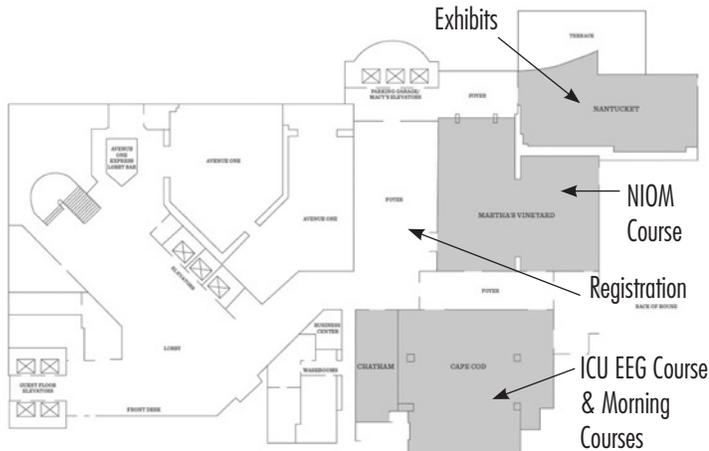
## Venue & Floor Plan

The Hyatt Regency Boston is the location of the 2014 Fall Courses.

### HYATT REGENCY BOSTON

1 Avenue de Lafayette  
Boston, MA 02111

### THIRD FLOOR



## Registration Desk

Location: 3<sup>rd</sup> Floor Foyer

Friday, September 19	5:00 – 7:00 PM
Saturday, September 20	6:00 AM – 5:00 PM
Sunday, September 21	6:00 AM – 4:00 PM

## Internet Access

For your convenience, complimentary Wi-Fi access is available in the Hyatt Regency Boston third floor meeting space.

### WIRELESS ACCESS INSTRUCTIONS

To log on to the wireless network:

1. View Available Wireless Networks
2. Select Access Point: Hyatt Meeting
3. When prompted to login with access code: acns2014

(Password is case sensitive, all lower case letters.)

## Photography and Recording Policy

Photographing or video or audio recording of sessions, materials presented in sessions, 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 the property of ACNS.

## Complimentary Dining

The following meals will be provided to Fall Courses delegates:

Saturday, September 20, 2014

Breakfast	7:00 – 9:00 AM
Lunch	12:00 – 1:30 PM
Reception	6:00 – 7:00 PM (hors d'oeuvres and cash bar)

Sunday, September 21, 2014

Breakfast	7:00 – 9:00 AM
Lunch	12:00 – 1:30 PM

Beverages will also be provided during scheduled breaks on Saturday and Sunday.

## Nearby Restaurants

The following list includes restaurants within walking distance or a short cab ride from the Hyatt Regency; please use this list for your convenience should you wish to leave the hotel for dinner.

Avenue One  
*Upscale New England*  
1 Avenue de Lafayette  
(617) 422-5579  
Located in the Hyatt Regency Boston

Back Deck  
*American*  
2 West Street  
(617) 670-0320

Barking Crab  
*Seafood*  
88 Sleeper Street  
(617) 426-2722

Blu Restaurant  
*New American*  
4 Avery Street  
(617) 375-8550

Boston Common Coffee Co.  
*Bakery and Café*  
515 Washington Street  
(617) 542-0595

Kingston Station  
*Globally Inspired Bistro*  
25 Kingston Street  
(617) 482-6282

Panera Bread  
*Bakery and Café*  
115 Stuart Street  
(617) 722-8234

Papagayo  
*Mexican*  
15 West Street  
(617) 426-2350

Ruth's Chris Steak House  
*Steak House*  
45 School Street  
(617) 742-8401

Sorriso Trattoria  
*Italian*  
107 South Street  
(617) 259-1560

Stoddard's Fine Food and Ale  
*Gastropub*  
48 Temple Place  
(617) 426-0048

The Corner Mall  
*Food Court*  
*Bourbon Street Café*  
*Casserole Café*

*Charley's Philly Steaks*  
*Dunkin' Donuts*  
*McDonald's*  
*Sarku Japan*

*Salsa's Mexican Grill*  
*Subway*  
*Sushi Time*  
*Sweet Tooth Boston*

*Thai Accent*  
*Wong's of Boston*  
417 Washington Street

# General Meeting Information

## Certificate of Attendance

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Certificates of Attendance will be provided online. CME certificates will be available to pre-registered delegates immediately upon the close of the meeting at [www.acns.org](http://www.acns.org). Delegates who registered on-site will be able to obtain a CME certificate at [www.acns.org](http://www.acns.org) starting October 24, 2014.

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 Fall 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](mailto:info@acns.org) for any questions. ACNS asks that all CME certificates be claimed no later than December 31, 2014.



# Continuing Medical Education (CME) Information

## Purpose

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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

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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

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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

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

## Gaps and Needs

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

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

### GAP 1. EMERGING AREAS OF PRACTICE

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

### GAP 2. GENERAL PRACTICE OF CLINICAL NEUROPHYSIOLOGY

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

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

These include:

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

## Changes in Behavior/Practice

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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

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The updated ACCME accreditation criteria are designed to integrate with the new requirements for maintenance of certification (for more information see [www.ABPN.org](http://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.

# Continuing Medical Education (CME) Information

## Policy on Financial Disclosures

It is the policy of ACNS to ensure balance, independence, objectivity and scientific rigor in all its individually sponsored or jointly sponsored educational programs. In order to comply with the ACCME's Updated Standards for Commercial Support, ACNS requires that anyone who is in a position to control the content of an educational activity discloses all relevant financial relationships with any commercial interest pertaining to the content of the presentation. Should it be determined that a conflict of interest exists as a result of a financial relationship of a planner of the CME activity, the planner must recuse himself or herself from the planning for that

activity or relevant portion of that activity. All presentations for which the presenter disclosed a potential conflict of interest are peer reviewed by two members of the ACNS CME Committee with no relationships. If bias is found, the presenter is asked to make changes to the presentation and it is re-reviewed for bias before final approval. Refusal to disclose a conflict or the inability to resolve an identified conflict precludes participation in the CME activity. Complete conflict of interest disclosure information is printed in the final program for the activity. A learner may request additional information regarding the nature of a planner or speaker's disclosure if "No Relevant Relationships" has been indicated below. To request additional information, contact the ACNS Executive office at [info@acns.org](mailto:info@acns.org).

## Conflict of Interest Disclosure

Key:  
 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.)

