ACNS 2013

Annual Meeting & Courses

MIAMI, FLORIDA

February 5-10, 2013

Final Program

AMERICAN CLINICAL NEUROPHYSIOLOGY SOCIETY

2014 Annual Meeting & Courses

ATLANTA

FEBRUARY 4-9, 2014 | ATLANTA, GA

Westin Peachtree Plaza

www.acns.org

ACNS 2013
Annual Meeting & Courses
MIAMI, FLORIDA
February 5-10, 2013

Table of Contents

President's Message 2
Message from Program and Course Chairs 3
ACNS Information 4
- Officers & Council 2012–13 4
- Annual Meeting & Courses Committee Members 5
- Past Presidents 6

General Meeting Information 7
- Registration Desk 7
- Internet 7
- Certificate of Attendance & CME Certificate 7
- Business Meeting 7
- Poster Sessions 7
- Publication of Abstracts 7
- Exhibits 7
- Venue Information 8
- Miami Marriott Hotel Floorplan 8
- Nearby Restaurants 9

CME Information 11
Annual Courses Program 16
Annual Meeting Program 22
Speaker Abstracts 34
Poster Abstracts 40
Exhibit Hall Information 71
- Exhibit Hours 71
- Exhibit Hall Floorplan 71
- Exhibitors 72

Annual Course Outline ..... Inside Back Cover
Annual Meeting Outline ..... Back Cover

President's Message

Dear Fellow ACNS Members and Colleagues,

I enthusiastically welcome you to the American Clinical Neurophysiology Society's (ACNS) 2013 Annual Meeting & Courses. This promises to be a week filled with outstanding educational opportunities, renewal of friendships, and unlimited networking opportunities. Without question, you'll be happy you're here!

This year's program presents no lack of exceptional educational opportunities. "Kicking off" the meeting are the Annual Courses on Tuesday, Wednesday and Thursday, organized by Course Chair William Tatum, DO and his hard-working committee. There are several new exciting additions to the Annual Courses this year. The first, thanks to members' requests, is a new full-day course on Intracranial EEG and a return of the Video-EEG Course. Secondly, we now have expanded evening courses for attendees to take advantage of a relaxed non-CME environment where they may "ask the experts". We've tried to make our sessions as interactive as possible, so please join in!

Beginning Friday, the three-day Scientific Program will feature a record number of poster abstracts. Due to the large number of abstracts, for the first time this year, we will split the poster viewing times into two different sessions. There will be 54 posters presented on Friday and 55 posters on Saturday. Program Committee Co-Chairs Jonathan Edwards, MD and Mark Hallett, MD and their committee members deserve enormous credit for their hard work in organizing an excellent program that, as you will see, covers all aspects of the latest advances in clinical neurophysiology.

I am proud to welcome Jeffery H. Gertsch, MD and C.J. Stam, MD, PhD, as our guest lecturers during Friday and Saturday's Opening Sessions. Dr. Gertsch's Plenary Lecture on Friday titled "Interventional Neurophysiology for the 21st Centruy" and Dr. Stam's Plenary Lecture on Saturday titled "Small World Networks" give a look into the future of clinical neurophysiology. My sincere congratulations go to each of our very impressive award recipients: the Robert S. Schwab Award recipient, Charles Bolton, MD; the Herbert H. Jasper Award recipient, Richard P. Brenner, MD; and the Pierre Gloor Award recipient, Rodolfo Llinas, MD, PhD. These plenary and award lectures will set a strong tone for the rest of the scientific meeting, with exciting sessions in nearly every area of clinical neurophysiology.

This year, we have an outstanding turnout of companies who have come to exhibit during the Annual Meeting. I want to strongly encourage everyone to visit the Exhibit Hall to see all the new and familiar faces. We have set up breakfast, lunch and coffee breaks, as well as our ACNS Reception, in the Exhibit Hall to provide everyone extra opportunities to not only network with the exhibitors, but your colleagues as well.

This has been an outstanding year for me serving as your President. I've had phenomenal support and guidance from our Officers, Council, and committee chairs and members. Through the work of everyone, this society continues to thrive and grow. I especially want to thank our new partners at Executive Director, Inc: Kay Whalen, our Consulting Partner, Megan Kelley, our Executive Director, and Sara Theis, our Meeting and Membership Manager. They have done an exceptional job organizing this meeting. Please be sure to seek them out to welcome and thank them. We can all look back with pride at the tremendous positive changes our Society has undergone during this pivotal transition year. Thank you for allowing me the privilege and honor of being your president.

My very best to all of you,

Susan T. Herman, MD

WELCOME RECEPTION

Dr. Susan Herman would like to formally invite everyone to attend the ACNS Welcome Reception on Friday, February 8, from 6:45 – 8:00 PM in the Grand Ballroom Salons A – E, Level 3. There will be complimentary hors d'oeuvre provided and you will get a chance to see all the new and familiar exhibitors.

Message from Program and Course Chairs

Welcome to Miami, the 2013 Annual Meeting and Courses and what promises to be a week of outstanding educational opportunities!

The Annual Meeting and Course programs have been designed to provide a solid review of the fundamentals and the latest scientific advances in both "central" and "peripheral" clinical neurophysiology.

We're excited to announce several changes to the Annual Course program for 2013, made at the suggestion of ACNS members and meeting attendees:

- The theme of the Annual Courses in 2013 is "intervention."
- Courses on NIOM have been expanded to two full days.
- A new half-day course on Video-EEG has been introduced to update attendees' knowledge of epilepsy monitoring.
- Breakfast seminars on both Wednesday, February 6 and Thursday, February 7 will act as "primers" related to many aspects of clinical neurophysiology.
- Look for expanded evening hours at this year's Courses. Attendees will have the opportunity to take advantage of a relaxed non-CME environment where they may "ask the experts."

This year's Annual Meeting program will again feature the latest scientific advances in clinical neurophysiology presented by leading national and international experts in the field. This dynamic program has more choices than ever, including parallel sessions providing simultaneous sessions for interests in EEG, electrodiagnosis, and monitoring.

The General Session on Friday, February 8 will focus on intensive and intraoperative care, featuring ACNS President Dr. Susan T. Herman describing the utility of EEG in the ICU and a lecture by Dr. Jeffrey Gertsch concerning intraoperative monitoring. Dr. Charles Bolton will discuss critical care neuropathy and myopathy, disorders that he put on the map. Saturday's General Session will highlight brain rhythms, centered around presentations by Dr. Rodolfo Llinas, a pioneer in many aspects of neuroscience, and Dr. C.J. Stam, who will discuss graph theory and its implications for understanding brain function.

And in what has become a hallmark of the ACNS Annual Meeting, we once again invite you to take part in the excitement of the Annual ACNS "Neurophys Bowl" on Friday, February 8, the educational quiz show hosted Drs. Larry Hirsch and Mark Ross.

We are certain that these diverse course offerings, symposia, hands-on workshops and special interest groups will ensure that whether your 'slant' is central or peripheral, you will find much of interest and utility to your practice at this year's ACNS meeting.

Welcome, enjoy your week!

Jonathan C. Edwards, MD and Mark Hallett, MD
Program Co-Chairs

William O. Tatum IV, DO
Course Chair

ACNS ANNUAL BUSINESS MEETING

The ACNS Annual Business Meeting is on Saturday, February 9, from 5:30 – 6:00 PM in the Grand Ballroom Salon F, Level 3.
Everyone is welcome to attend, but only ACNS members may vote.

COMMITTEE MEETINGS

If you are on an ACNS Committee please attend your respective committee meetings on the dates and times listed below:
Course Committee— Thursday, February 7, 5:15 – 6:00 PM in Dodge Island, Level 3
CME Committee—Friday, February 8, 7:00 – 8:00 AM in Biscayne Island, Level 3
Membership Committee—Friday, February 8, 8:00 – 9:00 PM in Dodge Island, Level 3
Program Committee—Saturday, February 9, 7:00 – 8:00 PM in Watson Island, Level 2

ACNS Information

OFFICERS & COUNCIL 2012–13

Susan T. Herman, MD, *President*
Beth Israel Deaconess Medical Center
330 Brookline Avenue, Neurology, Baker 5, Boston, MA 02215
Phone 617.632.8930
Fax 617.632.8931
sherman2@bidmc.harvard.edu

Frank W. Drislane, MD, *First Vice President*
Beth Israel Deaconess Medical Center
330 Brookline Avenue, Neurology, Baker 5, Boston, MA 02215
Phone 617.667.2395
Fax 617.667.7919
fdrislan@bidmc.harvard.edu

Aatif M. Husain, MD, *Second Vice President*
Duke University Medical Center
Box 3678, 202 Bell Building, Durham, NC 27710
Phone 919.684.8485
Fax 919.684.8955
aatif.husain@duke.edu

Jonathan C. Edwards, MD, *Secretary*
Medical University of South Carolina
P.O. Box 250606, Charleston, SC 29425
Phone 843.792.3263
Fax 843.782.8626
edwardjc@musc.edu

Stephan Schuele, MD, MPH, *Treasurer*
Northwestern University-Neurology
710 N Lake Shore Dr, Abbott Hall 1425, Chicago, IL 60622
Phone 312.926.1673
Fax 312.908.5073
s-schuele@northwestern.edu

Douglas R. Nordli, Jr., MD, *Immediate Past-President*
Children's Epilepsy Center
2300 Children's Plaza, #29, Chicago, IL 60614
Phone 773.883.6159
Fax 773.868.8904
dnordli@childrensmemorial.org

Peter W. Kaplan, MB, FRCP, *Past-President*
Johns Hopkins Bayview Medical Center
4940 Eastern Avenue, Baltimore, MD 21224
Phone 410.550.0630
Fax 410.550.0539
pkaplan@jhmi.edu

COUNCIL MEMBERS

Cecil D. Hahn, MD, MPH
Terrence D. Lagerlund, MD
Suzette LaRoche, MD
Tobias Loddenkemper, MD
Jaime R. López, MD
Suraj Ashok Muley, MD
Piotr W. Olejniczak, MD
William O. Tatum IV, DO
Francis O. Walker, MD

EX-OFFICIO

Mark Nuwer, MD, PhD, *AMA Officer*
John Ebersole, MD, *Journal Editor*

EXECUTIVE OFFICE

555 East Wells Street
Suite 1100
Milwaukee, WI 53202
Phone 414.918.9803
Fax 414.276.3349
www.acns.org
info@acns.org

EXECUTIVE DIRECTOR

Megan M. Kelley, CMP
mkelley@acns.org

MEETINGS & MEMBERSHIP MANAGER

Sara E. Theis
stheis@acns.org

ACNS Information

ANNUAL MEETING & COURSES COMMITTEE MEMBERS

PROGRAM COMMITTEE

Jonathan C. Edwards, MD, Co-Chair
Mark Hallett, MD, Co-Chair

Nicholas S. Abend, MD
Pegah Afra, MD
Anto Bagić, MD
Jean E. Cibula, MD
Frank W. Drislane, MD
Ronald G. Emerson, MD
Morris A. Fisher, MD
Gloria Galloway, MD
Andres A. Gonzalez, MD
Cecil D. Hahn, MD, MPH
Susan T. Herman, MD
Lawrence J. Hirsch, MD
Aatif M. Husain, MD
Peter W. Kaplan, MB, FRCP
Alan D. Legatt, MD, PhD
Jaime R. López, MD
Suraj A. Muley, MD
Douglas R. Nordli, Jr, MD
Cormac A. O'Donovan, MD
Mark A. Ross, MD
Jehuda Sepkuty, MD
Mirela V. Simon, MD
Cynthia V. Stack, MD
Leopold J. Streletz, MD
William O. Tatum IV, DO
Francis O. Walker, MD

COURSE COMMITTEE

William O. Tatum IV, DO, Chair

Nicholas S. Abend, MD
Elliott Dimberg, MD
Jonathan C. Edwards, MD
Charles M. Epstein, MD
Mark Hallett, MD
Susan T. Herman, MD
Lawrence J. Hirsch, MD
Arturo Leis, MD
Suzette LaRoche, MD
Jaime R. López, MD
Yafa Minazad, MD
Donald L. Schomer, MD
Francis O. Walker, MD
Gregory A. Worrell, MD

CONTINUING MEDICAL EDUCATION (CME) COMMITTEE

Susan T. Herman, MD, Co-Chair
Stephan Schuele, MD, MPH, Co-Chair

Nicholas S. Abend, MD
Jayant Acharya, MD
Meriem Bensalem-Owen, MD
Rohit Das, MD, PhD
Kitti Kaiboriboon, MD
Jong Woo Lee, MD, PhD
Arturo Leis, MD
Michael L. McGarvey, MD
Daniel L. Menkes, MD
Gregory A. Worrell, MD

ACNS Information

PAST PRESIDENTS

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

The American Clinical Neurophysiology Society gratefully acknowledges the following companies for their support of the 2013 Annual Meeting & Courses.

Cadwell

Compumedics

Esaote

Magstim

Natus Neurology

Persyst

General Meeting Information

REGISTRATION DESK

Location: Bayview Ballroom Foyer
Tuesday, February 5: 8:00 AM – 5:00 PM
Wednesday, February 6: 6:30 AM – 5:00 PM
Sunday, February 10: 7:00 AM – 12:00 PM

Location: Grand Ballroom Foyer
Thursday, February 7: 6:30 AM – 5:00 PM
Friday, February 8: 7:00 AM – 5:00 PM
Saturday, February 9: 7:00 AM – 5:00 PM

INTERNET

For your convenience, there will be free Wi-Fi access in the hotel lobby located on the first floor.

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, 2013.

BUSINESS MEETING

The ACNS Annual Business Meeting will be held in Grand Ballroom Salon F, Level 3 on Saturday, February 9 from 5:30 – 6:00 PM. This meeting is open to all attendees, but only ACNS Members may vote.

POSTER SESSIONS

Authors will be present between 7:00 – 8:00 AM for discussion. Poster abstracts and presentation dates can be found on page 34.

Friday, February 8, 2013

7:00 AM – 1:30 PM	Bal Harbor Island
	Fisher Island
	Hibiscus Island
	Lummus Island

Saturday, February 9, 2013

7:00 AM – 1:30 PM	Bal Harbor Island
	Fisher Island
	Hibiscus Island
	Lummus Island

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

PUBLICATION OF ABSTRACTS

Speaker abstracts and poster abstracts will be published in the *Journal of Clinical Neurophysiology*.

EXHIBITS

Those attending the Annual Meeting are encouraged to visit the Exhibit Hall located in the Grand Ballroom Salons A – E. All meals and coffee breaks on Friday, February 8 and Saturday, February 9 will be held in the Exhibit Hall. Exhibit Hall hours are listed below:

Friday, February 8, 2013

7:00 AM – 5:00 PM	Exhibit Hall Open
7:00 – 8:00 AM	Continental Breakfast
10:00 – 10:30 AM	Coffee Break
12:30 – 1:30 PM	Lunch
3:30 – 4:00 PM	Coffee Break

Saturday, February 9, 2013

7:00 AM – 1:30 PM	Exhibit Hall Open
7:00 – 8:00 AM	Continental Breakfast
10:00 – 10:30 AM	Coffee Break
12:30 – 1:30 PM	Lunch

General Meeting Information

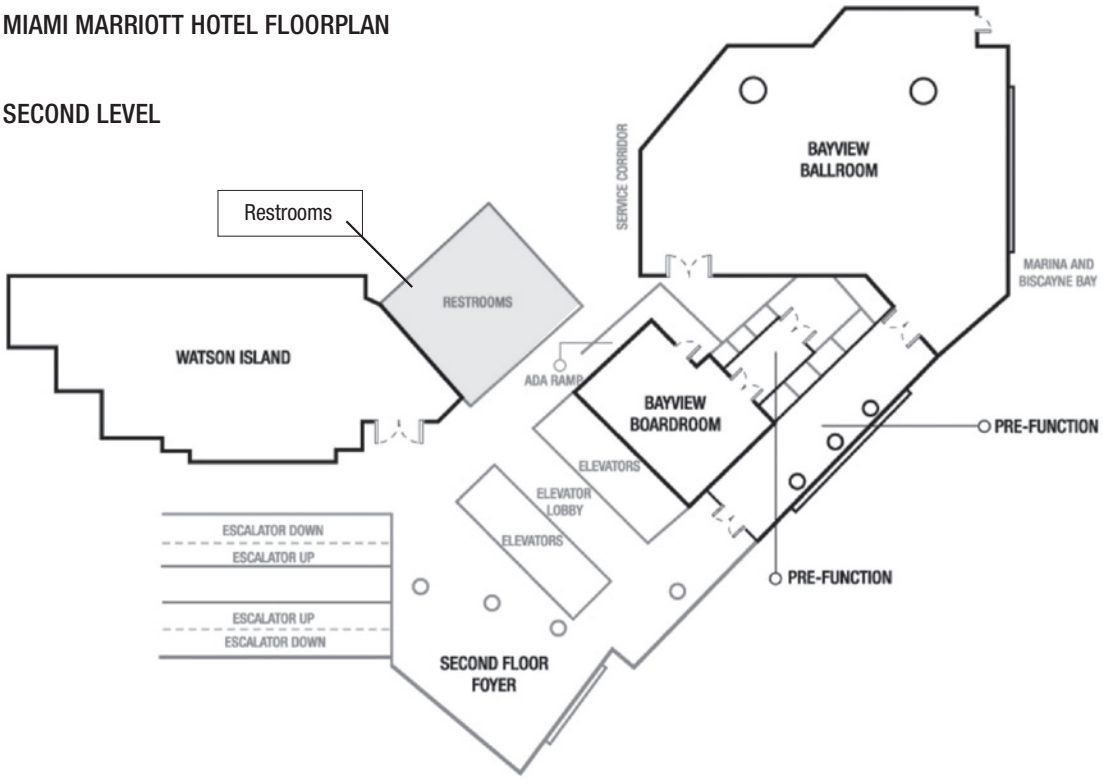
VENUE INFORMATION

The Miami Marriott Biscayne Bay is the location for the 2013 Annual Meeting and Courses. Calls should be directed to the American Clinical Neurophysiology Society Registration Desk.