Council		
Jeffrey W. Britton, MD, FACNS	Mayo Clinic	No Relationships
Richard C. Burgess, MD, PhD, FACNS	Cleveland Clinic Foundation	No Relationships
Frank W. Drislane, MD, FACNS	Beth Israel Deaconess Medical Center	LWW (g)
Jonathan C. Edwards, MD, FACNS	Medical University of South Carolina	No Relationships
Gloria Galloway, MD, FACNS	Ohio State University Medical Center	No Relationships
Cecil D. Hahn, MD, MPH, FACNS	Hospital for Sick Children-Neurology	No Relationships
Susan T. Herman, MD, FACNS	Beth Israel Deaconess Medical Center	UCB Pharma (a); Eisai, Inc (e)
Aatif M. Husain, MD, FACNS	Duke University Medical Center	UCB Pharma (a, d, e); Jazz Pharma (b, c); Demos Publisher (g); Sage Pharmaceuticals (e)
Suzette M. LaRoche, MD, FACNS	Emory University School of Medicine	Demos Publishing (g)
Tobias Loddenkemper, MD, FACNS	Children's Hospital Boston	Lundbeck (a); Eisai (a)
Jaime Lopez, MD, FACNS	Stanford University	No Relationships
Marc R. Nuwer, MD, PhD, FACNS	University of California, Los Angeles	Sleep Med (f); Corticare (c)
Stephan U. Schuele, MD, MPH, FACNS	Northwestern University-Neurology	GSK (e); Lundbeck (e); Danny Did Foundation (a); NIH (a); Sunovion
Raj D. Sheth, MD, FACNS	Mayo Clinic/Nemours Clinic	No Relationships
William O. Tatum, DO, FACNS	Mayo College of Medicine	No Relationships
Course Committee (if not listed above)		
Nicholas S. Abend, MD	Children's Hospital of Philadelphia	Demos Medical Publishing (g)
Lawrence J. Hirsch, MD, FACNS	Yale University	UCB, Inc (a); Upsher-Smith (a, b); Lundbeck (a, b); Neuropace (c); Natus (c); Allergan (c); UpToDate Neurology (g); Atlas of EEG in Critical Care, Wiley (g)
Juan Ochoa, MD	University of South Alabama	No Relationships
Saurabh R. Sinha, MD	Duke University Medical Center	Cyberonics, Inc. (a, d); Lundbeck (e); Accordia (e); Upsher Smith Laboratories (a, e); UCB Pharmaceuticals (a)

# Continuing Medical Education (CME) Information

## Conflict of Interest Disclosure

CME Committee (if not listed above)		
Jayant Acharya, MD	Penn State Hershey Medical Center	Upsher-Smith Laboratories, Inc (e); Sunovion Pharmaceuticals, Inc (e)
Meriem Bensalem-Owen, MD, FACNS	University of Kentucky	UCB (a); Eisai (a); Sunovion (a); Lundbeck (a)
Rohit Das, MD, FACNS	Indiana University School of Medicine	Upsher Smith (b)
Jong Woo Lee, MD, PhD, FACNS	Brigham & Women's Hospital	UCB, Inc (a); DigiTrace, Inc (f); Sunovion, Inc (a); Duke Clinical Research Institute (a)
Mirela V. Simon, MD, FACNS	Massachusetts General Hospital	Demos Medical Publishing (g)
Christa Swisher, MD	Duke University Medical Center	No Relationships
Greg Worrell, MD	Mayo Clinic	No Relationships
Course Faculty (if not listed above)		
Ronald Emerson, MD, FACNS	Hospital for Special Surgery	Amgen (c); Dow Chemical (c); Eli Lilly (c); Express Scripts (c); General Electric (c); Johnson & Johnson (c); Thermo Fisher Scientific (c); Allergan (c); Bristol Myers Squibb (c); Teva Pharm (c)
Gregory L. Holmes, MD, FACNS	University of Vermont College of Medicine	Janssen (b); Eisai (e); NHBLI (e); NINDS (e)
Alan D. Legatt, MD, PhD, FACNS	Montefiore Medical Center	Several companies that market health care goods or services; none of them are related to the subject of my presentation. (c)
Phillip L. Pearl, MD, FACNS	Boston Children's Hospital	No Relationships
Stanley Skinner, MD, FACNS	Abbott Northwestern Hospital	Medtronic (g)
Elizabeth A. Thiele, MD, PhD	Massachusetts General Hospital	No Relationships
Courtney J. Wusthoff, MD	Stanford University	No Relationships
Executive Office Staff		
Megan M. Kelley, CMP	ACNS	No Relationships

Key:

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.)

# Continuing Medical Education (CME) Information

## Program Description

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 neurophysiologic techniques and knowledge in the diagnosis and management of patients with disorders of the peripheral and central nervous system.

The 2014 Fall Courses are designed around two of the new and rapidly-evolving areas of clinical neurophysiology, Neurological Intraoperative Monitoring (NIOM) and Intensive Care Unit EEG Monitoring (ICU EEG). Educational activities will cover both basic methodologies for those practitioners new to NIOM and ICU EEG, and innovative techniques. New to the Fall Courses in 2014 are morning courses covering two additional topics, Epilepsy Surgery and Pediatric EEG.

## Learning Objectives

### EPILEPSY SURGERY UPDATE

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

1. Understand the principles underlying Stereo EEG and how to choose the appropriate method for an individual patient;
2. Discuss several approaches to minimally invasive epilepsy surgery;
3. Understand the principles of different stimulation techniques in the treatment of epilepsy and chose the appropriate intervention for individual patients.

### PEDIATRIC EEG UPDATE: SPECIAL APPLICATIONS OF EEG TO PEDIATRIC EPILEPSY

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

1. Recognize a relationship between interictal spikes and cognitive impairment;
2. Interpret data consistent with prefrontal epileptiform activity and deficits in attentional and social behavior in animals;
3. Identify multifactorial components of epileptogenicity in tuberous sclerosis;
4. Understand the relationship between EEG findings and cognitive regression in tuberous sclerosis;
5. Identify EEG patterns associated with inherited metabolic epilepsies;
6. Utilize a diagnostic and treatment algorithm in treatable metabolic epilepsies.

### INTENSIVE CARE UNIT ELECTROENCEPHALOGRAPHY MONITORING (ICU EEG) AT THE CONCLUSION OF THIS COURSE, PARTICIPANTS WILL BE ABLE TO:

1. Recognize common indications for CEEG in the ICU setting in neonates, children, and adults;
2. Interpret EEG patterns encountered in the ICU, including seizures and periodic patterns;
3. Utilize quantitative EEG methods for data reduction and real-time detection of EEG changes in the ICU;
4. Select appropriate equipment for ICU-EEG monitoring, including networking and data storage options, and;
5. Determine optimal staffing, data review, and reporting of results.

### NEUROPHYSIOLOGIC INTRAOPERATIVE MONITORING (NIOM)

#### AT THE CONCLUSION OF THIS COURSE, PARTICIPANTS WILL BE ABLE TO:

1. Identify the various types of NIOM and discuss their utilization;
2. Interpret NIOM Case based presentations;
3. Utilize NIOM Case based presentations to differentiate between usual and atypical changes;
4. Illustrate technical challenges associated with NIOM;
5. Describe the current issues relevant to the practice environment of NIOM.

## 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 the ACCME to provide continuing medical education for physicians.

## Credit Designation

ACNS designates the Fall Courses for the maximum number of *AMA PRA Category 1 Credit(s)*<sup>™</sup> and ASET CEUs indicated below:

Intensive Care Unit EEG Monitoring (ICU EEG):  
12.75 *AMA PRA Category 1 Credits*<sup>™</sup>

Neurophysiologic Intraoperative Monitoring (NIOM):  
12.75 *AMA PRA Category 1 Credits*<sup>™</sup>

Epilepsy Surgery:  
1.5 *AMA PRA Category 1 Credits*<sup>™</sup>

Pediatric EEG:  
1.5 *AMA PRA Category 1 Credits*<sup>™</sup>

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

## ASET-CEU Designation

ASET – The Neurodiagnostic Society has granted ASET Continuing Education (ACE) credits as follows for this program. Such crediting, however, should not be construed by program participants as an endorsement of any type of instruments or supplies mentioned or involved in these presentations.

Intensive Care Unit EEG Monitoring (ICU EEG)      15.5 ASET-CEUs

Neurophysiologic Intraoperative Monitoring (NIOM)      16 ASET-CEUs

# Program Agenda

## Saturday

### EPILEPSY SURGERY UPDATE

7:00 – 8:30 AM

Room: Cape Cod, 3rd Floor

Course Co-Chairs: Stephan U. Schuele, MD, MPH, FACNS and William O. Tatum, DO, FACNS

#### Invasive Exploration with Stereo EEG vs. Subdural Electrodes

*Stephan U. Schuele, MD, MPH, FACNS*

#### Lasers, Gamma Knife or Electrocoagulation: A Brave New World to Minimal Invasive Epilepsy Surgery

*William O. Tatum, DO, FACNS*

#### Stimulation Therapy for Epilepsy: RNS, DBS or VNS

*Lawrence J. Hirsch, MD, FACNS*

8:30 – 9:00 AM

#### Breakfast

Room: Exhibit Hall (Nantucket, 3rd Floor)

### INTENSIVE CARE UNIT ELECTROENCEPHALOGRAPHY MONITORING (ICU EEG)

9:00 AM – 6:00 PM

Room: Martha's Vineyard, 3rd Floor

Course Co-Chairs: Nicholas S. Abend, MD and Saurabh R. Sinha, MD, PhD

9:00 – 9:10 AM

#### Introductory Remarks and Historical Perspective

*Lawrence J. Hirsch, MD, FACNS*

9:10 – 10:30 AM

#### Indications for CEEG – Seizure Identification Adults

*Saurabh R. Sinha, MD, PhD*

#### Neonates

*Courtney J. Wusthoff, MD*

#### Pediatrics

*Nicholas S. Abend, MD*

10:30 – 10:50 AM

#### Break & Visit Exhibits

Room: Exhibit Hall (Nantucket, 3rd Floor)

10:50 AM – 12:30 PM

#### Indications for CEEG – Other Ischemia Detection

*Susan T. Herman, MD, FACNS*

#### Prognosis – Adult

*Suzette M. LaRoche, MD, FACNS*

#### Prognosis – Neonates and Children

*Courtney J. Wusthoff, MD*

12:30 – 1:30 PM

#### Lunch & Visit Exhibits

Room: Exhibit Hall (Nantucket, 3rd Floor)

1:30 – 3:30 PM

#### CEEG Interpretation Background

*Saurabh R. Sinha, MD, PhD*

#### Seizures and Periodic Patterns

*Lawrence J. Hirsch, MD, FACNS*

#### Neonatal

*Courtney J. Wusthoff, MD*

3:30 – 4:00 PM

#### Break & Visit Exhibits

Room: Exhibit Hall (Nantucket, 3rd Floor)

4:00 – 6:00 PM

#### Seizure Management

#### Neonates and Children

*Tobias Loddenkemper, MD, FACNS*

#### Adults

*Aatif M. Husain, MD, FACNS*

#### Cases

*Courtney J. Wusthoff, MD; Cecil D. Hahn, MD, MPH, FACNS; Suzette M. LaRoche, MD, FACNS; Lawrence J. Hirsch, MD, FACNS*

# Program Agenda

## Saturday

<b>NEUROPHYSIOLOGIC INTRAOPERATIVE MONITORING (NIOM)</b> 9:00 AM – 6:00 PM Room: Cape Cod, 3rd Floor Course Co-Chairs: Marc R. Nuwer, MD, PhD and Gloria Galloway, MD	2:40 – 3:00 PM	<b>Break &amp; Visit Exhibits</b> Room: Exhibit Hall (Nantucket, 3rd Floor)
9:00 – 9:05 AM	<b>Introduction and Welcome</b> <i>Marc R. Nuwer, MD, PhD, FACNS</i>	<b>Case Discussion: Spinal Cord Monitoring</b> <i>Ronald Emerson, MD, FACNS</i>
9:05 – 10:25 AM	<b>SEP</b> <i>Aatif M. Husain, MD, FACNS</i>	<b>Case Discussion: Spinal Cord Monitoring</b> <i>Mirela V. Simon, MD, FACNS</i>
	<b>MEP</b> <i>Ronald Emerson, MD, FACNS</i>	<b>Case Discussion: Selective Dorsal Rhizotomy</b> <i>Gloria Galloway, MD, FACNS</i>
10:25 – 10:45 AM	<b>Break &amp; Visit Exhibits</b> Room: Exhibit Hall (Nantucket, 3rd Floor)	<b>Q &amp; A Panel Discussion</b>
10:45 AM – 12:00 PM	<b>Cranial Nerves</b> <i>Alan D. Legatt, MD, FACNS</i>	<b>Reception &amp; Visit Exhibits</b> Room: Exhibit Hall (Nantucket, 3rd Floor)
	<b>EEG</b> <i>Marc R. Nuwer, MD, PhD, FACNS</i>	
12:00 – 1:00 PM	<b>Lunch &amp; Visit Exhibits</b> Room: Exhibit Hall (Nantucket, 3rd Floor)	
1:00 – 2:40 PM	<b>BAEP</b> <i>Alan D. Legatt, MD, PhD, FACNS</i>	
	<b>NIOM in Spastic CP</b> <i>Gloria Galloway, MD, FACNS</i>	
	<b>Case Discussion: CPA Surgery</b> <i>Alan D. Legatt, MD, PhD, FACNS</i>	

# Program Agenda

## Sunday

### PEDIATRIC EEG UPDATE: SPECIAL APPLICATIONS OF EEG TO PEDIATRIC EPILEPSY

7:00 – 8:30 AM

Room: Cape Cod, 3rd Floor

Course Co-Chairs: Gregory L. Holmes, MD and Philip L. Pearl, MD

#### EEG as a Biomarker for Cognition

*Gregory L. Holmes, MD, FACNS*

#### EEG Correlations in Tuberous Sclerosis

*Elizabeth A. Thiele, MD, PhD*

#### EEG in Genetic-Metabolic Epilepsies

*Phillip L. Pearl, MD, FACNS*

8:30 – 9:00 AM

#### Breakfast

Room: Exhibit Hall (Nantucket, 3rd Floor)

### INTENSIVE CARE UNIT ELECTROENCEPHALOGRAPHY MONITORING (ICU EEG)

9:00 AM – 4:00 PM

Room: Martha's Vineyard, 3rd Floor

Course Co-Chairs: Nicholas S. Abend, MD and Saurabh R. Sinha, MD, PhD

9:00 – 10:30 AM

#### Quantitative EEG

##### Utility

*Cecil D. Hahn, MD, MPH, FACNS*

#### Workshop and Cases

*Susan T. Herman, MD, FACNS*

10:30 – 10:50 AM

#### Break & Visit Exhibits

Room: Exhibit Hall (Nantucket, 3rd Floor)

10:50 AM – 12:00 PM

#### Administrative Issues

##### Equipment, Networking, and Electrodes

*Saurabh R. Sinha, MD*

#### Staffing, Personnel, Workflow, and Logistics

*Cecil D. Hahn, MD, MPH, FACNS*

#### Billing and Coding

*Marc R. Nuwer, MD, PhD, FACNS*

#### Questions and Discussion

12:00 – 1:00 PM

#### Lunch & Visit Exhibits

Room: Exhibit Hall (Nantucket, 3rd Floor)

1:00 – 2:30 PM

#### Other Topics

##### Impact of Seizures – Bench Studies

*Gregory L. Holmes, MD, FACNS*

##### Impact of Seizures – Clinical

*Nicholas S. Abend, MD*

2:30 – 3:00 PM

#### ICU EEG Guidelines

*Suzette M. LaRoche, MD, FACNS*

#### Break

Room: 3rd Floor Foyer

3:00 – 4:00 PM

#### Questions and Discussion

### NEUROPHYSIOLOGIC INTRAOPERATIVE MONITORING (NIOM)

9:00 AM – 4:00 PM

Room: Cape Cod, 3rd Floor

Course Co-Chairs: Marc R. Nuwer, MD, PhD and Gloria Galloway, MD

9:00 – 9:05 AM

#### Welcome

*Gloria Galloway, MD, FACNS*

9:05 – 10:25 AM

#### Anesthesia

*Ronald Emerson, MD, FACNS*

#### EMG and Peripheral Nerves

*Stanley Skinner, MD, FACNS*

10:25 – 10:45 AM

#### Break & Visit Exhibits

Room: Exhibit Hall (Nantucket, 3rd Floor)