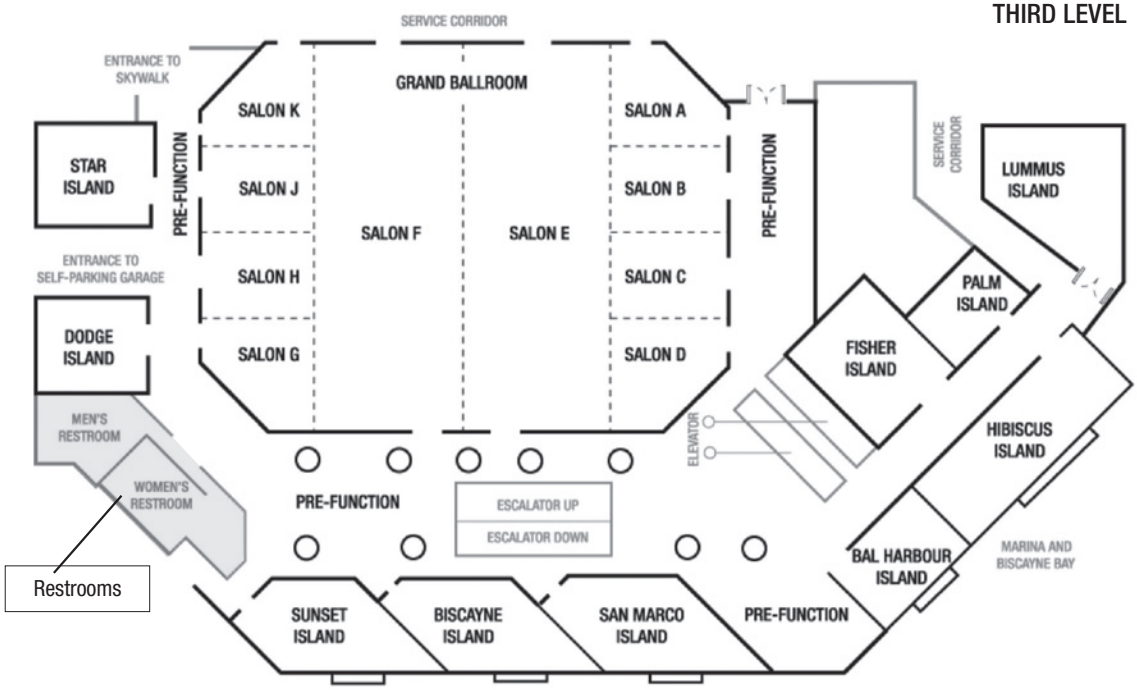
1633 North Bayshore Drive
Miami, FL 33132
(305) 374.3900
http://www.marriott.com/hotels/travel/miabb-miami-marriott-biscayne-bay/

MIAMI MARRIOTT HOTEL FLOORPLAN

SECOND LEVEL



THIRD LEVEL



General Meeting Information

NEARBY RESTAURANTS

The Miami Marriott Biscayne Bay and surrounding area offers a wide array of dining options. Please keep in mind ACNS does not provide lunches during the Courses Tuesday, February 5 through Thursday, February 7. Listed below are nearby restaurants.

**Located inside hotel
*Located within walking distance

Entrée Prices
$ = 10 – 20
$$ = 20 – 30
$$$ = 30 – 40

American		
Michael's Genuine Food & Drink $$$	130 NE 40th St, Miami	305.573.5550
Vesper American Brasserie $$$	1801 Collins Ave, Miami Beach	305.341.1500
The Dutch $$$	2201 Collins Ave, Miami Beach	
*City Hall $$	2004 Biscayne Blvd, Miami	305.764.3130
660 at the Angler $$	660 Washington Ave, Miami Beach	305.534.9600
Asian/ Pan		
Asia de Cuba $$$	1100 West Ave, Miami Beach	305.514.1940
*Nove kitchen & Bar $$	1750 North Bayshore Drive, Miami	786.871.7727
Lantao Kitchen $$	1717 Collins Ave, Miami Beach	305.604.1800
Chinese		
Miss Tao Chinese Café $$	437 Lincoln Rd, Miami Beach	305.534.5488
P.F. Chang's China Bistro $$	901 S Miami Ave, Miami	305.358.0732
Churrasqueria/ Brazilian		
Fogo de Chao $$$	836 1st Street, South Beach	305.672.0011
Flame $$	447 Espanola Way, Miami Beach	305.397.8950
Camilas's (Buffet)	129 SE 1st Ave, Miami	305.375.0715
Contemporary European/ American/ Latin Flavors		
Symcha's $$$	22 Washington Ave, Miami Beach	305.604.0000
The Dining Room $$$	413 Washington Ave, Mimai Beach	305.397.8444
Bin No 18 $	1800 Biscayne Boulevard #107, Miami	786.235.7575
Cuban		
Yuca $$	501 Lincoln Road, Miami Beach	305.532.9822
De Rodriguez de Cuba $$	101 Ocean Dr, Miami Beach	305.672.6624
Larios $$	820 Ocean Dr, Miami Beach	305.532.9577
Versailles $	3555 SW 8th St, Miami	305.444.0240
Puerto Sagua $	700 Collins Ave, Miami Beach	305.673.1115
French		
La Goulue $$$	9700 Collins Ave, Bal Harbour	305.865.2181
db Bistro Moderne $$	255 Biscayne Boulevard Way, Miami	305.421.8800
A La Folie Café $	516 Espanola Way, Mimai Beach	305.538.4484
Italian		
Prime Italian $$$	101 Ocean Drive, South Beach	305.695.8484
Fratelli Lyon $$	4141 NE 2nd Ave, Miami Design District	305.572.2901
IL Gabbiano $$	335 S. Biscayne Blvd, Miami	305.373.0063
Joey's $	NW 2nd Ave, Miami	305.438.0488

General Meeting Information

International and Tapas		
Sugarcane $$$	3250 NE 1st Ave, Miami	786.369.0353
Dolores by you can call me Lolita $$	1000 S Miami Ave, Miami	305.764.3130
Michy's $$	6927 Biscayne Blvd, Miami	305.759.2001
Sra. Martinez $$	4000 NE 2nd Ave, Miami	305.573.5474
Wynwood Kitchen & Bar $$	2550 NW 2nd Ave, Miami	305.722.8959
El Carajo Tapas & Wine $$	1180 SW 57th Ave, Miami	305.264.8740
Indian		
Bengal $$	2010 Biscayne Blvd, Miami	305.403.1976
Guru $	232 12th St, Miami	305.534.3996
Japanese		
Nobu $$$	1901 Collins Ave, Miami Beach	305.695.3232
Zuma $$$	270 Biscayne Boulevard Way, Miami	305.577.0277
Kopas Restaurant $$	5757 SW 8th St, West Miami	305.265.7139
Mediterranean/ Middle Eastern/ Greek		
Vita Restaurant & Lounge $$$	1906 Collins Ave, Miami	305.674.8822
Tantra $$$	1445 Pennsylvania Ave & Espanola Way, Miami Beach	305.672.4765
Maroosh $$	223 Valencia Ave, Coral Gables	305.476.9800
Mexican		
Rosa Mexicano $$$	1111 Lincoln Rd, Miami Beach	305.695.1005
Mercadito $$	3252 NE 1st Ave, Miami	786.369.0430
Parrilla Argentina		
La Parrilla Liberty $$	609 Washington Ave, Miami Beach	305.532.7599
Asa2 $$	9489 Harding Ave, Surfside	305.866.6400
Seafood		
**Catch Grill & Bar $$		305.374.3900
A Fish Called Avalon $$	700 Ocean Dr, Miami Beach	305.532.1727
Area 31 $$	270 Biscayne Boulevard Way, Miami	305.424.5234
Garcia's Seafood Grill $	398 NW North River Dr, Miami	305.375.0765
C-viche $	105 NE 3rd Ave, Downtown	305.577.3454
Spain & Paellas		
Casa Juancho Restaurant $$$	2436 SW 8th St, Miami	305.642.2452
Casa Panza $$	1620 SW 8th St, Miami (Dinner Show)	305.644.3444
Steak		
Capital Grill $$$	444 Brickell Ave, Miami	305.374.4500
Prime 112 $$$	112 Ocean Dr, Miami Beach	305.532.8112
Red the Steakhouse $$$	119 Washington Ave, Miami Beach	305.534.3688
Meat Mearket Restaurant $$$	915 Lincoln Rd, Miami Beach	305.532.0088
Steak and Seafood		
Truluck's $$$	777 Brickell Ave, Miami	305.579.0035
Oceanaire Seafood $$$	900 South Miami Ave, Miami	305.372.8862
Quinn's $$$	640 Ocean Dr, Miami Beach	305.673.6400
Atrio Restaurant & Wine Room $$	1395 Brickell Ave, Miami	305.503.6529
Sushi		
Sushi Samba $$$	600 Lincoln Rd, Miami Beach	305.673.5337
Doraku $$	1104 Lincoln Rd, Miami Beach	305.695.8383

CME Information

EDUCATIONAL MISSION STATEMENT

Purpose: 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.

Types of Activities: The educational activity of the Society consists of an Annual Course which includes didactic lectures and accompanying syllabus, and the Annual Meeting which consists of presentation of scientific papers, thematic symposia, seminars, and workshops. The Society also publishes the *Journal of Clinical Neurophysiology* which includes review articles and original papers.

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

Gaps

In compliance with the Updated Accreditation Criteria of the Accreditation Council for Continuing Medical Education (ACCME), the Continuing Medical Education (CME) 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
- Magnetoencephalograpy Sleep, Use of new scoring system, implications for patient care

Changes in Behavior/Objectives

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

Evaluation

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

Thank you in advance.

Susan T. Herman, MD
Co-Chair, CME
Stephan Schuele, MD, MPH
Co-Chair, CME

CME Information

MEETING DESCRIPTION

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

ANNUAL MEETING LEARNING OBJECTIVES

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

1. Discuss recent advances in electroencephalography, evoked potentials, polysomnography, magnetoencephalography, electromyography, nerve conduction studies and other clinical neurophysiology techniques.

2. Apply advances in clinical neurophysiology techniques to improve the diagnosis of neurologic disorders.

Please see the Annual Meeting Program, pages 22–33, for specific objectives related to each session.

COURSE LEARNING OBJECTIVES

Please see the Course Program, pages 16–21, for specific objectives related to each course.

TARGET AUDIENCE

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

ACCREDITATION STATEMENT

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

CREDIT DESIGNATION

ACNS designates the Annual Meeting for a maximum of 18.5 *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:

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

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

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

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

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

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

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

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

EMG
3.0 AMA PRA Category 1 Credit(s)™

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

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

Electrophysiological and Pathological Findings in Neuromuscular Disease: A Case-Based Approach
2.5 AMA PRA Category 1 Credit(s)™

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

CME Information

POLICY ON FINANCIAL DISCLOSURE

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

Conflicts of Interest

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		
Frank Drislane, MD	Beth Israel Deaconess Medical Center	No Relationships
Jonathan Edwards, MD	Medical University of South Carolina	No Relationships
Cecil D. Hahn, MD, MPH	Hospital for Sick Children	No Relationships
Susan T. Herman, MD	Beth Israel Deaconess Medical Center	Lundbeck (a); UCB (a)
Aatif M. Husain, MD	Duke University Medical Center	UCB (d); USC (d); Jazz Pharma (d)
Peter Kaplan, MB, FRCP	Johns Hopkins Bayview Medical Center	No Relationships
Terrence D. Lagerlund, MD	Mayo Clinic	No Relationships
Suzette M. LaRoche, MD	Emory University School of Medicine	No Relationships
Tobias Loddenkemper, MD	Children's Hospital Boston	No Relationships
Jaime R. Lopez, MD	Stanford University	No Relationships
Suraj Muley, MD	Barrow Neurological Institute	No Relationships
Douglas R. Nordli, Jr., MD	Children's Epilepsy Center	No Relationships
Piotr W. Olejniczak, MD	Louisiana State University	No Relationships
Stephan Schuele, MD, MPh	Northwestern University	No Relationships
William O. Tatum IV, DO	Mayo College of Medicine	No Relationships
Francis O. Walker, MD	Wake Forest University	Siena Biotech (a); Elsevier (g); Merz (b)
CME Committee (if not listed above)		
Nicholas Abend, MD	Children's Hospital of Philadelphia	No Relationships
Jayant Acharya, MD	Penn State Hershey Medical	No Relationships
Meriem Bensalem-Owen, MD	University of Kentucky	UCB (a); Eisai (a); Sunovion (a); Lundbeck (a)
Rohit Das, MD, PhD	Indiana University	No Relationships
Kitti Kaiboriboon, MD	Univeristy Hospitals Case Medical Center	No Relationships
Michael McGarvey, MD	Hospital of the University of Pennsylvania	No Relationships
Arturo Leis, MD	Arturo Leis Electrodiagnostic Consultant	No Relationships
Daniel Menkes, MD	University of Connecticut Health Center	No Relationships
Jong Woo Lee, MD, PhD	Brigham & Women's Hospital	UCB (a); Cephalon, Inc (a); SleepMed/DigiTrace (f)
Gregory Worrell, MD	Mayo Clinic	No Relationships
Program Committee (if not listed above)		
Pegah Afra, MD	Univeristy of Utah	UCB (b,f); Sunovion (b)
Anto Bagic, MD	University of Pittsburgh	No Relationships
Jean Cibula, MD	University of Florida	No Relationships
Ronald G. Emerson, MD	New York Presbyterian Hospital	No Relationships
Morris Fisher, MD	Hines VAH Loyola	No Relationships
Gloria Galloway, MD	Massachusetts General Hospital	Cambridge Publishing (g)
Andres Gonzalez, MD	USC Neurologists, Inc. (HCCII)	No Relationships
Mark Hallett, MD	National Institue of Health	Brainsway (g)

CME Information

Lawrence J. Hirsch, MD	Yale University	UCB-Pharma (a,b); Upsher-Smith (a,b); Lundbeck (a,b); GlaxoSmithKline (b); Wiley Blackwell (g); Up-to-Date (g)
Alan D. Legatt, MD, PhD	Montefiore Medical Center	No Relationships
Cormac O'Donovan, MD	Wake Forest University	No Relationships
Mark Ross, MD	Mayo Clinic Arizona	No Relationships
Jehuda Sepkuty, MD	Swedish Medical Center Seattle	No Relationships
Mirela Simon MD, MSc	Massachusetts General Hospital	No Relationships
Cynthia Stack, MD	Lurie Children's Hospital	No Relationships
Leopold Streletz, MD	Weill Cornell Medical College in Qatar	No Relationships
Annual Meeting Program Directors (if not listed above)		
Andrea Alexopoulos, MD	The Cleveland Clinic Foundation	No Relationships
Richard C. Burgess, MD, PhD	Cleveland Clinic Epilepsy Center	No Relationships
Joseph Drazkowski, MD	Mayo School of Graduate Medical Education	No Relationships
Dawn Eliashiv, MD	University of California, Los Angeles	UCB (b); Lundbeck (b)
Brent P. Goodman, MD	Mayo Clinic	No Relationships
Jonathan J. Halford, MD	Medical University of South Carolina	See Addendum
Gabriel Martz, MD	Medical University of South Carolina	No Relationships
Jonathan Norton, PhD	University of Alberta	No Relationships
Eva Ritzl, MD	Johns Hopkins University	No Relationships
Catherine Schevon, MD	Neurological Institute of New York	No Relationships
Jay L. Shils, PhD, DABNM, FASNM	The Lahey Clinic	Interstitial Technologies (g)
Stanley A Skinner, MD	Abbott Northwestern Hospital	Medtronic Corp (g)
Erik K. St. Louis, MD	Mayo Clinic	No Relationships
M. Brandon Westover, MD, PhD	Massachusetts General Hospital	No Relationships
Courtney Wusthoff, MD	Lucile Packard Children's Hospital at Stanford	No Relationships
Annual Meeting Speakers (if not listed above)		
Jeffrey Arle, MD, PhD	Beth Israel Deaconess Medical Center	No Relationships
Keith Aronyk, MD	University of Alberta Hospital	See Addendum
Lisa Bateman, MD	Columbia University	No Relationships
Selim R. Benbadis, MD	University of South Florida	No Relationships
Edward H. Bertram, MD	University of Virgina	No Relationships
Paul Richard Carney, MD	University of Florida	No Relationships
Bernard Chang, MD	Beth Israel Deaconess Medical Center	No Relationships
Jasvinder Chawla, MD, MBA, FAAN.	Edward Hines, Jr. VA Hospital	No Relationships
ShiNung Ching, MD	See Addendum	See Addendum
Robert Clancy, MD	The Children's Hospital of Philadelphia	See Addendum
Robert S. Fisher, MD, PhD	Stanford University Medical Center	Medtronic (a); Stanford (a); Consulting for Cyberonics (a)
Michael Funke, MD	The University of Texas at Houston School of Medicine	No Relationships
Bruno Gallo, MD	University of Miami School of Medicine	See Addendum
Edward Gallo, CNIM, R EEG T	Neurological Institute of New York	No Relationships
Jean Gotman, PhD	Montreal Neurological Institute	Blackrock Microsystems (g)
Monica Islam, MD	Nationwide Children's Hospital	No Relationships
Mithri Junna, MD	Mayo Clinic	No Relationships
Robert C. Knowlton, MD, MSPH	University of Texas MS	No Relationships
Shafeeq S. Ladha, MD	Barrow Neurological Institute	No Relationships
Paul Maccabee, MD	SUNY Health Sciences	No Relationships
Yafa Minazad, MD	Huntington Hospital	No Relationships

Conflicts of Interest

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.)