10:45 AM – 12:00 PM

#### Case Discussion: Peripheral Surgery

*Mirela V. Simon MD, FACNS*

#### Case Discussion: Lower Spinal Surgery

*Stanley Skinner, MD, FACNS*

#### Q & A Panel Discussion

12:00 – 1:00 PM

#### Lunch & Visit Exhibits

Room: Exhibit Hall (Nantucket, 3rd Floor)

1:00 – 2:20 PM

#### Billing and Regulatory Issues

*Marc R. Nuwer, MD, PhD, FACNS*

#### Cortical Mapping and Stimulation

*Marc R. Nuwer, MD, PhD, FACNS*

2:20 – 2:35 PM

#### Break

Room: 3rd Floor Foyer

2:35 – 4:00 PM

#### Case Discussion: Brain Tumor Surgery

*Mirela V. Simon, MD, FACNS*

#### Case Discussion: Aortic Surgery

*Atif M. Husain, MD, FACNS*

#### Q & A Panel Discussion

# Presentation Abstracts

## INTENSIVE CARE UNIT ELECTROENCEPHALOGRAPHY (ICU EEG)

### Indications for CEEG - Seizure Identification

#### Adults

*Saurabh R. Sinha, MD, PhD*

Seizure detection is the most common indication for ICU EEG monitoring. The incidence of seizures in this population is high: ranging from 10-30% depending on the underlying diagnosis and patient selection. Moreover, most seizures in this population are non-convulsive with no or only subtle clinical manifestations. This means that without EEG, they would commonly be missed. Routine EEGs also miss a substantial portion of these seizures, but 24-48 hours of continuous EEG will capture most. The relevance of detecting these seizure is somewhat less certain. The use of ICU EEG to detect or rule out seizures has a significant impact on clinical decision-making. Furthermore, clinical and pre-clinical evidence suggests that seizures in patients with neurological insults leads to poorer outcomes. In this presentation, we will discuss the specific indications for ICU EEG in adult patients, emphasizing the likelihood of seizures and the impact this has on outcome.

#### Neonates

*Courtney J. Wusthoff, MD*

This session will critically review the evidence basis regarding the use of CEEG to identify seizures in neonates. We will identify why neonatal seizures are unique from seizures in older children and adult patients. Data regarding which neonatal groups are at highest seizure risk will be synthesized into suggested practice algorithms. Recent studies of neonatal seizures during therapeutic hypothermia and in preterm infants will be included. The ACNS Guideline on EEG monitoring in the neonate will be examined, along with the evidence supporting these recommendations.

#### Pediatrics

*Nicholas S. Abend, MD*

Electrographic seizures refer to seizures evident on electroencephalographic (EEG) monitoring, and they are common in critically ill children with acute encephalopathy. Most electrographic seizures have no associated clinical changes; EEG monitoring is required for identification. In current clinical practice, most clinicians monitor for 1-2 days when screening for seizures. Some clinical predictors have been identified, and recent work has developed seizure prediction models which may help guide use of limited EEG monitoring resources. A number of recent studies have addressed the test characteristics of various quantitative EEG modalities, and while imperfect implementation may improve EEG monitoring efficiency.

### Indications for CEEG - Other

#### Prognosis – Adult

*Suzette M. LaRoche, MD, FACNS*

For decades, routine EEG has been an important tool to assist in the determination of prognosis, particularly for patients with anoxic encephalopathy following cardiac arrest. The 2006 AAN practice parameter for prediction of outcome in comatose survivors of cardiac arrest suggested that burst-suppression or generalized epileptiform discharges predicted poor outcomes but with insufficient prognostic accuracy (Level C). However, this recommendation was based upon literature review of studies that predominantly evaluated brief, routine EEGs and prior to the era of therapeutic hypothermia. Today, with expanding utilization of continuous EEG monitoring beyond mere seizure detection and treatment, there is growing evidence for the value of cEEG in assisting in prediction of prognosis; not only for patients following therapeutic hypothermia after cardiac arrest but also for patients suffering from other primary brain injuries as well as status epilepticus. Current knowledge regarding cEEG findings predictive of prognosis in various disease states in adults will be discussed.

#### Prognosis - Neonates and Children

*Courtney J. Wusthoff, MD*

CEEG is often used in children and neonates not just for seizure detection, but also for prognostication. Assessment of EEG background features, seizures, and interictal abnormalities can provide real-time information about neurologic function for use in short-term decision making, and also in understanding expected outcomes for children and neonates with acute brain dysfunction. This session will evaluate how CEEG may best be used for prognostication in critically ill children, with an emphasis on similarities and differences between prognostic features in pediatric patients and adults. Special attention will be given to neonatal patients, and how CEEG can be used to prognosticate in neonatal encephalopathy.

# Presentation Abstracts

## CEEG Interpretation

### Background

*Saurabh R. Sinha, MD, PhD*

Most ICU EEG monitoring is performed for the purposes of seizure detection and management. However, careful interpretation of ICU EEG can provide additional important information to aid in the management of these patients. Like routine EEGs, the background activity can suggest the presence of focal and diffuse disturbances in cerebral function as well as the potential for epileptic seizures. In addition, there are other background patterns frequently seen in ICU EEG (both due to the patient population and the prolonged recording period) such as periodic discharges, rhythmic activity, and stimulus-induced activity that are less common in routine EEGs. In prolonged recordings, it is also important to note changes in the background over time. Changes in background EEG over time can supplement the clinical exam and inform the ICU team about the patient's condition and the impact of manipulations on cerebral function. It can be an indicator of worsening function, like delayed cerebral ischemia in patients with subarachnoid hemorrhages. In certain clinical situations, for example postcardiac arrest, the background EEG and how it changes with time/manipulation can aid in prognosis. In this presentation, I will review important aspects of the background EEG in adult patients, including terminology, and the implications of specific patterns.

### Seizures and Periodic Patterns

*Lawrence J. Hirsch, MD, FACNS*

Nomenclature for periodic patterns has recently been standardized by the ACNS. Periodic patterns (PDs) are simply divided into lateralized (LPDs) and generalized (GPDs), with descriptions of frequency, morphology (including triphasic morphology), sharpness, stability (static, fluctuating or evolving), etc. LPDs (previously known as PLEDs) are seen with any acute epileptogenic process (most commonly stroke) and are highly associated with seizures in the acute illness (about 75% overall). LPDs are occasionally unequivocally ictal (e.g., when they have a clinical correlate, or when treatment leads to immediate neurologic improvement). In general, treatment is to prevent definite seizures rather than to obliterate the LPDs. If and when LPDs themselves cause neuronal injury remains unclear. Bilateral independent PDs (BIPDs) are similarly associated with seizures, but more associated with coma and worse prognosis than LPDs. GPDs are also associated with seizures in the acute illness (about 50% overall in the one large controlled study), but not as highly as LPDs, and GPDs are most associated with nonconvulsive seizures/status. GPDs are somewhat associated with worse outcome (varies between studies). When and how aggressively to treat GPDs remains unclear, but we typically treat the GPDs themselves only when they reach >2 Hz or have a clinical correlate. Whether characteristics of the GPDs themselves can help determine whether they are seizure-related or not remains unclear; there are group differences, but this determination is very difficult in a given patient given extensive overlap and fluctuations over time with prolonged recordings. PDs (and seizures) are commonly elicited or exacerbated by alerting stimulation in the critically ill; this does not help determine whether or not the pattern is ictal or whether or not to treat them. An acute IV antiepileptic drug treatment trial can occasionally prove that a pattern with PDs is ictal by leading to rapid neurologic improvement, but there is no known method to prove a pattern is not ictal.