CME Information

Pradeep Modur, MD	UT Southwestern Medical Center	No Relationships
Osvaldo Nascimento, MD	See Addendum	See Addendum
Viet Nguyen, MD	Stanford University	No Relationships
Thien Nguyen, MD	Johns Hopkins University	No Relationships
Katherine Noe, MD, PhD	Mayo Clinic Arizona	No Relationships
Marc Nuwer, MD, PhD	University of California, Los Angeles	No Relationships
Andrew Papanicolaou, MD, PhD	LeBonheur Children's Hospital	No Relationships
Patrick Purdon, MD	See Addendum	See Addendum
Elayna O. Rubens, MD	Weill Cornell Medical College	No Relationships
Devon I. Rubin, MD	Mayo Clinic	American Association of Neuromuscular and Electrodiagnostic Medicine (g); AAN (g)
Ueli Rutishauser, PhD	Cedars-Sinai Medical Center	No Relationships
Rani Sarkis, MD	Brigham and Women's Hospital	Sunovion Pharmaceuticals (g)
Mouhsin Shafi, MD	Beth Israel Deaconess Medical Center	See Addendum
Renee Shellhass, MD	UMichigan Pediatric Neurology	No Relationships
Semyon Slobounov, PhD	Penn State University	Head Rehab, LLC (g)
C.J.Stam, MD, PhD	VU University Medical Center	No Relationships
Mark M. Stecker MD, PhD	Winthrop University Hospital	No Relationships
Andrew Trevelyan, D Phil	Newcastle University	No Relationships
Tammy Tsuchida, PhD	Children's National Medical Center	No Relationships
Michael Wagner, PhD	Compumedics Germany GmbH	Compumedics Germany GmbH (f)
Charles D Yingling, PhD	Stanford School of Medicine	No Relationships
G. Bryan Young, MD	Western University	No Relationships
Course Committee (if not listed above)		
Elliott Dimberg, MD	Mayo Clinic	No Relationships
Donald Schomer, MD	Harvard University	SleepMed/ Digi-Trace, Inc. (b) (g)
Charles Epstein, MD	Emory Clinic	Neuronetics, Inc. (g)
Course Co-Directors (if not listed above)		
Deborah Briggs, MD	SBSI/UTSW-Austin Dept. of Neurology	No Relationships
John S. Ebersole, MD	University of Chicago Medical Center	No Relationships
James Chris Sackellares, MD	Optima Neuroscience, Inc.	Optima Neuroscience, Inc. (f)
Course Faculty (if not listed above)		
Sydney Cash, MD, PhD	Massachusetts General Hospital	See Addendum
Dennis Dlugos, MD	University of Pennsylvania	No Relationships
Susan Ebersole, R EEG T	Illinois (MEG) & Ambulatory EEG Centers	No Relationships
Stephen Hantus, MD	Cleveland Clinic	No Relationships
Kevin Kelly, MD	Allegheny General Hospital	No Relationships
Dileep Nair, MD	Cleveland Clinic	No Relationships
Roy Sharma, R ET, R EPT	The Hospital for Sick Children	No Relationships
Deng-Shan Shiau, PhD	Optima Neuroscience, Inc.	Optima Neuroscience, Inc. (f)
Michael Sperling, MD	Thomas Jefferson University	No Relationships
Executive Office Staff		
Jacquelyn Coleman, CAE	ACNS	No Relationships
Megan Kelley, CMP	ACNS	No Relationships
Sara Theis	ACNS	No Relationships
Marie Westlake	ACNS	No Relationships

Conflicts of Interest

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.)

Annual Courses Program — Tuesday, February 5 and Wednesday, February 6

Tuesday, February 5, 2013

**Lunch will not be provided today, but a list of nearby restaurants is on page 9.*

INTRAOPERATIVE MONITORING, PART I

9:00 AM - 5:00 PM
Location: Bayview Ballroom, Level 2

Course Co-Directors: Jaime R. López, MD and Michael L. McGarvey, MD

Objectives:

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

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

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

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

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

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

BAEP Monitoring
Alan D. Legatt, MD, PhD

SEP Monitoring
Andres A. Gonzalez, MD

MEP Monitoring
Ronald G. Emerson, MD

EEG and Doppler Ultrasound Monitoring
Michael L. McGarvey, MD

Monitoring of Spinal Nerve Roots
Monica P. Islam, MD

Monitoring of Motor Cranial Nerves and Cranial Nerve Nuclei
Jaime R. López, MD

Monitoring of Peripheral Nerve Surgery
Bruno Gallo, MD

Anesthetic Management and IOM
Dileep R. Nair, MD

Case Presentations and Discussion
Panel

Wednesday, February 6, 2013

**Lunch will not be provided today but a list of nearby restaurants is on page 9.*

EP READING SESSION

7:00 AM - 8:30 AM
Location: Bayview Ballroom, Level 2

Course Director: Alan D. Legatt, MD, PhD

Objectives:

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

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

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

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

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

Visual Evoked Potentials (VEP)
Elayna O. Rubens, MD

Sensory Evoked Potentials (SEP)
Ronald G. Emerson, MD

NEONATAL EEG

7:00 AM - 8:30 AM
Location: Watson Island, Level 2

Course Director: Nicholas S. Abend, MD

Objectives:

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

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

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

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

Epidemiology and Electroencephalographic Characteristics
Dennis J. Dlugos, MD

Outcome
Nicholas S. Abend, MD

Management
Courtney Wusthoff, MD

Annual Courses Program — Wednesday, February 6

EEG COURSE: INTRACRANIAL EEG
9:00 AM - 5:00 PM
Location: Watson Island, Level 2

Course Directors: Gregory A. Worrell, MD, and Donald L. Schomer, MD

Objectives:

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

1. Discuss the appropriate indications for and limitations of intracranial EEG in patients with drug resistant epilepsy and recurrent focal seizures.

2. Design a comprehensive plan for invasive EEG monitoring, including electrode type, electrode placement, minimization of risks, and discussion of risks and potential benefits with patients.

3. Evaluate the results of intracranial EEG monitoring, including identification of the epileptogenic zone and of nearby eloquent cortex, to develop a surgical plan most likely to result in seizure freedom and minimize surgical risks.

4. Incorporate new EEG analysis techniques into presurgical evaluations, such as analysis of infra-slow and high frequency EEG activity, to improve identification of the epileptogenic zone and to gain new research insights into normal and pathological brain function.

Introduction and Historical Overview
Donald L. Schomer, MD

Overview of Electrodes
Donald L. Schomer, MD

Technical Aspects
Michael R. Sperling, MD

Recording and Analysis Techniques
Michael R. Sperling, MD and Gregory A. Worrell, MD

Stimulation Procedures
Tobias Loddenkemper, MD

Research Uses
Sydney Cash, MD, PhD

Case Demonstrations
Panel

Summary
Donald L. Schomer, MD

INTRAOPERATIVE MONITORING PART II
9:00 AM - 5:00 PM
Location: Baview Ballroom, Level 2

Course Directors: Jaime R. López, MD and Michael L. McGarvey, MD

Objectives:

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

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

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

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

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

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

Monitoring Cerebral and Spinal Endovascular Procedures
Viet Nguyen, MD

Electrocorticography During Pediatric Epilepsy Surgery
Douglas R. Nordli Jr., MD

Mapping of Eloquent Cortex
Mirela V. Simon, MD

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

Localization and Mapping of Central Motor and Sensory Pathways
Charles Yingling, PhD

Monitoring of Spinal D-Waves
Eva Ritzl, MD

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

Troubleshooting During IOM
Edward Gallo, R. EEG T, CNIM

Case Presentations and Discussion
Panel

EEG AND MEG SOURCE MODELING *(sponsored by Compumedics®)*
5:00 PM - 7:00 PM
Location: Bayview Ballroom, Level 2

CME credits are not available for this session.

Course Director: John S. Ebersole, MD

Objectives:

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

1. Discuss the indications, strengths, and limitations of EEG source modeling.

2. Describe the indications, strengths, and limitations of MEG source modeling.

3. Critically appraise and differentiate EEG and MEG source modeling techniques, as demonstrated in a case presentation format, to improve use of these complementary techniques in presurgical evaluations for drug resistant epilepsy.

Faculty:
John S. Ebersole, MD
Susan M. Ebersole, R.EEG T

Thursday, February 7, 2013

**Lunch will not be provided today, but a list of nearby restaurants is on page 9.*

EMG AND EEG TECHNOLOGY
7:00 AM - 8:30 AM
Location: Bayview Ballroom, Level 2

Course Director: Charles M. Epstein, MD

Objectives:

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

1. Describe the fundamental operation of neurophysiologic recording equipment, including differential amplifiers, common-mode noise rejection, grounds, and filters.

2. Explain the concepts of analog-to-digital conversion, aliasing and general frequency analysis.

3. Evaluate and select neurophysiologic equipment based on knowledge of appropriate technical specifications for clinical or research use.

BUSINESS IN CLINICAL NEUROPHYSIOLOGY
7:00 AM - 8:30 AM
Location: GrandBallroom, Level 3

Course Directors: Yafa Minazad, DO and Deborah Briggs, MD

Objectives:

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

1. Plan and develop an interdisciplinary team for neurophysiological practice in the neurophysiology laboratory, video-EEG monitoring unit, intensive care unit and operating room.

2. Apply business principles, quality assurance, and efficiency guidelines to neurophysiologic practice to improve clinical effectiveness and cost effectiveness of patient care.

3. Incorporate risk minimization strategies into neurophysiology practice, particularly in regards to invasive and remote monitoring.

Annual Courses Program — Thursday, February 7

ICU EEG
9:00 AM - 5:00 PM
Location: Grand Ballroom Salon F, Level 3

Course Directors: Lawrence J. Hirsch, MD and Cecil Hahn, MD, MPH

Objectives:

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

1. Discuss current guidelines and evaluate various practice models for ICU EEG monitoring to improve patient care.

2. Apply the revised ACNS nomenclature to ICU EEG recordings, to improve standardization of ICU EEG reports and communication between providers.

3. Recognize controversial EEG patterns in ICU patients with altered mental status, and formulate a rational plan for treatment based on these EEG patterns.

4. Develop a comprehensive ICU EEG monitoring program, including equipment selection, training of interdisciplinary staff, quality improvement, and risk management.

Overview of ICU EEG Monitoring
Nicholas S. Abend, MD

Current Guidelines for ICU EEG Monitoring
Susan T. Herman, MD
Courtney Wusthoff, MD

Nomenclature for ICU EEG Monitoring
Lawrence J. Hirsch, MD

The Ictal-Interictal Continuum
Suzette M. LaRoche, MD

Technical Aspects of ICU EEG Monitoring
Roy Sharma, R.ET, R.EPT

Postanoxic Coma and Seizures
Peter W. Kaplan, MB, FRCP

The Business of ICU EEG Monitoring
Stephen T. Hantus, MD

Quantitative EEG Techniques
Cecil Hahn, MD, MPH

Case Presentations - Neonatal, Pediatric and Adult Cases

EMG
9:00 AM - 12:00 PM
Location: Hibiscus Island, Level 3

Course Directors: Devon I. Rubin, MD and Francis O. Walker, MD

Objectives:

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

1. Apply basic and advanced EMG techniques to diagnose common entrapment neuropathies.

2. Incorporate advances in electrodiagnostic techniques and avoid technical pitfalls in evaluation of radiculopathies and plexopathies.

3. Recognize characteristic EMG patterns of neuropathic and myopathic disorders and interpret the clinical significance to improve neurologic diagnosis.

Advances in Assessment of Common Entrapment Neuropathies in the EMG Laboratory
Francis O. Walker, MD

Assessment of Radiculopathies and Plexopathies: EDX Approaches and Limitations
Daniel Menkes, MD

Advancing EMG Waveform Recognition Skills: Identifying Unknown Waveforms
Devon I. Rubin, MD

Annual Courses Program — Thursday, February 7

VIDEO-EEG

9:00 AM - 12:00 PM
Location: Bayview Ballroom, Level 2

Course Directors: William O. Tatum IV, DO and Tobias Loddenkemper, MD

Objectives:

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

1. Describe the technical requirements for optimal video-EEG monitoring in inpatient and outpatient settings.

2. Recognize the electroencephalographic and clinical features of seizures and nonepileptic events in adults and children commonly encountered in the video-EEG monitoring unit.

3. Translate EEG and video interpretations into clinical reports which accurately describe diagnosis, seizure localization, and implications for patient management, including candidacy for epilepsy surgery.

4. Determine the localization of seizure onsets based on combined video and intracranial EEG recordings.

Practical Technology of Video-EEG
John S. Ebersole, MD

Outcomes and Video-EEG Monitoring in the EMU
William O. Tatum IV, DO

Multi-Modal Pediatric Video-EEG Monitoring
Tobias Loddenkemper, MD

Ambulatory Video-EEG
Donald L. Schomer, MD

Coupling iEEG with Video: Lessons Learned
Michael R. Sperling, MD

Cases: Psychogenic, Physiologic or Epileptic
Panel

APPLIED AUTONOMIC NEUROPHYSIOLOGY

1:00 PM - 2:30 PM
Location: Bayview Ballroom, Level 2

Course Director: Arturo Leis, MD

Objectives:

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

1. Discuss the anatomy and physiology of the autonomic nervous system.

2. Explain the strengths and weaknesses of neurophysiologic tests used in assessment of the autonomic nervous system.

3. Develop a rational treatment plan for patients with autonomic disorders based on the results of specific autonomic tests, including skin biopsy

Clinical Application of Autonomic Neurophysiology for the Clinician
Jasvinder Chawla, MD

Technique and Value of Skin Biopsy in Small Fiber Autonomic Neuropathies
Mark A. Ross, MD

ELECTROPHYSIOLOGICAL AND PATHOLOGICAL FINDINGS IN NEUROMUSCULAR DISEASE: A CASE-BASED APPROACH

2:30 PM - 5:00 PM
Location: Bayview Ballroom, Level 2

Course Director: Elliot Dimberg, MD

Objectives:

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

1. Discuss the electrophysiological findings in neuromuscular disease, particularly muscle disease.

2. Recognize pathologic findings in neuromuscular disease, particularly muscle disease.

3. Design a stepwise evaluation of neuromuscular disorders, including clinical examination, electrodiagnostic techniques, and muscle and nerve biopsy.

Faculty:
Elliot Dimberg, MD
Suraj Ashok Muley, MD

Annual Courses Program — Thursday, February 7

BILLING AND CODING IN CLINICAL NEUROPHYSIOLOGY

5:00 PM - 7:00 PM

Location: Bayview Ballroom, Level 2

CME credits are not available for this session.

Course Directors: Yafa Minazad, DO and Deborah Briggs, MD

Objectives:

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

1. Describe the appropriate use of ICD 9 and CPT codes in neurophysiologic testing.

2. Establish and monitor billing processes and benchmarks for neurophysiologic practice.

3. Assess the impact of new health care models on practices based on traditional billing and coding.

Faculty:
Deborah Briggs, MD

Yafa Minazad, DO

Mark Nuwer, MD, PhD

SEIZURE DETECTION

5:00 PM - 7:00 PM

Location: Grand Ballroom, Salon F

CME credits are not available for this session.

Course Director: J. Chris Sackellares, MD

Objectives:

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

1. Explain the principles underlying seizure detection methods used in the epilepsy monitoring unit and intensive care unit.

2. Assess the pros and cons of high sensitivity versus high false-positive rates of various seizure detection algorithms in commonly encountered clinical settings, such as the epilepsy monitoring unit, intensive care unit.

3. Interpret long-term EEG recordings accurately using automated seizure detection algorithms.

Introduction
J. Chris Sackellares, MD

Do We Need Automated Seizure Detection? Man vs. Machine
Jonathan Halford, MD

Seizure Detection in the Epilepsy Monitoring Unit
Kevin M. Kelly, MD

Seizure Detection in the Intensive Care Unit
Suzette M. LaRoche, MD

What's This Pattern? Or Seizures, Periodic Discharges and the Ictal-Interictal Continuum
J. Chris Sackellares, MD
Deng-Shan Shiau, PhD

Annual Meeting Scientific Program — Friday, February 8

7:00 AM – 8:00 AM **Continental Breakfast**
Location: Grand Ballroom Salons A-E, Level 3

Poster Presentation
Location: Bal Harbor Island, Hibiscus Island, Fisher Island and Lummus Island, Level 3

A complete list of poster abstracts can be found on page 40.

Opening Ceremony

8:00 AM - 8:05 AM **WELCOME**
Location: Grand Ballroom Salons F-K, Level 3

8:05 AM - 8:45 AM **PRESIDENTIAL LECTURE: Continuous EEG Monitoring in the ICU: Defining a New Standard of Care**
Susan T. Herman, MD

Objectives:

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

1. Discuss the various practice models for continuous EEG monitoring in the intensive care unit.

2. Implement a quality assurance and improvement program for continuous EEG monitoring, to enable rapid integration of advances in monitoring techniques to patient care.

3. Propose clinical protocols and clinical research studies using continuous EEG monitoring to rapidly detect and treat brain injury in critically ill patients, and to assess the impact on patient outcomes.

8:45 AM - 9:20 AM **ROBERT S. SCHWAB AWARD PRESENTATION AND LECTURE: The Clinical Neurophysiology of Critical Illness Polyneuropathy and Myopathy**

The Robert S. Schwab Award is given to an individual for outstanding contributions to peripheral clinical neurophysiology research.

Presented to Charles F. Bolton, MD

Objectives:

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

1. Describe the patient populations at risk for critical illness polyneuropathy and myopathy.

2. Design appropriate electrodiagnostic investigation for diagnosis of critical illness polyneuropathy and myopathy.

3. Incorporate the results of electrodiagnostic testing into a comprehensive treatment plan and patient counseling regarding prognosis.

9:20 AM - 10:00 AM **PLENARY LECTURE: Interventional Neurophysiology for the 21st Century**
Jeffrey H. Gertsch, MD

Objectives:

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

1. Discuss recent advances in intraoperative neurophysiologic monitoring.

2. Incorporate recent advances in intraoperative monitoring into clinical practice.

3. Develop interdisciplinary teams for intraoperative neurophysiologic monitoring.

10:00 AM – 10:30 AM **Coffee Break – Visit Exhibits**
Location: Grand Ballroom Salons A-E, Level 3

Annual Meeting Scientific Program — Friday, February 8

Concurrent Sessions

10:30 AM - 12:30 PM

SYMPOSIUM: MEMORY AND LANGUAGE INSIGHTS FROM INVASIVE AND NON-INVASIVE STUDIES
Location: Grand Ballroom Salons F-K, Level 3
Chair: Dawn S. Eliashiv, MD

Objectives:

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

1. Discuss the technical aspects of techniques for localizing memory and language function.

2. Distinguish the strengths and weaknesses of the Wada test, functional imaging, magnetoencephalography, and intracranial neurophysiologic recordings for localization of memory and language.

3. Design a rational approach for assessment of memory and language in individual presurgical patients.

fMRI for Language and Memory Localization: Progress and Pitfalls
Susan Bookheimer, PhD

Atypical Language Representation in Epilepsy Surgery Candidates: The Role of MEG
Dawn S. Eliashiv, MD

Insights from Invasive Recordings on the Neuronal Mechanisms of Declarative Memory Formation in the Human Medial Temporal Lobe
Ueli Rutishauser, PhD

MEG: Will It Replace WADA?
Andrew C. Papanicolaou, PhD

10:30 AM - 12:30 PM

SYMPOSIUM: NEUROPHYSIOLOGIC EVALUATION OF PERIPHERAL AND CENTRAL SMALL FIBER NERVE PATHWAYS
Location: Watson Island, Level 2
Chair: Brent P. Goodman, MD

Objectives:

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

1. Explain the role of autonomic testing and skin biopsy in the evaluation of small fiber neuropathy.

2. Incorporate the use of contact heat-evoked potential stimulation (CHEPS) into the diagnostic evaluation of small fiber neuropathy.

3. Appraise the relative value of autonomic testing, skin biopsy, CHEPS, and MIBG myocardial scintigraphy in the evaluation of small fiber neuropathy.

Autonomic Testing and Skin Biopsy in Small Fiber Neuropathy
Brent P. Goodman, MD

Clinical Utility of CHEPS in Small Fiber Neuropathy
TBD

MIBG Myocardial Scintigraphy in Peripheral Evaluation of Small Fiber Autonomic Function
Osvaldo J.M. Nascimento, MD

Annual Meeting Scientific Program — Friday, February 8

10:30 AM - 12:30 PM **SYMPOSIUM: INTRAOPERATIVE NEUROPHYSIOLOGIC MONITORING DURING FUNCTIONAL NEUROSURGERY**
Location: Bayview Ballroom, Level 2
Chair: Jay L. Shils, PhD

Objectives:

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

1. Explain the neuroanatomy and neurophysiology underlying the mechanism of action of deep brain stimulation.

2. Design protocols for intraoperative neurophysiologic testing during placement of deep brain stimulation electrodes.

3. Communicate results of intraoperative neurophysiologic testing to the surgeon to optimize surgical outcomes.

Introduction
Jay L. Shils, PhD

The Basic Science of DBS: Physics and Physiology
Mark Stecker, MD, PhD

The Decision Interface: Surgeon, Neurophysiologist, and Making the Best Decisions During Surgery
Jeff E. Arle, MD, PhD

Intra-operative Methodology and Communication
Jay L. Shils, PhD

12:30 PM – 1:30 PM **Lunch & Visit Exhibits**
Location: Grand Ballroom Salons A-E, Level 3

Boxed lunches will be provided.

Professional Development Mentoring Program
Location: Biscayne Island, Level 3
If you have signed up as a Mentor or Mentee on your Meeting Registration Form, please pick up a boxed lunch in Grand Ballroom Salons A–E and then proceed to Biscayne Island for the Professional Development Mentoring Business Lunch.

Concurrent Sessions

1:30 PM - 3:30 PM **SYMPOSIUM: THE CHALLENGES OF NON-LESIONAL FOCAL EPILEPSY: WHAT CAN NEWER NON-INVASIVE NEUROPHYSIOLOGY TOOLS TELL US TODAY?**
Location: Grand Ballroom Salons F-K, Level 3
Co-Chairs: Richard C. Burgess, MD, PhD and Andreas Alexopoulos, MD

Objectives:

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

1. Describe the techniques and clinical utility of commonly employed methods for evaluation of nonlesional focal epilepsy, including MEG, functional MRI, infra-slow and high frequency EEG analysis, and MRI post-processing techniques.

2. Evaluate the utility of combined imaging and neurophysiologic techniques for localization of nonlesional focal epilepsy.

3. Develop efficient and cost-effective protocols for diagnostic evaluation of nonlesional focal epilepsy.

Tools for Investigating MRI-Negative Epilepsy: Current Practice and Future Challenges
Richard C. Burgess, MD, PhD

MEG Detection of Cryptogenic Pathology and Discrimination of Outcome in MRI-Negative Epilepsy Surgery
Robert C. Knowlton, MD, MSPH

High Frequency Oscillations and Infra-slow Activity: Two Useful Tools in Seizure Onset Localization
Pradeep Modur, MD

Utility of Voxel-Based Morphometric MRI Post-Processing in Detection of Subtle Cortical Dysplasia in MRI-Negative Epilepsy
Andreas Alexopoulos, MD

EEG-fMRI, A Powerful Tool to Localize Epileptic Foci
Jean Gotman, PhD

Panel Discussion

PROFESSIONAL DEVELOPMENT MENTORING PROGRAM

ACNS is happy to continue their newly-initiated program, the Professional Development Mentoring Program, on Friday, February 8, from 12:30 – 1:30 PM. If you signed up to be a Mentor or Mentee, please pick up a boxed lunch in Grand Ballroom Salons A – E and join us in Biscayne Island, Level 3!

Annual Meeting Scientific Program — Friday, February 8

1:30 PM - 3:30 PM

SYMPOSIUM: CONTROVERSIAL TOPICS IN EMG
Location: Watson Island, Level 2
Chair: Mark A. Ross, MD

Objectives:

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

1. Discuss the clinical utility of ultrasound in the diagnosis of neuromuscular disorders.

2. Assess the risk of bleeding and risk minimization strategies in patients who are anticoagulated who are referred for EMG.

3. Determine which patients with neuromuscular disorders require needle EMG studies and which can be diagnosed with nerve conduction studies alone.

Introduction
Mark A. Ross, MD

An EMG Lab Should Have Ultrasound Capability and Use It Regularly in EMG Practice
Francis O. Walker, MD
Jasvinder Chawla, MD

Fibrillation Potentials Are Not Necessary for the EMG Diagnosis of ALS
Shafu Ladha, MD
Mark A. Ross, MD

Anticoagulated Patients Need INR Before EMG and the Results Influence the Muscles Studied
Jasvinder Chawla, MD
Devon Rubin, MD

All Patients Undergoing Nerve Conduction Studies Must Have a Needle Exam
Arturo Leis, MD
Mark A. Ross, MD

1:30 PM - 3:30 PM

SYMPOSIUM: CHALLENGES IN INTRAOPERATIVE NEUROPHYSIOLOGIC MAPPING ALONG THE NEUROAXIS
Location: Bayview Ballroom, Level 2
Co-Chairs: Mirela V. Simon, MD and Eva K. Ritzl, MD

Objectives:

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

1. Design strategies to map eloquent cortex during supratentorial neurosurgical procedures.

2. Employ new techniques to map spinal cord function during surgical procedures.

3. Develop protocols to monitor the peripheral nervous system during surgery.

Supratentorial Mapping
Mirela V. Simon, MD
Eva K. Ritzl, MD

Brainstem Mapping
Jaime R. López, MD

Spinal Cord Mapping
Eva K. Ritzl, MD

Peripheral Nervous System Mapping
Thien Nguyen, MD

3:30 PM – 4:00 PM

Coffee Break & Visit Exhibits
Location: Salons A-E, Level 3

Concurrent Sessions

4:30 PM - 5:30 PM

SYMPOSIUM: NEUROMONITORING OF THE PEDIATRIC PATIENT
Location: Bayview Ballroom, Level 2
Chair: Jonathan Norton, PhD

Objectives:

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

1. Identify unique technical aspects of intraoperative neurophysiologic monitoring in the pediatric patient.

2. Employ optimal multimodality monitoring techniques during spine surgery in pediatric patients.

The Challenges of the Young Nervous System
Keith Aronyk, MD

Monitoring the Pediatric Spine
Charles Yingling, PhD

Non AIS Spine Surgery
Jonathan Norton, PhD

Annual Meeting Scientific Program — Friday, February 8 and Saturday, February 9

4:30 PM - 5:30PM

SYMPOSIUM: BRAIN STIMULATION
Location: Grand Ballroom Salons F-K, Level 3
Chair: Mark Hallett, MD

Objectives:

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

1. Evaluate brain stimulation as a therapeutic option for patients with drug-resistant epilepsy.

2. Discuss new advances in the utility of deep brain stimulation.

3. Describe the research, diagnostic and therapeutic applications of transcranial magnetic stimulation of the motor system.

Brain Stimulation for Epilepsy
Robert S. Fisher, MD, PhD

Deep Brain Stimulation
Bruno V. Gallo, MD

Transcranial Magnetic Stimulation of the Motor System
Mark Hallett, MD

5:30 PM - 6:45 PM

NEUROPHYS BOWL
Location: Grand Ballroom Salons F-K, Level 3
Co-Chairs: Lawrence J. Hirsch, MD and Mark A. Ross, MD

Come cheer on your colleagues as they test their knowledge of clinical neurophysiology!

6:45 PM – 8:00 PM

Welcome Reception – Visit Exhibits
Location: Grand Ballroom Salons A-E, Level 3

7:00 AM – 8:00 AM

Continental Breakfast
Location: Grand Ballroom Salons A-E, Level 3

Poster Presentation
Location: Bal Harbor Island, Hibiscus Island, Fisher Island and Lummus Island, Level 3

A complete list of poster abstracts can be found on page 40.

Opening Session

8:00 AM - 8:50 AM

PIERRE GLOOR AWARD PRESENTATION AND LECTURE: THALAMOCORTICAL RHYTHMS AND DYSRHYTHMIAS
The Pierre Gloor Award is given to an individual for outstanding contributions to central clinical neurophysiology research.

Presented to Rodolfo Llinas, MD, PhD

Location: Grand Ballroom Salon F, Level 3

Objectives:

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

1. Describe the normal neurophysiology of thalamic neurons and thalamocortical networks.

2. Explain the mechanisms and neurotransmitters implicated in normal thalamocortical rhythms and dysrhythmias.

3. Evaluate the role of thalamocortical rhythms and dysrhythmias in neurologic disorders, psychiatric disorders, and pain.

Annual Meeting Scientific Program — Saturday, February 9

8:50 AM - 9:10 AM

HERBERT H. JASPER AWARD PRESENTATION

The Herbert H. Jasper Award is given to an individual for a lifetime of outstanding contributions to the field of clinical neurophysiology including research, teaching and mentoring. It is analogous to a lifetime achievement award.

Presented to Richard P. Brenner, MD

TRAVEL FELLOWS RECOGNITION CEREMONY

The Travel Fellowship Award is presented to the most outstanding poster submitted by a Fellow who is the First Author. For poster abstracts please see page 40.

The Travel Fellowship Award Recipients are:
Brian Moseley, MD
Brian Peterson, MD
Christa Swisher, MD
Dragos A. Nita, MD, PhD
Lidia Moura, MD
Shennan Aibel Weiss, MD, PhD
Teresa Maria Montes de Oca Domingo, MD

Travel fellowships have been funded, in part, by a grant from the North American Chapter of the International Federation of Clinical Neurophysiology (IFCN).

9:10 AM - 10:00 AM

PLENARY LECTURE: SMALL WORLD NETWORKS

C.J. Stam, MD, PhD

Objectives:

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

1. Discuss the functional connectivity of the brain from a small world complex networks perspective.

2. Evaluate the role of network dynamics in normal brain functioning, neurological disorders, psychiatric disorders, and sleep.

3. Apply neurophysiologic and imaging techniques to study brain connectivity in patients with neurologic disorders.

10:00 AM – 10:30 AM

Coffee Break & Visit Exhibits

Location: Grand Ballroom Salons A-E, Level 3

Concurrent Sessions

10:30 AM - 12:30 PM

SYMPOSIUM: HIGH DENSITY EEG AND OPTICAL IMAGING: ADVANCES IN SEIZURE LOCALIZATION

Location: Grand Ballroom Salon F, Level 3
Co-Chairs: Ronald G. Emerson, MD and Catherine Schevon, MD

Objectives:

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

1. Describe standard techniques currently used to localize the epileptogenic zone.

2. Compare emerging techniques for localization of seizure onset, including gamma activity and optical imaging.

3. Explain how these emerging techniques can help to understand pathophysiology underlying seizure generation.

Finally, Some Progress
Ronald G. Emerson, MD

The Focus and the Penumbra: What Exactly is a Seizure and How Do We Localize It?
Andrew Trevelyan, D Phil, MB BCh

High Gamma Bursting: A Rosetta Stone for EEG Seizure Localization
Catherine Schevon, MD

Optical Imaging of Seizures and Epilepsy: From Zones to Networks
Paul Carney, MD

Annual Meeting Scientific Program — Saturday, February 9

10:30 AM - 12:30 PM **SYMPOSIUM: ADVANCES IN THE NEUROPHYSIOLOGICAL ASSESSMENT OF CONCUSSION**
Location: Bayview Ballroom, Level 2
Chair: Jonathan C. Edwards, MD

Objectives:

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

1. Diagnose patients with concussion following head injury based on clinical criteria.

2. Utilize clinical neurophysiology and imaging techniques as adjuncts to the clinical diagnosis of concussion.

3. Describe how clinical neurophysiology and imaging techniques can help to predict prognosis after concussion.

Concussion: Definition, Mechanism, Manifestations, and Importance
Jonathan C. Edwards, MD

Current Clinical Neurophysiology and Imaging in Concussion
Marc R. Nuwer, MD, PhD

Emerging Neurophysiological and Imaging Techniques
Sam Slobounov, PhD

10:30 AM - 12:30 PM **WORKSHOP ON NEUROMUSCULAR ULTRASOUND**
Location: Grand Ballroom Salons G-K, Level 3
Chair: Francis O. Walker, MD

Support Acknowledgement: Equipment for this session has been provided by Esaote USA.

Objectives:

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

1. Identify patients in whom neuromuscular ultrasound may aid in diagnosis.

2. Accurately perform, interpret and provide clinical correlation for neuromuscular ultrasound testing.

3. Incorporate skills in neuromuscular ultrasound into diagnostic algorithms for neuromuscular disorders.

12:30 PM – 1:30 PM **Lunch & Visit Exhibits**
Location: Grand Ballroom Salons A-E, Level 3

Boxed lunches will be provided.