### Neonatal

*Courtney J. Wusthoff, MD*

Neonatal EEG is often considered particularly challenging by neurophysiologists and trainees. At the same time, the use of CEEG in Neonatal Intensive Care Units is expanding rapidly. This session will use the ACNS Guideline on Neonatal EEG Terminology and Classification as a framework for approaching interpretation. We will focus on high-yield features to identify in those neonates most likely to undergo CEEG, with an emphasis on the practical implications of each finding. Normal and abnormal background for term and preterm neonates will be illustrated. Seizures and interictal abnormalities will be reviewed. Common pitfalls in neonatal EEG will be identified, with discussion of how to evaluate tricky patterns.

### Seizure Management

#### Adults

*Aatif M. Husain, MD, FACNS*

Seizures most commonly seen in critically ill patients are either status epileptic (SE) or nonconvulsive seizures (NCS). SE can either be generalized convulsive (GCSE) or nonconvulsive (NCSE). GCSE is readily recognized and is treated aggressively as it is a neurologic emergency. NCSE and NCS are more difficult to diagnose, and their contribution to neurologic morbidity is less clear. Usually the same antiepileptic drugs (AEDs) and the same treatment algorithm are used to treat GCSE and NCSE/NCS. However, it is unclear whether NCSE/NCS should be treated as aggressively as GCSE. Clinicians are more reluctant to use anesthetic agents for the control of seizures in NCSE/NCS than GCSE. Other AED options are available and may be appropriate in NCSE/NCS. These issues will be explored in this presentation.

# Presentation Abstracts

## Quantitative EEG

### Utility

*Cecil D. Hahn, MD, MPH, FACNS*

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

## Administrative Issues

### Equipment, Networking and Electrodes

*Saurabh R. Sinha, MD, PhD*

The equipment and software needed for a successful ICU EEG program share many similarities with the equipment needed for long-term monitoring and event routine EEGs. The actual recording machine today is almost always a digital EEG system with video capabilities. Appropriate networking infrastructure is needed to allow for obtaining recording in different parts of the hospital while ideally allowing review of the data continuous and even from remote locations. Beyond routine EEG collection and review software, software for quantitative analysis of the EEG is often desirable. Standard cup metal electrodes are often used; however, the clinical, safety and practical concerns often dictate the use of special electrodes such as disposable, needle or MRI/CTcompatible electrodes. Although there are concerns about the quality of such recordings, the need for rapid application of the electrodes and application by personnel who are not trained EEG technologists have led to the use of templates for electrode placement to exploration of reduced montages or simplified electrode placements. In this presentation, we will review specific considerations and requirements for equipment, networking infrastructure, electrodes and electrode montages as they relate to ICU EEG.

### Staffing, Personnel, Workflow and Logistics

*Cecil D. Hahn, MD, MPH, FACNS*

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

### Billing and Coding

*Marc R. Nuwer, MD, PhD, FACNS*

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

# Presentation Abstracts

## Other Topics

### Impact of Seizures - Bench Studies

*Gregory L. Holmes, MD, FACNS*

Major co-morbidities of epilepsy include cognitive impairment and behavioral dysfunction. The type and degree of these co-morbidities is dependent upon age, seizure type and seizure frequency. The hippocampus and prefrontal cortex appear particularly vulnerable to seizure-induced dysfunction. Rodent studies allow investigators to understand the cellular basis for both the cognitive and behavior disturbances seen following prolonged or recurrent seizures. Cognitive impairment and behavioral disturbances are devastating co-morbidities of epilepsy, and may be far more impairing than the seizures themselves. While the etiology of the seizures is the most important determinant of outcome, there is now unequivocal data that prolonged or recurrent seizures can result in cognitive and behavioral deficits. A number of morphological changes can occur with repeated seizures including cell loss, synaptic reorganization and changes in neurogenesis. Likewise seizures can result in electrophysiological changes including changes in excitatory and inhibitory currents, alterations in brain oscillation strength and coordination, and impaired single cell firing patterns. Paralleling these morphological and physiological changes, rats subjected to seizures have considerable cognitive deficits including deficits of spatial cognition in the Morris water maze, impairment of auditory discrimination, altered activity level in the open field and reduced behavioral flexibility as well as behavioral abnormalities.

A major challenge is to determine which, if any of these morphological and physiological changes following seizures relate to life-long cognitive and behavioral deficits. Epilepsy is a disorder that affects neuronal networks, and cognitive and behavioral deficits related to seizures are due to the pathological interactions between many components of the developing and adult brain function. Recent studies have suggested that the cascade of morphological and molecular changes occurring after seizures result in a disturbance of hippocampal oscillations. Oscillations in brain structures provide temporal windows that bind coherently cooperating neuronal assemblies for the representation, processing, storage and retrieval of information. Both theta (4-10 Hz) and gamma oscillations (30-100 Hz) are critically involved in mnemonic function of the hippocampus. Seizure-induced changes at the molecular level will almost certainly affect the fine tuning of oscillatory activity. Accordingly, small errors in oscillation coordination can propagate swiftly across complex networks, and may even become amplified by cross-rhythms of cortical structures. These changes in hippocampal and prefrontal oscillation can lead to widespread deficits in behavior and cognition.

### Impact of Seizures – Clinical

*Nicholas S. Abend, MD*

Electrographic seizures are associated with worse outcomes in critically ill children and neonates. In part, seizures may serve as biomarkers of more severe brain injury. Additionally, however, there is increasing evidence that high electrographic seizure burdens are associated with worse clinical outcomes after adjustment for brain injury etiology and severity, indicating that a high electrographic seizure burden may independently contribute to secondary brain injury. This presentation will review the available data addressing associations between electrographic seizures and outcome, with a focus on studies which have included multivariate analysis to adjust for brain injury type and severity.

### ICU EEG Guidelines

*Suzette M. LaRoche, MD, FACNS*

Since the era of evidence based medicine and the prioritization of cost effective, quality healthcare, standardized guidelines have become increasingly important across many areas of medicine. Development of consistent standards to guide patient selection as well as technical and clinical protocols is especially important when new technologies are being developed, especially those that are resource intense such as continuous EEG monitoring. Recent surveys have shown that although the practices of continuous EEG monitoring for critically ill patients is growing rapidly, a wide variety of practices exist amongst practitioners and institutions. Although still an emerging technology, guidelines have been published to address a variety of relevant issues pertaining to the practice of ICU EEG monitoring for both neonatal and adult populations. These guidelines will be discussed in addition to recently proposed ACNS guidelines for ICU EEG monitoring of the adult population.

# Presentation Abstracts

## NEUROPHYSIOLOGIC INTRAOPERATIVE MONITORING (NIOM)

### SEP

*Atif M. Husain, MD, FACNS*

Somatosensory evoked potential (SEP) evaluate the dorsal column pathways. SEP monitoring is used when the spinal cord, brainstem and brain structures are at risk of injury during surgery. At times SEP monitoring is also used when the cauda equina and peripheral nerves are at risk. SEP monitoring is often used in combination with motor evoked potential monitoring, and together they provide complementary monitoring of the posterior and anterior aspects of the spinal cord. In this presentation, the anatomy of SEP will be discussed, followed by the technique used to obtain these potentials. Effects of anesthetics, mechanisms that lead to SEP changes and the warning criteria will be discussed. Finally, the shortcomings of SEP will also be noted.

### MEP

*Ronald Emerson, MD, FACNS*

This lecture will provide an introduction to transcranial motor evoked potentials, including physiology, anesthetic effects, recording techniques and interpretation.

### Cranial Nerves

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

The anatomy of the cranial nerves that innervate muscles (CN 3-7 and 9-12) and the techniques used to monitor them during surgery will be described. Motor cranial nerves are monitored by recording EMG from appropriate muscle groups. Somatosensory evoked potentials to stimulation of trigeminal nerve branches can be recorded, but this is technically challenging. The patient cannot be pharmacologically paralyzed during EMG recordings; the absence of neuromuscular blockade should be verified. Spontaneous EMG activity may reflect mechanical stimulation and irritation of cranial nerves. Repetitive neurotonic EMG discharges are more worrisome than isolated discharges, but can also be caused by irrigation with cold fluids. Recording of EMG elicited by stimulation within the surgical field can be used to localize and identify cranial nerves within the surgical field. When monitoring cranial nerve 7, EMG should be recorded from multiple facial nerve-innervated muscles, in case the nerve has been splayed into separated fascicles by a tumor. During surgery, conduction blocks may develop in nerves that remain in anatomic continuity, and will interfere with intraoperative monitoring of the nerves. A variety of artifacts may appear during EMG monitoring of motor cranial nerves; several examples will be shown.

### EEG

*Marc R. Nuwer, MD, PhD, FACNS*

EEG is used to monitor cases at risk for ischemia. Most often this is used in carotid endarterectomy to gauge whether a shunt is needed or blood pressure change is recommended. Sometimes this is used in other cardiothoracic surgery and vascular neurosurgery for similar reasons. EEG progressively loses fast activity, gains slow activity, and then loses total amplitude as the blood flow falls below 20 ml/100gm/min, eventually becoming isoelectric around 12 ml/100gm/min. Decreased fast activity is the earliest sign of clinically relevant impaired blood flow. Increased slow activity is an intermediate degree of change. An isoelectric EEG is the worst end of the spectrum of change. Confounding effects arise from anesthesia changes and hyperventilation during surgery. Occasionally EEG is monitored for a patient who has known risk factor such as epilepsy who is taken to surgery for a procedure elsewhere in the body.

### BAEP

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

BAEPs are useful for intraoperative monitoring of the ears, auditory nerves, and the brainstem auditory pathways up through the level of the mesencephalon. They are relatively unaffected by anesthesia, though they are affected by hypothermia. Technical aspects of auditory stimulation and of recording of BAEPs will be reviewed. During BAEP monitoring, each patient serves as his/her own control. Both amplitude and latency measurements should be followed. Wave I is generated in the distal eighth nerve. Subsequent components are composites of contributions from multiple generators, but wave III predominantly reflects activity in the caudal pons and wave V predominantly reflects activity in the mesencephalon. Adverse intraoperative changes in BAEPs can be caused by technical factors (including artifacts), hypothermia, acoustic masking, and localized dysfunction within the infratentorial auditory system. Possible causes of the latter include direct mechanical or thermal injury of neural tissue, compromise of the blood supply to the brainstem or to the cochlea, and stretch of or traction on the eighth nerve.

# Presentation Abstracts

## **NIOM in Spastic CP**

*Gloria Galloway, MD, FACNS*

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Effecting treatment for severe disabling spasticity particularly in the pediatric population can be very challenging. These severe cases of spasticity are most often seen in children with cerebral palsy and significant functional debilitation. This presentation will provide a background for the use of NIOM in cases of spasticity requiring selective dorsal rhizotomy and nonamenable to medication intervention. Because of the severity of clinical debilitation a rationale approach to NIOM in these patients is essential. Through this presentation a familiarity with the goals, indications and expectations for surgical intervention will be gained. Additionally the NIOM methods applied to achieve favorable outcomes will be discussed and current data will be incorporated in the discussion to support the use of these methods in maximizing clinical outcomes. Importantly, information on possible complications associated with surgical intervention in these cases will be reviewed along with the role of NIOM in reducing the risk of complication.

## **Case Discussion: CPA Surgery**

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

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Neurophysiologic intraoperative monitoring data recorded during several different cerebellopontine angle surgeries will be presented in an interactive session. The audience will be asked to analyze the NIOM data, and to discuss possible causes of the observed changes in the evoked potential data and the clinical significance of the NIOM findings.

## **Case Discussion: Spinal Cord Monitoring I**

*Ronald Emerson, MD, FACNS*

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This lecture will present a series of vignettes, illustrating the principles and interpretation of spinal cord monitoring.

## **Case Discussion: Spinal Cord Monitoring II**

*Mirela V. Simon, MD, FACNS*

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I will present a case of PNS mapping where different neurophysiologic techniques were used to identify nerves/roots, localize the lesion, assess function and thus preserve it by guiding the surgical management.

## **Case Discussion: Selective Dorsal Rhizotomy**

*Gloria Galloway, MD, FACNS*

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This format will apply information reviewed from the NIOM in Spasticity presentation to clinical cases. Cases of severe spasticity will be presented and an analysis of the appropriateness of surgical intervention, the methods used for monitoring; the goals to be achieved in the individual cases requiring SDR intervention and the role of NIOM in each case. Interpretation of electromyographic NIOM data in individual cases and correlation with clinical information will be done. Information on clinical outcome of SDR procedure cases will be reviewed.

## **Anesthesia**

*Ronald Emerson, MD, FACNS*

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This lecture will discuss the common anesthetic agents used during intraoperative neurophysiological monitoring. We will discuss the effects of the various agents on monitored signals, how anesthetic technique can be adjusted to optimize monitoring, and how monitoring technique can be adapted to various anesthetic techniques.

## **EMG and Peripheral Nerves**

*Stanley Skinner, MD, FACNS*

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In the laboratory, motor conduction studies are recorded using surface electrodes over the target muscle. Supramaximal stimulation of can synchronously depolarize all of the nerve's functioning motor axons with minimal temporal dispersion. Therefore, a well-placed surface electrode should capture a large, reproducible compound muscle action potential (CMAP). Needle electromyography (EMG) can effectively record a restricted field of a few cubic millimeters nearest the electrode tip. A few motor units can be assayed with each new passage of the needle. Near field recording makes possible motor unit analysis and acquisition of pathologic spontaneous activity. Therefore, typical EMG electrodes are not well suited to record CMAPs; not surprisingly, surface electrodes (including short "EEG" needles) often fail to record intraoperative neurotonics. In the OR, compromises are made. In the past, one could justify extra amplifiers to record both surface and intramuscular derivations to record both CMAPs and neurotonics, respectively. That is rarely possible now. Nevertheless, intraoperative navigation about cranial nerves, for example, depends on reliable neurotonic surveillance and acquisition of amply summated CMAPs after stimulation. Robust conduction studies (and neurotonic recording) can usually be performed if a sufficient length of bare wire (a partially denuded monopolar EMG electrode) can be implanted intramuscularly.

# Presentation Abstracts

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## Case Discussion: Peripheral Surgery

*Mirela V. Simon, MD, FACNS*

I will present a case of PNS mapping where different neurophysiologic techniques were used to identify nerves/roots, localize the lesion, assess function and thus preserve it by guiding the surgical management.