Consortium of Clinical Neurophysiology Program Directors
Location: Watson Island, Level 2

Concurrent Sessions

1:30 PM - 3:30 PM **SYMPOSIUM: NEW DIRECTIONS FOR QUANTITATIVE EEG IN NEUROCRITICAL CARE**
Location: Grand Ballroom Salon F, Level 3
Chair: M. Brandon Westover, MD, PhD

Objectives:

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

1. Describe quantitative EEG techniques commonly used in the intensive care unit for detection of seizures and ischemia.

2. Expand the use of quantitative EEG techniques to other patient populations, such as monitoring sedation or degree of encephalopathy.

3. Discuss the potential benefits and risks of quantitative EEG in automated closed loop treatments.

Quantitative EEG Monitoring of Seizures
Mouhsin Shafi, MD

Quantitative EEG Monitoring of General Anesthesia and Sedation
Patrick Purdon, MD

EEG Monitoring and Closed-Loop Control of Burst Suppression
M. Brandon Westover, MD, PhD
ShiNung Ching, MD

QEEG Analysis of Encephalopathy
Jong Woo Lee, MD
Rani Sarkis, MD

Annual Meeting Scientific Program — Saturday, February 9, 2013

1:30 PM - 3:30 PM

SPECIAL INTEREST GROUP: SLEEP AND EPILEPSY; STRANGE BEDFELLOWS NO MORE
Location: Bayview Ballroom, Level 2
Chair: Erik K. St. Louis, MD

Objectives:

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

1. Diagnose nocturnal epileptic seizures and parasomnias based on clinical and EEG characteristics.

2. Develop protocols for combined EEG and polysomnography monitoring.

3. Counsel patients about the interrelations between sleep, seizures, and circadian rhythms.

Differential Diagnosis of Nocturnal Events
Mithri Junna, MD

Identifying Interictal and Ictal Epileptiform Activity Utilizing Video-EEG Polysomnography
Selim Benbadis, MD

Sleep, Circadian Periodicity, and Epilepsy
Erik K. St. Louis, MD

1:30 PM - 3:30 PM

SPECIAL INTEREST GROUP: EPILEPSY NETWORK
Location: Grand Ballroom Salons G-K, Level 3
Chair: Gabriel Martz, MD

Objectives:

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

1. Describe how malformations of cortical development interrupt normal cortical connections, and how these disconnections may promote epileptogenesis and affect cognitive functioning.

2. Summarize results of EEG and fMRI investigations of network dynamics in generalized epilepsy.

3. Explain how networks facilitate seizure propagation and secondary generalization.

Cortical Connections and Disconnections in Gray Matter Heterotopias
Bernard S. Chang, MD, MMSC

How Focal is Generalized Epilepsy
Jean Gotman, PhD

Circuit Factor Influencing Seizure Generalization
Edward Bertram, MD

3:30 PM - 4:00 PM

Coffee Break
Location: Grand Ballroom Foyer, Level 3

Concurrent Sessions

4:00 PM - 5:30 PM

WORKSHOP: EEG-VIDEO: EXPERT CONSENSUS
Location: Grand Ballroom Salon F, Level 3
Co-Chairs: William O. Tatum IV, DO and Jonathan J. Halford, MD

Objectives:

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

1. Describe how EEG interpretation can be improved using a computer-based expert consensus.

2. Recognize localizing, lateralizing, and misleading clinical features of epileptic seizures and nonepileptic events.

3. Identify common pitfalls in video-EEG monitoring.

Computer-Based Expert Consensus in EEG
Jonathan J. Halford, MD

EEG & Video Individual Expert vs. Group Analysis
William O. Tatum IV, DO
Selim R. Benbadis, MD
Jonathan C. Edwards, MD
Peter W. Kaplan, MB, FRCP

Annual Meeting Scientific Program — Saturday, February 9

4:00 PM - 5:30 PM

SPECIAL INTEREST GROUP: THE COMING OF AGE OF MAGNETOENCEPHALOGRAPHY
Location: Grand Ballroom Salons G-K, Level 3
Chair: Anto Bagić, MD

Objectives:

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

1. Identify the benefits and limitations of MEG source localization.

2. Appropriately incorporate MEG into presurgical evaluations for epilepsy surgery.

3. Appraise the utility of MEG for mapping eloquent cortex.

Seeking the Sources: Audacity of Dealing with Ill-Posted Problems of MEG and EEG Source Localization
Michael Wagner, PhD

Myths Meet the Evidence: Gleanings for Increasing the Credence of MEG in Modern Epileptology
Richard C. Burgess, MD, PhD

What Do You Mean What and How I Feel? Current Role of MEG in Brain Mapping
Michael E. Funke, MD, PhD

Quo Vadis Clinical MEG?
Anto Bagić, MD, PhD

4:00 PM - 5:30 PM

SPECIAL INTEREST GROUP: INTRAOPERATIVE MONITORING
Location: Bayview Ballroom, Level 2
Chair: Jaime R. López, MD

Objectives:

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

1. Incorporate current ACNS/AAN guidelines on intraoperative neurophysiologic monitoring into clinical practice.

2. Assess the qualifications and experience necessary for neurophysiologic personnel to perform and/or interpret intraoperative monitoring.

3. Describe methods to improve the quality of intraoperative monitoring and assess the impact of IOM on patient outcomes.

Welcome
Jaime R. López, MD

Review of AAN's Evidence-Based Guideline Update: Intraoperative Spinal Monitoring
Marc R. Nuwer, MD, PhD

Qualifications for Neurodiagnostic Personnel: Report of Inter-Society Workgroup
Ronald G. Emerson, MD

Open Forum Discussion
Moderator: Jaime R. López, MD

5:30 PM – 6:00 PM

ACNS Annual Business Meeting
Location: Grand Ballroom Salon F, Level 3
This meeting is open to all attendees, but only ACNS members may vote.

Annual Meeting Scientific Program — Sunday, February 10

7:30 AM – 8:00 AM **Continental Breakfast**
Location: Bayview Ballroom Foyer, Level 2

Concurrent Sessions

8:00 AM - 10:00 AM **SPECIAL INTEREST GROUP: ICU EEG MONITORING**
Location: Bayview Ballroom, Level 2
Co-Chairs: Nicholas S. Abend, MD, Cecil Hahn, MD, PhD and Suzette LaRoche, MD

Objectives:

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

1. Write effective ICU EEG reports, based on standardized nomenclature and essential EEG features.

2. Discuss current controversies in ICU EEG monitoring.

3. Describe current research efforts to improve the utility and impact on outcomes of ICU EEG monitoring.

Current Controversies and Advances: Pediatric
Nicholas S. Abend, MD

Current Controversies and Advances: Adult
Suzette M. LaRoche, MD

Critical Care EEG Monitoring Research Consortium – Clinical and Research Database
Suzette M. LaRoche, MD

Current Research Questions and Study Design
Cecil D. Hahn, MD

Ongoing Research Summaries
Panel

8:00 AM - 10:00 AM **WORKSHOP: ADVANCED NERVE CONDUCTION STUDIES**
Location: Dodge Island, Level 2
Chair: Morris A. Fisher, MD

Support Acknowledgement: Equipment for this session has been provided by Natus Neurology Inc, Magstim, and Cadwell.

Objectives:

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

1. Perform and interpret neurophysiologic tests for evaluation of small nerve fibers.

2. Utilize magnetic stimulation for evaluation of nerve function.

3. Employ blink reflexes and F waves in appropriate populations for diagnosis of neuromuscular disorders.

The *Advanced Nerve Conduction Workshop* will demonstrate and discuss important but uncommonly used nerve conduction techniques as well as clinically meaningful aspects of some more commonly used studies.

Evaluation of Small/Unmyelinated Nerve Fibers – Mixed and Cutaneous Silent Periods, Sympathetic Skin Responses
Arturo Leis, MD

Magnetic Stimulation and Nerve Conduction Studies Including H Reflexes
Paul Maccabee, MD

Blink Reflexes, F-Waves
Morris Fisher, MD

Annual Meeting Scientific Program — Sunday, February 10

8:00 AM - 10:00 AM

SPECIAL INTEREST GROUP: MODELS OF PROFESSIONAL CARE IN IOM: A COLLEGIAL DEBATE
Location: Watson Island, Level 2
Chair: Stanley Skinner, MD

Objectives:

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

1. Describe the current clinical models for provision of intraoperative monitoring, and the pros and cons of each model.

2. Explain how intraoperative monitoring can be incorporated into new models of healthcare.

3. Collaborate with practitioners utilizing different practice models to design comparative cost and clinical effectiveness studies.

Remote IOM: an Internalized Debate
Charles Yingling, PhD

IOM in a University Based Setting (How We Do It at UCLA)
Marc R. Nuwer, MD, PhD

Patient-Centered IOM: Is It Doable?
Stanley Skinner, MD

New Issues in IOM
Panel

10:00 AM – 10:15 AM

Coffee Break
Location: Bayview Ballroom Foyer, Level 2

Concurrent Sessions

10:15 AM - 12:15 PM

SYMPOSIUM: CONTINUOUS EEG MONITORING IN NEONATES: THE NEW ACNS GUIDELINES
Location: Dodge Island, Level 3
Chair: Courtney Wusthoff, MD

Objectives:

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

1. Identify neonatal populations at highest risk for neonatal seizures who should be referred for continuous EEG monitoring.

2. Optimize performance of neonatal monitoring, including ways to improve 24-hour availability, optimize sensitivity and specificity of seizure detection, and correlate EEG with clinical findings.

3. Utilize standardized EEG terminology to facilitate communication between providers.

Indications for Long-Term EEG Monitoring in Neonates
Robert R. Clancy, MD

Methods and Reporting in Neonatal EEG Monitoring
Renee A. Shellhaas, MD

Standardizing Neonatal EEG Background Terminology
Tammy Tsuchida, MD, PhD

Seizure or Not? Categorizing Rhythmic Patterns
Courtney Wusthoff, MD

Annual Meeting Scientific Program — Sunday, February 10

10:15 AM - 12:15 PM

SYMPOSIUM: ENCEPHALOPATHIES: ELECTROPHYSIOLOGIC, CLINICAL AND IMAGING CORRELATIONS
Location: Bayview Ballroom, Level 2
Chair: Peter W. Kaplan, MB, FRCP

Objectives:

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

1. Recognize EEG patterns commonly encountered in encephalopathic patients.

2. Interpret ictal and interictal patterns in patients with encephalopathy.

3. Utilize imaging techniques in conjunction with electrophysiology to facilitate understanding of the pathophysiology and generators of abnormal EEG patterns.

Introduction
Peter W. Kaplan, MB, FRCP

The Encephalopathy of Infection
G. Bryan Young, MD

Ictal and Epileptiform Elements in Encephalopathy
Frank W. Drislane, MD

Imaging Correlations and EEG Patterns in Encephalopathy
Peter W. Kaplan, MB, FRCP

10:15 AM - 12:15 PM

SYMPOSIUM: THE CURRENT STATE OF SAFETY IN THE EMU
Location: Watson Island, Level 2
Chair: Joseph F. Drazkowski, MD

Objectives:

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

1. Describe the types and incidence of injuries and adverse events in the epilepsy monitoring unit.

2. Develop strategies to minimize risk of injuries and adverse events during video-EEG monitoring.

3. Develop quality improvement programs to continuously monitor and improve safety in the EMU.

Overview of Safety in the Hospital and EMU
Joseph F. Drazkowski, MD

EMU Safety: What Does the Evidence Show?
Katherine Noe, MD, PhD

EMU Safety: Cardiac and Respiratory Issues and Emergencies
Lisa Bateman, MD

EMU Safety and Case Studies

Panel Discussion and Case Studies

Speaker Abstracts

Presidential Lecture

Please reference page 22 for lecture details.

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

Susan T. Herman, MD

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

Symposium: Memory and Language Insights from Invasive and Non-Invasive Studies

Please reference page 23 for session details.

Insights from Invasive Recording on the Neuronal Mechanisms of Declarative Memory Formation in the Human Medical Temporal Lobe

Ueli Rutishauser, PhD

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

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

Symposium: Intraoperative Neruophysiologic Monitoring During Functional Neurosurgery

Please reference page 24 for session details.

The Decision Interface: Surgeon, Neurophysiologist, and making the best decisions during surgery

Jeff E. Arle, MD, PhD

Intraoperative Neurophysiology or Monitoring (IONM) has become standard care in many surgical procedures, but the interaction between surgeon and neurophysiologist goes beyond simply performing the monitoring of neural function during a surgical case. There are important aspects of the interaction that allow the surgeon to make the best decisions even when information is sparse. Moreover, the relationship and the mutual understanding of what can be accomplished in the operative setting allows for the potential to develop new techniques, methods and therapies. Finally, a strong interaction between the IONM staff and the surgeon can result in the ability to troubleshoot devices that have already been implanted and need evaluation and potential repair, but beyond the means of the company representative or the simple replacement of the device. This talk gives examples and explores each of these important areas in the IONM repertoire, including decisionmaking in the use of MER for DBS surgery, the development of a new method for safer and more accurate placement of paddle electrodes in spinal cord stimulation, and the formal assessments needed to examine lead or wire breakage in many different systems.

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

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

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

In order for the neurophysiologist to perform these actions they need to not only have a detailed knowledge of the anatomy, basic physiology, and equipment, they also need to be familiar with the affects of the disease on the physiology, and the affects of the particular operative technique on the physiology. For example the Internal Globus Pallidum

(GPi) has different single unit firing characteristics when recording from a PD patient as compared to a dystonia patient as compared to a Tourette's patient; the activity level of the zona incerta varies with age in PD patients; minor vascular artifacts can affect the interpreted rate of firing in all areas.

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

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

Symposium: The Challenges of Non-Lesional Focal Epilepsy: What can Newer Non-Invasive Neurophysiology Tools tell us today?

Please reference page 24 for session details.

High Frequency Oscillations and Infraslow Activity: Two Useful Tools in Seizure Onset Localization

Pradeep Modur, MD, MS

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

Symposium: Controversial Topic in EMG

Please reference page 25 for session details.

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

Jasvinder Chawla, MD, Devon Rubin, MD

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

All Patients Undergoing Nerve Conduction Studies Must Have a Needle Exam

Arturo Leis, MD, Mark A. Ross, MD

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

In muscle disorders, needle EMG is essential to identify the characteristic "myopathic" recruitment (excessive number of small MUPs are recruited relative to the force of contraction), which is the hallmark of myopathic disorders. In neuromuscular junction disorders, single fiber EMG (increased jitter) remains the most sensitive test for MG (sensitivity > 95% positive in generalized and ocular MG when facial muscles examined). In root lesions, EMG is crucial to delineate the distribution of affected muscles, to localize the root(s) involved, and to provide information about the severity and chronicity of the radiculopathy. In anterior horn cell disorders, EMG is indispensable to document progression (early stages show localized denervation, "neurogenic"

recruitment, and fasciculation potentials, while later stages show widespread denervation and chronic neuropathic changes involving multiple limbs or tongue). In most peripheral nerve disorders, EMG is also a essential diagnostic tool. In diffuse polyneuropathy, denervation or chronic neuropathic changes are often limited to distal muscles (in conjunction with distal weakness and stocking-glove distribution sensory loss). In plexopathy, EMG abnormalities demarcate the distribution of affected muscles and confirm the presence of axonal loss.

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

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

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

Symposium: Challenges in Intraoperative Neurophysiologic Mapping along the Neuroaxis

Please reference page 25 for session details.

Supratentorial Mapping

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

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

Symposium: Brain Stimulation

Please reference page 26 for session details.

Brain Stimulation for Epilepsy

Robert S. Fisher, MD, PhD

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

Transcranial Magnetic Stimulation of the Motor System

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

Transcranial magnetic stimulation (TMS) has now been around for about two decades. It has been shown to be useful for limited purposes in the clinic, particularly in regard to central motor conduction velocity. In relation to therapeutics, while it is now an approved therapy for the treatment of depression, it has not had any major successes in movement disorders. TMS has been a particularly fruitful technique for physiological investigations of the motor system (Hallett, 2007).

Speaker Abstracts

TMS can examine many facets of cortical excitability and transiently activate or inhibit the motor system. TMS methods can also be used to assess and manipulate cortical plasticity. These methods will be illustrated by examining studies of patients with focal hand dystonia. Studies of motor excitability show a decrease of surround inhibition, a failure of inhibition of muscles not intended to be moved. Studies of brain excitability show deranged motor cortex plasticity, both an exaggerated response and a failure of homeostatic plasticity.

Symposium: New Directions for Quantitative EEG in Neurocritical Care

Please reference page 28 for session details.

EEG Monitoring and Closed-Loop Control of Burst Suppression
M. Brandon Westover, MD, PhD, ShiNung Ching, MD

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

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

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

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

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

Workshop: EEG-Video: Expert Consensus

Please reference page 29 for session details.

EEG-Video: Expert Consensus
William O. Tatum IV, DO, Johnathan J. Halford, MD, Selim R. Benbadis, MD, Jonathan C. Edwards, MD, Peter W. Kaplan, MB, FRCP

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

Part 1—Computer-based expert consensus in EEG

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

The audience will be polled with the ARS.

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

Part 2—EEG & video individual expert v group analysis

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

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

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

Special Interest Group: Models of Professional Care in IOM: A Collegial Debate

Please reference page 32 for session details.

Models of Professional Care in IOM: A Collegial Debate
Stanley Skinner, MD

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

model presumes the close collegial relationships with the surgeon/proceduralist or the anesthesiologist that naturally develop when the IONM physician/professional is routinely physically present in the operating room.

Symposium: Continuous EEG Monitoring in Neonates: The New ACNS Guidelines

Please reference page 32 for session details.

Indications for Long-Term EEG Monitoring in Neonates

Robert R. Clancy, MD

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

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

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

Methods and Reporting in Neonatal EEG Monitoring

Renee A. Shellhaas, MD

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

Standardizing Neonatal EEG Background Terminology

Tammy Tsuchida, MD, PhD

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

Seizure or Not? Categorizing Rhythmic Patterns

Courtney Wusthoff, MD

Clinical neurophysiologists face myriad abnormal patterns as they review continuous EEG recordings from ill neonates. While some patterns are easily distinguished as pathologic or seizures, there exists a daunting spectrum of rhythmic patterns that are not so straightforward. The new ACNS guideline tackles this grey area. An approach to neonatal EEG interpretation, including standardized terminology, is presented to facilitate systematic analysis of difficult rhythmic patterns. Clear criteria are offered to distinguish seizures from other rhythmic discharges. Consistent descriptors are suggested to allow better description of seizures and rhythmic patterns. Finally, a uniform definition of status epilepticus is proposed. Application of this framework to difficult tracings will illustrate its usefulness for the clinical neurophysiologist. Overall, the ACNS guideline on neonatal EEG offers benefits in clear communication between physicians, a structured system for teaching and training, and is of high utility for research applications.

Symposium: Encephalopathies: Electophysiologic, Clinical and Imaging Correlations

Please reference page 33 for session details.

Ictal and Epileptiform Elements in Encephalopathy

Frank W. Drislane, MD

Encephalopathies involve widespread brain dysfunction, especially cortical. The EEG usually shows widespread slowing and often, lower voltage activity. Some encephalopathies also exhibit the abnormal cortical activity of epileptiform discharges and even seizures. Seizures involve excitatory rhythmic electrical activity and also cause cortical dysfunction and clinical deficits; they may also lead to a postictal encephalopathy. Clinically, seizures and encephalopathies can each

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

Imaging Correlations and EEG Patterns in Encephalopathy
Peter W. Kaplan, MD, FRCP

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

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

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

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

1) Gloor P, Ball G, Schaul N (1977) Brain lesions that produce delta waves in the EEG. Neurology 27;326-333..

2) Sutter R, Stevens RD, Kaplan PW. Clinical and imaging correlates of EEG patterns in hospitalized patients with encephalopathy. J Neurol 2012 Epub Nov 30.

Symposium: The Current State of Safety in the EMU

Please reference page 33 for session details.

The Current State of Safety in the EMU
Joseph F. Drazkowski, MD, Katherine Noe, MD, PhD, Lisa Bateman, MD

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

Poster Abstracts—Friday, February 8

Posters will be on display from 6:45 AM - 1:30 PM and authors will be present between 7:00 AM - 8:00 AM for discussion.

The Poster Hall is located in the Fisher Island, Lummus Island, Hibiscus Island and Bal Harbour Island rooms on Level 3 of the Miami Marriott Biscayne Bay.

Poster numbers indicate the day of presentation (F=Friday, S=Saturday) and the board number where the poster will be displayed.

F1

A Study of Autonomic Dysfunction and Sympathetic Skin Response in Motor Neuron Disease

Wang Xinning, MD, Cui Liying, MD, Liu Mingsheng, MD, Guan Yuzhou, MD

Objectives: The aim of our study was to investigate the symptoms of autonomic dysfunction and SSR abnormality in MND patients.

Methods: Collect the clinical features of autonomic dysfunction among the patients as dullness or pruritus of the skin, parahidrosis, xerostomia, salivation, abnormal skin temperature, orthostatic hypotension, posture-related cardiac arrhythmia, mydriasis, ptosis or abnormal pupillary light reflex, diarrhea, constipation, voiding dysfunction and sexual dysfunction. SSR was performed in all the patients. The result of SSR was judged according to the normal range of our laboratory. Abnormality rate in MND patients was calculated. The relationship between clinical symptoms and SSR parameters were analyzed by statistical methods.

Results: In a total of 142 MND patients, the incidences of symptoms of autonomic dysfunction were as follows: dullness (53.5%), pruritus of the skin (15.5%), parahidrosis (10.6%), xerostomia (9.1%), salivation (2.1%), abnormal skin temperature (14.8%), orthostatic hypotension (2.1%), posture-related cardiac arrhythmia (0.7%), diarrhea (4.2%), constipation (16.2%), voiding dysfunction (9.9%) and sexual dysfunction (1.4%). Abnormal SSR was found in 51(35.9%) of the 142 cases, 12(8.5%) in palmar and 47(33.1%) in plantar. The features of abnormal SSR included delayed latency of palmar ($P<0.05$) and decreased amplitudes of both palmar and plantar compared with normal ranges ($P<0.01$ respectively). The group of patients with lumbosacral onset had higher abnormal rate of SSR than those of other onset sites. There was no significant correlation between clinical symptoms and abnommal SSR parameters.

Conclusion: Patients of MND can demonstrate autonomic dysfunction of skin, gland secretion, cardiovascular system and sphincters. Some patients show abnormal SSR with prolonged latency and decreased amplitude. The abnormalities of SSR are not related to clinical features of autonomic dysfunction. Patients with onset of lower limbs have a higher rate of abnormal SSR.

F2

Protracted Post-ictal Trismus : A Case Report

Abdorasool Janati, MD, Abdulrahman Mohammad Almalik, MD, Naif Saad ALGhasab, MD, Ghassab ALGhassab, MD, Muhammad Umair, MD

Introduction: Trismus is a motor disturbance of the trigeminal nerve , especially spasm of the masticatory muscles, resulting in difficulty in opening the mouth. It has a number of potential causes which range from the simple and non-progressive to those that are potentially life-threatening. In this paper we report for the first time the occurrence of trismus in the post ictal state.

Method: This was a case study which was conducted at King Khalid Hospital.

Results: After a series of generalized convulsive seizures the patient developed a sustained trismus in the postictal phase, lasting for three days.

Conclusion: A number of physiological and metabolic factors have been implicated in the termination of seizure activity and transition to post-ictal state by creating inhibitory signals. In our patient the trismus may have signified a partial failure of the afore-mentioned inhibitory mechanisms at the mesencephalic-pontine level , causing a disruption of projections of the mesencephalic trigeminal nucleus to the pontine nucleus, resulting in a state of hypertonicity in the latter.

F3

Electrographic Seizures in Critically Ill Children

Nicholas S. Abend, MD, Daniel Arndt, MD, Jessica L Carpenter, MD, Kevin E Chapman, MD, William B Gallentine, DO, Christopher C Giza, MD, Joshua L Goldstein, MD, Cecil D Hahn, MD, MPH, Jason T Lerner, MD, Tobias Loddenkemper, MD, Joyce H Matsumoto, MD, Kristin McBain, MS, Kendall B Nash, MD, Eric Payne, MD, Sarah M Sanchez, BA, Ivan Sanchez Fernandez, MD, Justine Shults, PhD, Korwyn Williams, MD, PhD, Amy Yang, BS, Dennis J Dlugos, MD

We aimed to describe the characteristics of electrographic seizures in critically ill children. Eleven hospitals each retrospectively enrolled 50 consecutive critically ill children (1 month to 21 years) who underwent EEG monitoring. We collected information on the following variables: age, gender, historical neurologic disorders including prior epilepsy and intellectual disability, acute neurologic disorder, clinical seizures prior to EEG monitoring, mental status at EEG onset, initial EEG background, and inter-ictal epileptiform discharges. 550 patients (one EEG study per patient) were included. EEG monitoring duration was < 24 hours in 50%, 24-48 hours in 23%, and >48 hours in 23%. Electrographic seizures occurred in 162 (29.5%). Electrographic status epilepticus occurred in 61 of 162 (37.7%) [continuous seizure lasting >30 minutes: 28 (45.9%), recurrent seizures >50% per hour: 31 (50.8%)]. Fifty-nine subjects (36.4%) had only subclinical seizures while a clinical correlate occurred with all seizures in 43 (26.5%) and some seizures in 55 (34.0%). Multivariable analysis showed that risk factors for electrographic seizures were: young age, clinical seizures prior to EEG

monitoring, abnormal initial EEG background, inter-ictal epileptiform discharges, and prior diagnosis of epilepsy. Our results provide the largest epidemiologic characterization of electrographic seizures in critically ill children.

F4

Improving ICU Research Recruitment: Lessons from the DETECT Study

Kristin McBain, MSc, Helena Frndova, Eric Payne, MD, Margaret Wilkinson, Roy Sharma, Judith Van Huyse, RN, Carter Snead, MD, Christopher Parshuram, MD, PhD, Jamie Hutchison, MD, Cecil D Hahn, MD, MPH

Recruitment of research subjects in the high-stress ICU environment is challenging. Although historical data can be used to estimate how many patients may be eligible for a study, many unforeseen factors can reduce recruitment. The prospective observational DETECT study is employing continuous EEG (cEEG) monitoring to screen for seizures among comatose critically-ill children. In its first year, the study encountered several recruitment challenges. By analyzing the CONSORT diagram we identified several barriers to recruitment and developed strategies to overcome them. Creation of an online screening dashboard allowed us to continually screen for eligible subjects in real time. Initially, 47% of eligible patients were not enrolled because parents/guardians were unavailable for consenting. Since cEEG represents minimal risk, a deferred consent approach was approved by our Research Ethics Board, permitting cEEG initiation even when parents/guardians were initially unavailable, resulting in successful enrollment of 10/11 additional subjects. Weekend screening permitted an additional 11/13 eligible subjects to be enrolled over 12 months. A creative, multipronged approach can successfully increase research recruitment in the challenging ICU environment.

F5

Features of PLEDs Stratifies Risk in the Ictal-Interictal Continuum

Stephen T. Hantus, MD, Christopher Newey, DO

Introduction: PLEDs are often found in association with acute structural lesions and may have risk-stratifying features. We looked at electrographic features of PLEDs on cEEG to identify predictors of electrographic seizures.

Methods: Retrospective review of 100 consecutive patients with PLEDs. PLEDs described based on electrographic features: triphasic morphology, sharply contoured, overlying fast frequencies, and/or rhythmicity (loss of inter-discharge interval lasting > 1 second). EEG seizures were defined as evolving in frequency, distribution, or morphology at >2 Hz for >10 seconds.

Results: Overall, electrographic seizures occurred in 54% of patients with PLEDs. PLEDs with sharply contoured morphology (n=45/71) were more likely to develop seizures (OR 3.85 (CI 1.52-9.68); p=0.0041). PLEDs with overlying fast (n=29/37) were also significantly likely to develop seizures (OR 5.51 (CI 2.17-13.98); p=0.0002). Rhythmicity (n=44/56) was most significant for predicting seizures (OR 12.47 (4.82-32.27), p<0.0001). The presence of triphasic morphology (n=9/29) had the lowest risk for seizures (OR 0.26 (CI 0.10-0.65); p=0.0041).

Conclusions: Determining seizure risk for patients with acute structural lesions may benefit from using the electrographic features of PLEDs. Sharply contoured morphology, overlying fast or rhythmicity showed progressively higher risk of seizures on cEEG, while triphasic morphology appeared to be protective against seizures.

F6

Current EEG Monitoring Practice in Critically Ill Children

Sarah M Sanchez, BA, Daniel Arndt, MD, Jessica L Carpenter, MD, Kevin E Chapman, MD, Dennis J Dlugos, MD, William B Gallentine, DO, Christopher C Giza, MD, Joshua L Goldstein, MD, Cecil D Hahn, MD MPH, Jason T Lerner, MD, Tobias Loddenkemper, MD, Joyce H Matsumoto, MD, Kristin McBain, MS, Kendall B Nash, MD, Eric Payne, MD, Ivan Sanchez Fernandez, MD, Justine Shults, PhD, Korwyn Williams, MD, PhD, Amy Yang, BS, Nicholas S. Abend, MD

We aimed to describe the clinical utilization of EEG monitoring in critically ill children. We retrospectively enrolled 550 consecutive critically ill children (1 month to 21 years) who underwent EEG monitoring (50 from each of 11 hospitals). EEG monitoring indications were: encephalopathy with possible seizures in 368 (67%), event characterization in 209 (38%), management of refractory status epilepticus in 62 (11%), and management of intracranial pressure in 16 (3%). Acute diagnoses were: traumatic brain injury in 61 (11%), cardiac arrest in 69 (13%), ECMO in 25 (5%), and therapeutic hypothermia management in 19 (3%). Monitoring lasted <12 hours in 16%, 12-24 hours in 34%, 24-48 hours in 23%, 48-72 hours in 8%, and >72 hours in 17%. The mean monitoring duration was longer in children who were comatose (41 hours) than those who were obtunded (32 hours) or had normal mental status (25 hours) (p<0.001) and in children with rather than without inter-ictal epileptiform discharges (40 versus 28 hours, p<0.001). Monitoring onset occurred outside standard hours (17:00-08:00) in 47%. Our study provides the first systematic assessment of the clinical use of EEG monitoring in critically ill children.

F7

Time Savings and Sensitivity of CSA in ICU EEG

**Lidia M V R Moura, MD, Marcus Ng, MD, FRCPC, Sandipan Pati, MD, Mouhsin M Shafi, MD, PhD, Andrew J Cole, MD, FRCPC, M. Brandon Westover, MD, PhD*

Objective: To evaluate the time savings and sensitivity achievable by using compressed spectral arrays (CSA) for screening ICU EEG recordings.

Background: Increasing patient volumes demand increased efficiency in reviewing ICU cEEG studies. CSA is often used, but its performance in clinical practice is unknown. We hypothesized that using CSA for screening studies would save time while providing good sensitivity compared with review of the raw data.

***Travel Fellowship Award Recipient:** The Travel Fellowship Award is given to the most outstanding poster submitted by a Fellow who is the First Author.

Methods: Three neurophysiologists (group 1) reviewed the first 30 minutes of each cEEG, then used CSA to guide subsequent review. Reviewers were allowed to view 120 seconds of raw EEG surrounding suspicious CSA segments. Two independent neurophysiologists (group 2) performed standard interpretation of all cEEGs. We recorded review times and, for group 2, detection and miss rates (vs group 2) for pathological patterns.

Results: Both groups reviewed 594 hours of cEEG. Average review times were: Group 1: 8 minutes, vs Group 2: 24 minutes ($t = 8.0302$, $df = 30$, $p<0.0001$). Sensitivity of CSA-guided review was: seizures 87,05% (195/224), PEDs 84,6% (11/13), RDA 62,5(5/8), focal slowing 76,1% (16/21), generalized slowing 96,2% (26/27); epileptiform discharges: 80% (16/20).

Conclusions: CSA-guided review reduces cEEG review time by 2/3 with modest reduction in sensitivity compared with exhaustive review.

F8

The Inter-Burst Interval Can be Modulated by Photic Stimulation

**Dragos A Nita, MD, PhD, Cecil D Hahn, MD, MPH, Mihai Moldovan, MD, PhD*

Background: The EEG pattern of burst-suppression (BS) reflects severe encephalopathy due to a variety of brain pathologies. Our aim was to explore whether the BS pattern is reactive to photic stimulation (PS) and whether BS responsiveness to PS is more likely to reflect the severity of encephalopathy than the baseline BS pattern.

Methods: Five consecutive critically ill children undergoing continuous EEG monitoring with BS at the onset of monitoring were included in this study, irrespective of the underlying etiology. One minute long trains of 1 Hz photic stimuli were applied and the influence of PS on inter-burst intervals (IBI) and burst duration (BD) was quantified and compared to IBI and BD during baseline epochs.

Results: PS consistently elicited bursts with less than 1 second latency and similar BD. At stimulation onset there was an increased bursting rate followed by a decreased bursting rate at stimulation offset. A mathematical model of cumulative increases in the threshold of subsequent burst generation followed by an exponential recovery can reproduce the IBI behaviour during PS.

Conclusions: IBI is modulated by PS. Further studies are needed to assess if BS reactivity is a biomarker that can assist with prognostication in critically ill children.

F9

Baseline EEG Pattern and Incidence of Seizures on Continuous ICU EEG Monitoring

**Christa Swisher, MD, Aatif M. Husain, MD*

Objective: To identify the probability of detecting non-convulsive seizures (NCS) based on the initial pattern seen in the first 20-30 minutes of continuous electroencephalographic (cEEG) monitoring.

Methods: CEEG monitoring reports from 243 adult patients were reviewed, assessing the baseline cEEG monitoring pattern in the first 20-30 minutes and the presence of seizures during the entire monitoring period. The baseline EEG patterns were classified into nine categories: seizures, periodic lateralized epileptiform discharges (PLEDs), generalized periodic epileptiform discharges (GPEDs), focal epileptiform discharges, burst suppression, focal slowing, diffuse slowing, triphasic waves and normal.
Results: Overall, 51 patients (21%) had NCS at any time during cEEG monitoring. Notably, 112 patients had diffuse slowing as the initial EEG pattern and none of these patients were noted to have seizures. Similarly, no patients with triphasic waves (n=3) developed seizures. Seizure rates were as follows: PLEDs (56%, n=9), burst suppression (50%, n=10), GPEDs (50%, n=2), normal (33%, n=3), focal epileptiform discharges (31%, n=35) and focal slowing (11%, n=46). Patients in the high-risk group (PLEDs, GPEDs, burst suppression and focal epileptiform discharges, n=56) were more likely to have seizures compared with the low risk group (diffuse slowing, focal slowing, triphasic waves and normal, n=164), odds ratio 16.9 ($p < 0.0001$).
Conclusion: Patients with diffuse slowing, focal slowing and triphasic waves seen on the baseline EEG recording are very unlikely to develop seizures on subsequent cEEG monitoring. This data can be used to decide how long to continue cEEG monitoring in patients based on their initial EEG findings.