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## Case Discussion: Lower Spinal Surgery

*Stanley Skinner, MD, FACNS*

A reliable intraoperative bulbocavernosus reflex (BCR) may be recorded after train (or double train) stimulation at the genitalia. BCR is contingent upon somatic sensory and motor fibers of the pudendal nerve as well as Onuf's nucleus (conus medullaris). Therefore, BCR testing elaborates other IONM recordings (SEP, MEP, EMG). External anal sphincter (EAS) recording may be included in low thoracic and lumbosacral settings. Free-running EAS EMG, EAS recording after sacral root stimulation (M wave), and BCR are suggested during cauda equina/conus level approaches. MEPs may include the EAS as a specific measure of corticospinal, sacral root, and pudendal efferent function. At cauda equina level, BCR and EAS MEP become surrogate tests for pelvic parasympathetic function (parasympathetic fibers associated with sacral somatic roots at cauda level). Lateral approaches to the upper lumbar spine place the lumbar plexus and genitofemoral nerve at risk. Men may complain of postoperative "dropped" testicle or pain referred to the testicle (or labium major in women). The cremaster is accessed by needle electrode insertion at the inguinal ring. Testing permits: 1) observation of the recruitment of genitofemoral neurotonic ("injury") discharges and 2) threshold stimulation to determine nerve proximity to electrified instruments.

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## Billing and Regulatory Issues

*Marc R. Nuwer, MD, PhD, FACNS*

Coding, billing and adherence to regulations are necessary for Intraoperative Neuromonitoring practice. Three main codes now are used for time monitoring the operating room. They differ by physician location (remote vs in-room) and by case load (one only vs simultaneous cases). Base codes define what modalities are monitored. A variety of regulations govern use of time, supervision, documentation, and related aspects of monitoring. Alternate codes are available for functional cortical localization and for electrocorticography, as well as to deep brain and vagal nerve procedures. Recommendations for code use are discussed in this session.

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## Cortical Mapping and Stimulation

*Marc R. Nuwer, MD, PhD, FACNS*

Cortical stimulation is used to localize eloquent cortex during respective neurosurgery. Most often this is conducted during surgery for patients with intractable epilepsy or malignant hemispheric tumors. Cortex localized includes language, motor and sensory cortices. Stimulation is delivered through a wand held directly onto the exposed cortex. The electrical stimulation disrupts the approximate square centimeter nearest to the wand. Testing of the patient may require awakening the patient during the craniotomy so as to question the patient about his or her experiences and abilities while the local cortical function is disrupted temporarily. Potential problems include provoking seizures from excessive stimulation. To protect against seizures, the electrocorticogram is monitored to watch for signs of incipient seizure activity. A variety of regions are tested at and nearby the regions proposed for resection. This assists the surgeon in decisions about what tissue safely to resect.

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## Case Discussion: Brain Tumor Surgery

*Mirela V. Simon, MD, FACNS*

I will present a case of motor mapping and monitoring during brain tumor surgery as a start point for discussing several challenges in cortical and subcortical motor mapping/monitoring.

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## Case Discussion: Aortic Surgery

*Aatif M. Husain, MD, FACNS*

Aortic surgeries carry some of the highest morbidity and mortality of any type of surgery. The neurologic morbidity of these procedures can be as high as 30-40%. With the advance of surgical techniques and the use of neurophysiologic intraoperative monitoring (NIOM), neurologic morbidity has greatly decreased. In this presentation several cases will be presented that illustrate the various types of aortic surgeries that can benefit from NIOM. With each case, the neurologic structures at risk will be identified, and the best NIOM modality that can be used will be noted. Interpretation of the NIOM data and the outcomes of the cases will be discussed.

# Presentation Abstracts

## EPILEPSY SURGERY UPDATE

### Invasive Exploration with Stereo EEG vs. Subdural Electrodes

*Stephan Schuele, MD, MPH, FACNS*

Approximately 20-30% of surgical epilepsy patients require an invasive evaluation prior to resection. Chronic subdural grids offer systematic mapping and seizure recording optimal for patients with a suspected epileptogenic zone close to functional cortex. Depth electrodes seem to cause less morbidity and complications and can be placed more predictably including deeper areas inaccessible to grids. Delineation of functional cortex may not be possible with depth electrodes and intraoperative mapping may be necessary. The three dimensional exploration with depth electrodes (Stereo EEG) could offer a better outcome for patient with non-lesional extratemporal lobe epilepsy than results in patients with subdural grids have shown. In the near future, more and more epilepsy centers will need to be able to offer individualized invasive approaches to optimize outcome.

### Lasers, Gamma Knife or Electrocoagulation: A Brave New World to Minimal Invasive Epilepsy Surgery

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

Surgical resection is a standard of care for many patients with refractory focal epilepsy syndromes and concordant neurophysiological localization compared to continued use of anti-seizure medication. However, open resection may be associated with post-operative neurocognitive deficits. Minimally invasive surgery (MIS) techniques are emerging that limit tissue sacrifice and therefore unlike conventional craniotomy may reduce adverse consequences by minimizing "collateral damage" by sparing more viable tissue. Early results using laser ablation of mesial temporal structures suggest that seizure-free outcomes are similar to those from a randomized controlled trial of temporal lobectomy. Stereotactic laser ablation promises a less invasive and more desirable approach for a wider patient population than standard and more invasive procedures. Gamma knife stereotactic radiosurgery has demonstrated benefit in patients with mesial temporal lobe epilepsy in prospective multicenter trials. Delayed results of seizure outcome involve ionizing radiation and despite the non-invasive nature creates concerns of post-operative adverse events including radiation necrosis. Coagulation associated with stereotactic radiofrequency amygdalohippocampotomy has shown efficacy in some studies, yet surgical margins may be more indistinct and associated with technical constraints. The approach to MIS offers greater appeal to patients by novel, potentially curative strategies to a wide population of patients who otherwise are likely to remain drug-resistant.

### Stimulation Therapy for Epilepsy: RNS, DBS, or VNS

*Lawrence J. Hirsch, MD, FACNS*

The era of stimulation for treatment of epilepsy has arrived and is expanding. Vagus nerve stimulation (VNS) has withstood the test of time (approved in 1997) and remains a reasonable long-term option for those with severe epilepsy who are not candidates for resective surgery. Although seizure reduction was only modest in the major blinded trials, it was superior to the low-stimulation group, and efficacy improved over time in many long term uncontrolled studies, reaching about a 50% responder rate and seizure reduction rate after several years. Quality of life (QOL) also improves, but seizure freedom is rare. This is clearly the least invasive of the three stimulation options.

Deep brain stimulation (DBS) of the anterior nucleus of the thalamus bilaterally for patients with multifocal epilepsy showed a 40% decline in seizure frequency in the final month of the blinded phase of the SANTE trial, superior to the 14.5% decline in the control (no stimulation) group. In the long term open-label extension, median decline in seizures and responder rate surpassed 50% by 2 years. 13% were seizure free for at least 6 months at some point. Overall QOL improved at 1 year, but there was an increase in self-reported depression and subjective memory impairment (not corroborated on formal testing). DBS is approved for treatment of epilepsy in Canada and Europe, but not in the US.

Responsive neurostimulation (RNS) at the seizure focus or at 2 foci was recently approved for adults in the U.S. The device is cranially implanted and stimulates in response to abnormal intracranial EEG activity. By the end of the blinded period, there was a mean 41.5% decline in seizure rate in the active stimulation group vs 9.4% in the sham stimulation group. QOL improved overall and on many subtests, including memory and attention. Median seizure reduction at two years (open-label) was 53% with a responder rate of 55%. RNS has the additional advantage of providing long-term ambulatory intracranial EEG recordings, particularly useful for those with bitemporal epilepsy. 9% were seizure free for the final 3 months.