F10

Brief Ictal Rhythmic Discharges (BIRDs) in the Critically Ill

Ji Yeoun Yoo, MD, Nicolas Gaspard, MD, PhD, Nishi Rampal, MD, Ognen A. C. Petroff, MD, Lawrence J. Hirsch, MD

Background: Brief ictal rhythmic discharges (BIRDs) have been described mainly in the neonates, and their significance is still unclear. We aimed to identify BIRDs in non-neonatal critically ill patients and explore their association with seizures and other EEG findings.

Methods: We screened our EEG database for patients with brief focal rhythmic discharges. BIRDs were defined as rhythmic discharges of theta or higher frequency lasting less than 10 seconds.

Results: Using these criteria, we retrospectively identified BIRDs in 10 out of 540 patients undergoing continuous EEG (cEEG) monitoring. Typical frequency and duration of BIRDs were 4-7 Hz and 1-3 seconds. Nine out of 10 patients had acute CNS injury (3 intracranial hemorrhage, 1 ischemic stroke, 2 subarachnoid hemorrhage, 2 metastatic tumor, 1 mitochondrial disease). Five had altered mental

***Travel Fellowship Award Recipient:** The Travel Fellowship Award is given to the most outstanding poster submitted by a Fellow who is the First Author.

status. Six out of 10 had seizures (clinical and/or on cEEG). In all 5 patients with seizures on cEEG, BIRDs preceded the seizure onset. All 5 patients had co-localizing lateralized periodic discharges (LPDs) and 3 also had co-localizing rhythmic delta activity (RDA).

Conclusion: The present data demonstrate the presence of BIRDs in non-neonatal population and its association with seizures. A larger prospective study is needed to better understand their clinical and prognostic significance.

F11

Cortical Stimulation Combined with White Matter Tractography.

Leonardo Bonilha, MD, PhD, Ekrem Kutluay, MD, Steven S Glazier, MD, Gabriel Martz, MD, Jonathan C. Edwards, MD

The identification of eloquent cerebral cortex prior to epilepsy surgery traditionally relies on intracranial cortical stimulation to map function to brain areas that should be avoided during the resection of epileptogenic tissue. Even though intracranial cortical stimulation can identify the functional anatomy of the cerebral cortex, the excision of the epileptogenic tissue often encompasses subcortical regions underlying the lesion. We suggest that cortical stimulation should be combined with anatomical mapping of white matter pathways in order to avoid disconnection of eloquent cortex. Recent advances in diffusion MRI can enable white matter tractography to be performed in a clinically feasible matter. We argue that increased anatomical precision of pre-surgical mapping can be achieved by combining white matter tractography with direct cortical stimulation. We report a case of a patient with a dorsolateral frontal focal cortical dysplasia in close anatomical proximity with the somatosensory cortex. White matter tractography enabled the determination of the pathways traversed by the pre-central cortex underlying the cortical dysplasia. The resection was then tailored to avoid eloquent cortical areas defined by intracranial cortical mapping, and also to avoid the fibers arising from the eloquent cortex. Post-surgical seizure freedom was achieved with no functional somatosensory impairment.

F12

Power Spectral Density of Scalp and Subdural EEG – Beta and Gamma Bands

Hitten P Zaveri, PhD, Ognen A Petroff, MD, Irina I Goncharova, PhD

Background: The first studies recording both scalp and cortical EEG noted a significant loss of EEG power and fidelity in the scalp recordings. Subsequent work focused on the loss of fidelity of epileptiform activity and evoked potentials.

Methods: Twenty patients (age 18–55) from the Yale Comprehensive Epilepsy Center undergoing intracranial EEG monitoring for surgical evaluation were recruited. EEG was recorded simultaneously from the scalp and subjacent subdural electrodes (Clin Neurophysiol 2010;121:311-317). The power spectral density (millivolts2) of artifact-free EEG segments was obtained for each electrode contact and the ratio of scalp electrode to subjacent subdural electrode signal power was calculated between 0.1 and 80 Hz in 0.5 Hz increments.

Results: The ratio of scalp to intracranial median power spectral density in the beta (13 - 25 Hz) frequency band was 0.063 (95%CI 0.060 - 0.064) and 0.057 (95%CI 0.055 - 0.081) for the gamma frequencies (25 - 55 Hz). The scalp to intracranial ratio increased linearly with frequency from 40 to 80 Hz.

Conclusion: Extra-cranial signals and recording system noise may account for the relative increase in scalp EEG power spectral density above 40 Hz and would confound measurement of the faster gamma frequencies using scalp EEG.

F13

Evolving Cyclicity on Quantitative EEG: A New Form of NCSE

Asma Zakaria, MD, Nishi Rampal, MD

Two middle aged women were transferred to the hospital with a diagnosis of status epilepticus and were placed on cEEG monitoring. Both patients had a history of seizures and were non-compliant with medications prior to admission. Initial EEG revealed a theta background which was abruptly interrupted by a cyclic pattern of generalized rhythmic delta activity. There was a clear onset and offset to these cycles but no evolution or spread which would characterize them as ictal. Twenty-four hour cEEG trends revealed a striking rhythmic pattern to these cycles which occurred in clusters, evolved in frequency and amplitude and then broke down in a pattern reminiscent of a single epileptic seizure. Both patients were treated aggressively for these events, and resolution of the cycles was associated with clinical improvement and subsequent extubation. Although each individual event did not meet ictal criteria, we propose that clusters of evolving generalized rhythmic delta activity in comatose patients with prior status epilepticus lies on the continuum of non-convulsive status epilepticus, are ictal, and should be treated as such.

F14

QEEG Analysis Reveals Changes Evoked by Intranasal Midazolam in Intracranial Recordings

Bin Tu, MD, PhD, Derek J Chong, MD, MSc, Lawrence J. Hirsch, MD

Intranasal application of midazolam is a convenient and effect way for immediate seizure control. EEG changes induced by intranasal midazolam application have not been previously reported. In this study, we obtained scalp EEGs (n=5) and intracranial EEGs (n=2) before, during, and after intranasal 3 mg midazolam application. EEG changes were demonstrated using quantitative EEG (QEEG) values, which include alpha, beta, delta, and theta powers and aEEG. Significant increase in beta power was seen in both cases of intracranial EEG recordings starting 5 and 6 min after application, peak at 11 and 27 min. Other QEEG values did not significantly change. No significant changes were seen in Scalp recordings. These results suggested that it is possible to evaluate effects of intranasal midazolam on brain functions using intracranial EEGs. Further studies are proposed to investigate more intracranial stud-

ies, and also to improve scalp EEG quality, in order to better show QEEG changes. We believe QEEG is a useful tool to evaluate time course of subtle changes in cerebral rhythms after nasal midazolam application.

F15
Slow Frequency Components of EEG Epileptiform Transients
Fumisuke Matsuo, MD

EEG polygraphic recognition of focal interictal epileptiform transients (FIET) predictive of epilepsy (spike or sharp waves: SSW) has depended on high frequency components. Wicket spikes (WS) often mimic SSW, and differentiation between SSW and atypical WS (aWS) challenges clinicians. When applied to SSW analysis, PGCO (polygraphic channel overlay) could improve detection of slow frequency components (Matsuo, 2012 AES Annual Meeting, www.aesnet.org). Mixed 121 FIET (72 SSW and 49 aWS) were blindly ranked by visual inspection in multi-channel polygraphic montages. Ranked FIET were segmented into 11 tiers of succeeding 11 FIET to construct 11-by-11 grid. PGCO was generated by manually superimposing high contrast digital display image. Display gain was controlled in common average reference derivations with frequency window and temporal resolution set at 0.16 – 70 Hz and 150 mm/s, respectively. Each PGCO replaced corresponding FIET on grid and each tier was examined as group. Lower tiers of FIET grid consisted of increasing numbers of aWS. When slow frequency components were examined in PGCO, 26 (21 %) of FIET classifications were questioned. PGCO also facilitated comparison of FIET amplitude, compromised in conventional multi-channel polygraphic display. It is suggested that PGCO can assist differentiation between SSW and WS.

F16
Periodic Complexes at 2-4 Second Intervals in EEE
Joseph W McSherry, MD, PhD

Among the periodic complexes with relatively specific disease correlates, periodic transients at 2-4 second intervals are usually associated with Herpes Simplex Encephalitis. We recently had EEGs of two patients ultimately diagnosed with Eastern Equine Encephalitis. In one patient periodic complexes in the left temporal area occurred at 4 second intervals. Remarkably the right temporal area showed complexes at 3 second intervals. Subsequent monitoring for nonconvulsive status epilepticus revealed several other patterns. The second patient showed complexes at 2-4 second intervals, occasionally with PLEDS at 1 second intervals. During subsequent monitoring complexes at 2-4 second intervals occur for periods, mixing with other periodicity at other times. Key distinctions between traditional periodic complexes* of HSE and those we saw in EEE are the impersistence of the fixed periodicity on long term monitoring and the amplitude. In the usual 20-30 minute sample a periodic complex at fixed 3 or 4 second intervals may be confusing. The amplitude, however is usually 100-500uV in HSE and about 30uV on the very low voltage background in these two cases of EEE. *p554 in Niedermeyer's Electroencephalography, Sixth Edition, Schomer, DL and Lopes da Silva, FH eds Wolters Kluwer Lippincott Williams & Wilkins, 2011

F17
EEG Scalp Potential Computer Simulation
Steven Tobochnik, Ellie Pavlick, Mercedes P. Jacobson, MD, Camilo A. Gutierrez, MD

An understanding of spike fields is critical for accurate interpretation of the EEG. We developed a computer simulation tool that takes a user-defined scalp potential distribution as input and produces the associated EEG spike-wave complex in longitudinal bipolar, transverse bipolar, and referential montages simultaneously. Users choose single or multiple foci of maximum potential on a 2-dimensional electrode map to create EEG spikes with fields of variable complexity on an organized user-adjustable background. Distances between electrodes were determined by their coordinates in 3-dimensional space, and used to calculate normalized voltages that spread according to an exponential decay function. The length-constant used for the decay function can be adjusted by the user to manipulate the scalp potential spread and size of the EEG spike field. Using this simplified model, the simulation successfully translates a scalp potential input into the expected EEG. This simulation would be useful both as a teaching tool and for interpretation of EEG spikes with complex fields.

F18
Automatic Spike Detection with Patient Specific Templates
Hannes Perko, Martin Weinkopf, Manfred Hartmann, Gerhard Gritsch, Tilmann Kluge, PhD, Franz Fuerbass

We present a novel method in the context of epilepsy diagnosis that automatically detects epileptic spikes in the electroencephalogram (EEG). In a first step our new approach detects spikes based on a non-restrictive definition of their morphology. The detected spikes are then subject to a hierarchical clustering. Representative group averages are presented to the user who can select groups of interest as templates which are used to refine the detection. In a second step, we derive a statistical description of the selected spikes and utilize it in a hypothesis testing. In contrast to the first step, all available EEG channels are included in order to detect spikes featuring a similar field as the selected groups. With this approach we were able to reject artifacts which are distributed arbitrarily over electrodes. Our results were obtained from the EEG of three patients with recording durations up to 4 hours and containing up to 128 spikes, which were marked by experienced EEG experts. On average a sensitivity of 69% with a false alarm rate of only 0.37 false alarms per minutes was achieved. Compared to the results only using step one, we significantly increased the sensitivity by 16% with constant false alarm rate.

F19

Ictal Catatonia Associated with Segmental Catalepsy

Abdorasool Janati, MD, Naif S Alghasab, MD, Abdulrahman Mohammad Almalik, MD, Ghassab Alghassab, MD

Introduction: Non-convulsive status epilepticus (NCSE) is an epileptic condition lasting more than 30 minutes in which continuous or recurrent seizure activity on the electroencephalogram (EEG) is associated with diverse clinical symptoms including alteration of mental state, abnormal behavior, perceptual disturbances or altered consciousness.

Methods: This was a case study conducted at King Khalid Hospital.

Results: Our patient presented with catatonia and segmental catalepsy associated with non-convulsive status epilepticus.

Conclusion: The simultaneous occurrence of non-convulsive status epilepticus and catatonia/catalepsy in our patient suggests a common pathophysiological mechanism. Furthermore, our data suggest that dysfunction of the posterior areas of the brain may be responsible for catalepsy.

F20

Generalized Epileptiform Discharges with Pregabalin

Michael Mendoza, MD, Ilya Bragin, MD, Andrew Bragdon, MD

Pregabalin is used to treat neuropathic pain, postherpetic neuralgia, partial seizures and restless leg syndrome (RLS). It acts on the alpha-2delta subunit of presynaptic voltage-gated calcium channels and is cleared almost entirely by the kidneys. Rarely, myoclonus and seizures have been reported in patients treated with pregabalin for partial seizures or neuropathic pain. To our knowledge, there has been no report of pregabalin-induced epileptiform discharges in a patient with end-stage renal disease (ESRD) being treated for RLS . A 56-year-old, non-epileptic, white male with ESRD (GFR 5 mL/min) on peritoneal dialysis was started on Pregabalin 75mg twice daily for RLS. He presented 5 days later with stuttering speech, myoclonus and ataxia. He had similar, but milder symptoms previously on gabapentin. The night of admission he had a generalized tonic-clonic seizure. The next day, an EEG showed generalized, bilaterally symmetrical spike-wave discharges including epileptiform K-complexes. Pregabalin was discontinued, he underwent peritoneal dialysis, and the myoclonus resolved by the next day. An EEG 5 weeks later showed complete resolution of the epileptiform discharges. Pregabalin may induce generalized epileptiform discharges, myoclonus and seizures even in the absence of epilepsy and independent of the indication for gabapentin. Renal failure likely increases the risk.

F21

Ictal Asystole vs. Convulsive Syncope

Roland Hamilton, MD, Jonathan Edwards, MD, Nolan Williams, MD, Jay Madey, MD

Ictal asystole is seizure-induced activation of the autonomic nervous system, which adversely affects cardiac innervation, leading to bradycardia and potentially lethal asystole. It is believed to be a contributing factor for sudden unexpected death in epilepsy. Convulsive syncope occurs when an individual experiences loss of consciousness (not related to epilepsy) followed by brief convulsions. The most common etiologies of convulsive syncope are cardiac arrhythmias and neurally mediated reflex. Distinguishing ictal asystole from convulsive syncope can be quite challenging for healthcare providers. Described here is a patient with a history of complex partial seizures presenting for medical evaluation of "syncope vs. seizure". Patient had multiple episodes of asystole which ultimately resulted in transcutaneous pacing and consideration of a cardiac pacemaker. Video electroencephalogram recording revealed seizure activity preceding his asystole suggesting ictal asystole as a possible etiology. However, the patient also had syncope with convulsion with no epileptic correlation on electroencephalogram, supporting the diagnosis of convulsive syncope. The medical evaluation of ictal asystole vs convulsive syncope will be reviewed, as well as the importance of making a timely and accurate diagnosis, to ensure that patients receive appropriate treatment for these potentially life threatening conditions.

F22

Episodic Low Amplitude Events: Outcome Implications

Jeffrey Britton, MD, Amy Z Crepeau, MD, Elson Lee So, MD

Background: Episodic low amplitude events (ELAEs) in coma have been reported to be associated with poor outcomes. We found ELAEs to be common in therapeutic hypothermia (TH) after cardiac arrest (CA), but the prognostic implication in this group of patients is unclear. We compared the prognostic value of ELAEs by comparing the seizure and mortality rates in TH and non-TH cohorts.

Methods: Patients with ELAEs were identified through query of our EEG report system and our TH database.

Results: 42 records with ELAEs were identified: 28 in the TH and 14 in the non-TH group. All TH and six non-TH patients were receiving sedation during the recording in which ELAEs were reported. Among TH patients, one had seizures, 3 (11%) died. Among non-TH patients, six had seizures, 5 (36%) died. There was a significantly higher proportion of seizures among non-TH patients ($p = 0.0054$), but no difference in mortality.

Conclusions: ELAEs have been described as being associated with a poor prognosis in comatose patients, but we did not reach the same conclusions. ELAEs were common in TH patients. Among non-TH patients identified, there was a higher proportion of seizures, but mortality was lower than previously reported.