When to choose one stimulator over the other will remain unclear for the near future and will likely depend mostly on physician and patient preference. Reasons to choose one or the other will be discussed.

# Presentation Abstracts

## PEDIATRIC EEG UPDATE: SPECIAL APPLICATIONS OF EEG TO PEDIATRIC EPILEPSY

### EEG as a Biomarker for Cognition

*Gregory L. Holmes, MD, FACNS*

Cognitive impairment is a common and often devastating co-morbidity of childhood epilepsy. While the etiology of the epilepsy is critical determinant of cognitive outcome, there is considerable evidence from both rodent and human studies that indicate that seizures and interictal epileptiform abnormalities can contribute to cognitive impairment. A critical feature of childhood epilepsy is that the seizures and epileptiform activity are occurring in a brain with developing, plastic neuronal circuits. The consequences of seizures and interictal epileptiform activity in the developing brain differ from similar paroxysmal events occurring in the relatively fixed circuitry of the mature brain. In animals it is possible to study interictal spikes independently from seizures and it has been demonstrated that interictal spikes are as detrimental as seizures during brain development. In the clinic distinguishing the differences between interictal spikes and seizures is more difficult, since both typically occur together. However, both seizures and interictal spikes result in transient cognitive impairment. Recurrent seizures, particularly when frequent, can lead to cognitive regression. While the clinical data linking interictal spikes to persistent cognitive impairment is limited, interictal spikes occurring during the formation and stabilization of neuronal circuits likely contributes to aberrant connectivity.

### EEG in Genetic-Metabolic Epilepsies

*Phillip L. Pearl, MD, FACNS*

An inherited metabolic epilepsy is highly suspect with onset of seizures, especially myoclonias or infantile spasms, in the neonatal period or during infancy. A typical clinical presentation would be a newborn with poor feeding, hypotonia, lethargy, respiratory distress, or lactic acidosis. EEGs classically show burst-suppression, multifocal discharges, or hypersarrhythmia. A family history of metabolic disorders or consanguinity should be sought, and a poor response to traditional antiseizure drugs is an important sign. Metabolic epilepsies may be classified based on pathogenesis, i.e. energy deficiency (e.g. GLUT1-deficiency, respiratory chain deficiency, creatine deficiency), toxic effects (e.g. amino and organic acidopathies, urea cycle defects), impaired neuronal function (e.g. storage disorders), disturbances of neurotransmitter systems (e.g. glycine encephalopathy, GABA transaminase or SSADH deficiency), associated brain malformations (e.g. peroxisomal disorders, CDG), vitamin or cofactor dependency (e.g. biotinidase deficiency, B6/P5P/folinic acid dependency, Menkes disease). Certain EEG patterns may be characteristic, e.g. comb-like rhythm in MSUD; fast central spikes in Tay Sachs and biotinidase deficiency; vanishing background in infantile NCL; marked photosensitivity in PME and NCL; RHADS in POLG encephalopathies (Alpers). EEG is a key diagnostic criterion to establish the diagnosis of certain genetic epilepsy syndromes, e.g. malignant migratory partial epilepsy of infancy and early myoclonic encephalopathy. EEG patterns may show important ontogenic changes over the lifespan in certain syndromes and serve as a biomarker for risk of SUDEP; Dravet syndrome serves as an example. Specific EEG examples of these disorders, diagnostic guidelines, and management protocols will be reviewed.

# Exhibit Hall Information

## Exhibit Hall Hours

SATURDAY, SEPTEMBER 20 7:00 AM – 7:00 PM

SUNDAY, SEPTEMBER 21 7:00 AM – 1:30 PM

ACNS provides exhibit space at scientific meetings for commercial exhibits related to the fields of basic and clinical neurophysiology. The Society makes no attempt to evaluate any aspect of the material exhibited. Noncompliance with guidelines published by the ACNS has not been considered by the Society in allotting commercial space. Hence, acceptance of these commercial exhibits should not be construed as indicated sponsorship or approval of their products by ACNS.

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Blackrock NeuroMed manufactures and distributes high-end EEG equipment which blends world-class research technology with clinical tools that offer the gold standard of care for patients worldwide.

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The Moberg CNS Monitor continuously records EEG and processed EEG trends, time-synchronized with other physiology. Multimodal monitoring with integrated video, EEG trends and physiology helps you evaluate patients' neurological statuses. View correlations between EEG and parameters from other devices including vital signs, ICP, brain oxygen, cerebral blood flow, and more. Remotely review and annotate EEG and multimodal physiology. Advanced integrated neurophysiological monitoring is available today with the CNS Monitor.

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For more than twenty years Persyst has produced the most trusted and innovative EEG analysis products. Today, Persyst is the world's leading supplier of seizure detection, spike detection, and quantitative analysis software to every leading EEG manufacturer for continuous EEG monitoring, LTM and ambulatory EEG.

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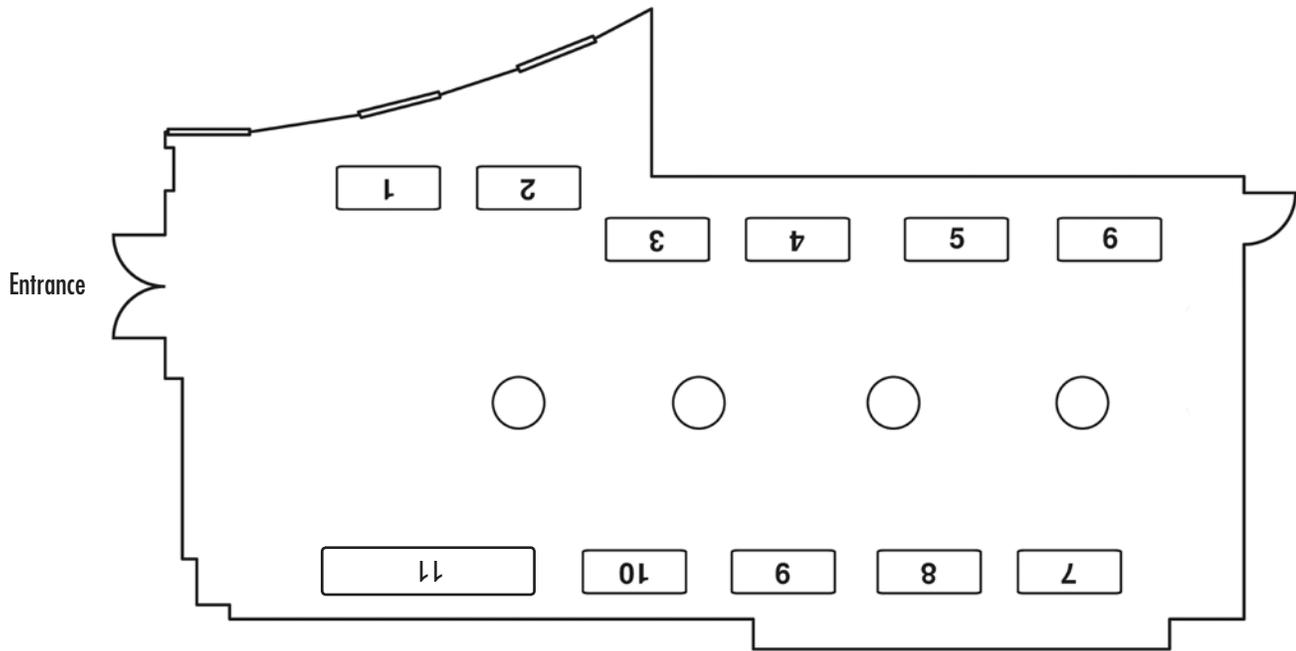


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