F23

Temporal Lobe Epilepsy and POTS in Temporal Lobe Cavernoma

Michael Mendoza, MD, Hyun Joo Sophie Cho, MD, Robert L. Beach, MD PhD

Postural orthostatic tachycardia syndrome (POTS) is a disorder of orthostatic intolerance mostly affecting young female patients. We report the first case of coexistence of POTS and temporal lobe epilepsy in the setting of temporal lobe cavernous hemangioma. A 22 year-old female presented to the cardiology service with recurrent chest discomfort, nausea, diaphoresis, hot flash and dizziness on standing that started 15 months after giving birth. Comprehensive cardiac work-up including head-up tilt test confirmed POTS. She responded minimally to fludrocortisone and midodrine. At age of 28, she developed different types of episodes which comprised of few seconds of perseveration, blank stare and automatism which was associated with loss of consciousness at times preceded by hot flash, nausea and epigastric warm sensation. Video EEG revealed rhythmic sharp delta activity over the left sphenoidal and subtemporal electrodes followed by polymorphic slowing that corresponded to clinical seizures. MRI of the brain revealed a left medial anterior temporal cavernous hemangioma. She continued to have seizures due to poor tolerance to antiepileptic medication; therefore surgical removal of the hemangioma was planned. Temporal lobe epilepsy and POTS can present similar paroxysmal clinical events. It is important to differentiate these syndromes to offer appropriate therapy.

F24

Physician Discussion Prior to Electromyography

Greg Thaera, MD, Amy Nielsen, DO, Srijana Zarkou, MD, Ales Hlubocky, MD, Mark A. Ross, MD

Background: Patients are often apprehensive about nerve conduction studies (NCS) and electromyography (EMG). At our institution, we give a brochure to patients before testing. However, patients expressed that face-to-face time with a physician before these studies could be helpful. The aim of this study is determine whether physician discussion prior to NCS/EMG affects their tolerability.

Methods: Patients undergoing NCS/EMG in May 2011 were randomized to physician face-to-face visit with brochure versus brochure alone. The physician discussed the tests, allowing time for patient questions. Following testing, patients rated experience with NCS/EMG on an ordinal scale. Patients in the study group also rated the helpfulness of physician discussion prior to testing. Statistical analysis was performed using Fisher's exact test.

Results: We enrolled 51 study and 53 control patients (n=102). 9% of study patients and 8% of controls found NCS unpleasant (p=0.73). Study patients more often reported EMG as better than expected (p=0.02). 94% found physician interaction helpful.

Conclusions: Face-to-face discussion with a physician prior to NCS/EMG is helpful for patients. The EMG was viewed to be a more positive experience in the study group. Electromyographers should consider addressing patient questions and concerns prior to the needle examination to optimize patient comfort.

F25

Surgery for Catastrophic Epilepsy (CE) Caused by Malformations of Cortical Development (MCDs) in Infants Younger than 6 Months of Age

Pramote Laoprasert, MD, Andrew White, MD, Brent O'Neill, MD, Michael Handler, MD

Object: To study efficacy of epilepsy surgery (ES) in infants younger than 6 months with CE caused by MCDs.

Methods: We retrospectively reviewed 14 infants with MCDs ages between 1 week and 6 months (median 2 months) who underwent ES for CE.

Results: The median age at surgery was 2 months. MCDs included 8 cortical dysplasia (CD), 5 hemimegalencephaly and 1 tuberous sclerosis. Hemisperectomy was performed in 9 and multilobar and focal resection in 5 patients. At median follow up of 70 months, 64% had seizure freedom (SF). SF was noted in hemispherectomy, focal and multilobar resection in 78%, 50% and 33% respectively. SF was noted in 83% of right hemispheric lesion (HL) and 50% in left HL. 14% of patients had normal development. 43% and 82% of patients with history of infantile spasms (IS) or Ohtahara syndrome (OS) and patients with no history of IS or OS had SF respectively.

Conclusions: Epilepsy surgery is effective in infants younger than 6 months with CE. Patients with hemispherectomy had the highest rate of SF. Most patient had DD despite having SF. Patients with right HL had a higher rate of SF. History of IS and OS is a poor prognostic factor.

F26

LPD Modifiers and Seizure Risk in Critical Care Patients.

Louis Manganas, MD, PhD, Nicolas Gaspard, MD, PhD, Nlshi Rampal, MD, Ognen A Petroff, MD, Lawrence J. Hirsch, MD

Objective: The purpose of this study was to analyze the risk of seizures in a critical care population with lateralized periodic discharges (LPDs) with and without specific modifiers.

Methods: Patients from the Critical Care EEG database at Yale with LPDs were selected for the following modifiers: Rhythmic activity (R), Fast activity (F), Rhythmic and Fast activity (FR) and Lateralized Rhythmic Delta Activity (LRDA).

Results: Our findings show the probability of seizures (%) in patients with LPDs alone was 48%, (12/25), versus 83% (15/18) in patients with LPDs plus a modifier.

Conclusion: These preliminary results suggest that critical care patients with LPDs plus a modifier, carry a higher risk of developing seizures when compared to patients with LPDs alone.

F27

Validation of Magnetoencephalography (MEG) as Presurgical Evaluaiton Tool in Pediatric Patients With Localization-Related Intractable Epilepsy.

Helen Barkan, MD, PhD

Objective: Magnetoencephalography is employed increasingly for interictal source localization, however remains an expensive clinical modality, unvalidated due to limited available data, especially in pediatric surgical epilepsy patients. We compiled an unprecendented series of 59 pediatric surgical epilepsy patients in whom MEG was used to aid decision-making as to feasibility of surgery, and to guide intracranial implant placement. We attempt to validate its use.

Methods: Retrospective electronic chart analysis of patient data from the MEG database was performed by a team of Epileptologists and Neuropsychologists. Pre-surgical MEG reports, MEG images/composites, and intracranial video-electrocorticography reports were examined for concordance of location of "primary" and "secondary" interictal abnormalities and ictal onsets, with the criteria of hemispheric lateralization, lobar localization, gyral focal localization, judged by an expert. Patients were stratified into "lesional" (by MRI) and "nonlesional". Seizure outcomes and cognitive-functional outcomes were reconstructed from office notes and post-surgical neuropsychological evaluations.

Results: This preliminary analysis suggests that MEG is a valid pre-surgical evaluation tool in pediatric epilepsy surgery, that yields concordant findings with intracranial EEG, and allows for confident implant planning, and for improved localization and outcomes, particularly in "lesional" cases. IRB submitted to two institutions pending expedited approval

F28

Ictal QTc Changes are Not Associated with Hypoxemia

**Brian D Moseley, MD, Jeffrey W Britton, MD*

Introduction: It was recently reported that peri-ictal QTc prolongation is associated with hypoxemia, suggesting it may be a factor in sudden unexpected death in epilepsy (Seyal et al, 2011). Accordingly, we evaluated for associations between peri-ictal cardiac repolarization abnormalities and hypoxemia.

Methods: Patients evaluated in our epilepsy monitoring units were prospectively recruited. Peri-ictal oxygen saturation was recorded utilizing digital pulse oximeters. Ictal QTc values were calculated using the Bazett formula.

Results: Fifty eight seizures from 29 patients were analyzed. There was no significant difference between the minimum (396+/-47.8 versus 388+/-56.2 ms, p=0.54) and maximum (451+/-54.3 versus 450+/-55.8 ms, p=0.97) QTc values in seizures with and without peri-ictal hypoxemia. Seizures with peri-ictal hypoxemia were not more likely to be associated with QTc lengthening >=60 ms (3/18 with hypoxemia versus 6/40 without, p=1), clinically significant QTc prolongation (2/18 with hypoxemia versus 6/40 without, p=1), marked QT prolongation >=500 ms (3/18 with hypoxemia versus 8/40 without, p=1), QTc shortening >=60 ms (0/18 with hypoxemia versus 7/40 without, p=0.087), or markedly short QT intervals <=340 ms (1/18 with hypoxemia, 3/40 without, p=1).

Conclusions: Peri-ictal cardiac repolarization abnormalities are not associated with hypoxemia. This suggests factors other than hypoxia result in ictal cardiac repolarization abnormalities.

F29

Consistent Localization in a Case of Ictal Whislting

Usman Moghal, MD, Evren Burakgazi-Dalkilic, MD

Ictal whistling is a rare yet interesting automatism that has not been well localized. A handful of cases have been reported in literature localizing to the frontal and temporal lobes. We describe a patient with complex partial seizures of two different types, one of which includes ictal whistling, consistently localizing to the left posterior temporal and occipital lobes. We present a 28 year old female with a history of lupus and complex partial seizures since age eleven. The seizures did not consist of an aura and began with prominent whistling and loss of consciousness. Although there appeared to be an association with her menstrual cycle, her seizure frequency was minimal with keppra, vimpat, and phenobarbital. Whistling is complex, requiring oral, perioral and respiratory muscles. Complicated neuronal networks involving the inferior Rolandic cortex, cingulate cortex, basal ganglia, amygdala, thalamus, and cerebellum have been shown to be involved using functional imaging. As can be concluded from previous case reports and our patient, ictal whistling is a rare and interesting ictal phenomenon but not a good localizing sign. However we suggest that it may predominantly be a posterior temporal lobe phenomenon given the consistency at which our patient showed activity from that region.

F30

A Case of Stimulus-Aborted Seizures: Rethinking Common Sense

Indranil Sen-Gupta, MD, Vivian Hoang, MD, MBA, James Chen, MD, PhD

We describe a 51 year-old woman whose seizures were consistently aborted by auditory or visual stimuli. The patient was admitted for increasingly frequent episodes of nausea, alterations of consciousness, and variable right leg and hand twitching that were previously well controlled with anti-epileptic medications. Continuous video EEG monitoring demonstrated left temporal slowing, multifocal spikes involving the bilateral temporal and left frontal regions, and multiple poorly localized and poorly lateralized seizures characterized by rhythmic bifrontal and bitemporal slowing with subsequent diffuse spread. Clinically, the seizures manifested primarily as episodes of behavioral arrest that consistently and promptly resolved without a post-ictal period in response to sound or to people entering the patient's room. Admission PET suggested left anteromedial temporal hypometabolism; prior MRIs were unrevealing. The constellation of findings suggested the seizures

***Travel Fellowship Award Recipient:** The Travel Fellowship Award is given to the most outstanding poster submitted by a Fellow who is the First Author.

likely involved the left insular region prior to rapid spread. To our knowledge, this is the first reported case of a seizure type other than absence demonstrating clearly abortive response to sensory stimuli. Moreover, the very act of evaluating the patient during ictal events concomitantly terminated her seizures (with the patient being instantly responsive and normally conversant), suggesting that bedside testing alone may prove misleading for assessing unusual seizures like these.

F31

Characterization of Atypical Benign Partial Epilepsy Suggesting Structural and Genetic Origins

Cyrus Boelman, MD, Hiro Otsubo, MD

Atypical benign partial epilepsy (ABPE) is a rare syndrome defined by the presence of atypical absences, atonic or negative myoclonic seizures with EEG findings of focal discharges and continuous spike-wave discharges in slow-wave sleep (CSWS). We reviewed EEG and clinical profiles of ABPE patients to understand the possible etiologies. All four ABPE cases (ages 7-9 years; 4 males) had negative myoclonus with atonic head and unilateral arm drop, lateralized central focal spike-wave discharges and CSWS starting between 3-4 years of age. The awake backgrounds and sleep features were normal. MRI brain imaging revealed focal increased signal in 3 cases: case1, left amygadala; case 2, bilateral deep white matter and cortex of the left inferior temporal gyrus; case 3, left thalamus & fronto-parieto-occipital areas secondary to .a neonatal stroke. Case 4 had delayed myelination and prominent perisylvian sulci. Case 4 had a chromosomal deletion and systemic dysmorphisms. Cases 1, 3 & 4 were treated with Ethosuximide and responded excellently, including resolution of the CSWS patterns, after unsuccessful trials of other anticonvulsants. Neurodevelopment and epilepsy improvements were despite the presence of known structural brain abnormalities. These cases highlight the potential role of subcortical structures and genetic abnormalities in the development of APBE.

F32

Parry-Romberg: A Rare Neurocutaneous Syndrome

**Brian W Peterson, MD, Edward C Mader, MD, Piotr W. Olejniczak, MD*

Parry-Romberg syndrome, or progressive hemifacial atrophy, is a rare neurocutaneous syndrome characterized by loss of soft tissue on half of the face without muscle weakness. Associated features are variable, but can include ipsilateral brain and limb atrophy, migraine headache, trigeminal neuralgia, ocular abnormalities, and epilepsy1. This syndrome was first described in 1825 and remains poorly understood. Many theories exist about the etiology: they range from sympathetic hyperactivity/hypoactivity, trauma, and focal "en coup de sabre" scleroderma. Serial MRI scans show that over time, there are progressive changes in the ipsilateral cerebral hemisphere2. Our patient was first diagnosed with PRS after a fall with occipital trauma at age five. He subsequently developed a "red line" down the center of his face from his forehead to his chin, which marked the onset of right hemifacial atrophy. At age 23, the patient had his first seizure. EEG showed right frontal semi-regular slowing in the theta and delta frequency bands with superimposed high amplitude sharp waves and sharp-slow wave complexes. These EEG changes correlate with focal cerebral atrophy seen on MRI. Our patient's seizures have become more frequent and disabling, but are significantly improved with a combination of phenytoin, levetiracetam, lacosamide, and diazepam.

F33

Neocortical Ictal High Frequency Oscillations (HFOs) are a Surrogate Marker of Increased Action Potential Firing Rate and Synchrony

**Shennan Aibel Weiss, Garrett Banks, Guy McKhann, III, Robert Goodman, Ronald G. Emerson, MD, Catherine Schevon, MD, Andrew Trevelyan*

Traditionally, the epileptogenic zone is characterized by the earliest and largest amplitude aberrant EEG activity in the Berger bands correlated with a clinical event. However, large EEG signals may arise from either focal discharges or by large synaptic currents that may prevent seizure spread in surrounding territories. It is not a simple matter to distinguish the core active regions from the surrounding territory. To overcome this ambiguity, we sought to identify EEG surrogate markers of increased action potentials in the underlying cortex. We analyzed ictal electrocorticography and microelectrode array recordings from neocortex in four human patients. We demonstrate that ictal high frequency oscillation HFOs detected in layer 4/5 of epileptogenic cortex are correlated with increased action potential firing rate and synchrony. During seizure recruitment action potentials are phase locked to the HFO suggesting that action currents generate these HFOs. In contrast, post-recruitment HFO bursts precede strongly synchronized action potentials. These HFO bursts produce strong signals detectible on the cortical surface by electrocorticography. We conclude that ictal HFOs detected on the cortical surface are indicative of increased neuronal activation in the underlying cortex and can distinguish cortical regions recruited into a seizure from the penumbra.

F34

Effect of Microradiosurgical Transections Upon Kainate Seizure Focus

David Anschel, MD, Alberto Bravin, PhD, Elke Brauer-Krisch, PhD, Geraldine Le Duc, PhD, Pantaleo Romanelli, MD

Multiple subpial transections (MST) sever horizontal intracortical fibers involved in the propagation of seizures, while preserving vertical fibers which are essential for brain function. Less damaging to the brain than a traditional resection, MST still exposes patients to the risks of craniotomy. Microradiosurgery uses synchrotron generated sub-millimetric beams of radiation. Arrays of tightly spaced microbeams produce a lethal effect only to those cells directly in the beam path. Low energy x-rays used for microradiosurgery have

***Travel Fellowship Award Recipient:** The Travel Fellowship Award is given to the most outstanding poster submitted by a Fellow who is the First Author.

a small tissue half-value layer making the technique ideal for treating superficial brain lesions. Video-EEG (VEEG) data was recorded immediately following kainate injected into sensorimotor cortex. Rats then underwent irradiation with an array of parallel microbeams delivered to the seizure focus.Subclinical electrographic ictal events remained frequent up to 10 hours post irradiation. More prolonged seizures exhibited typical rat complex partial seizure semiologies. These results demonstrate the feasibility of recording VEEG before and after precise microbeam irradiation from a synchrotron source; demonstrating a methodology which will be useful for future experiments serving to optimize microbeam doses and configuration in preparation for the treatment of epilepsy in humans. The results show some promise that microradiosurgical MST can limit the spread of seizures without causing clinically evident neurological damage.

F35

The Fallible Phase Reversal

Matthew A. Eccher, MD

Cortical recording of upper limb SSEP is commonly utilized for confirmation of the location of the central sulcus. It may be underappreciated, that this technique is susceptible to error. Case 1: A 21 year old male with medically refractory nocturnal seizures and normal MRI underwent invasive EEG evaluation for epilepsy surgery. Epileptogenic zone included the entire temporal and occipital lobes. This region was hypometabolic on PET; patient had contralateral homonymous hemianopia; a posterior disconnection procedure was therefore elected. Negative phase reversal recorded from implanted EEG electrodes was repeated at the time of definitive epilepsy surgery, with plan to disconnect through the sulcation behind that identified as central sulcus. Postoperative MRI demonstrated that the disconnection had gone through central sulcus, not behind it. Case 2: A 42 year old female with new onset seizures underwent awake craniotomy for resection of a lesion immediately subjacent to central sulcus. SSEP phase reversal appeared anteriorly displaced. Subsequent functional electrical stimulation mapping yielded results more in keeping with expected anatomy. Conclusion: In the setting of physiologic and/or anatomic disturbances, the negative phase reversal of the SSEP can be misleading, and should not be used as the sole means of anatomic localization.

F36

Large Amplitude Evoked Potentials (EPs) in 53 Non-Epileptic Patients

Guillermo Martin-Palomeque, MD, Pilar Pamplona-Valenzuela, MD, Antonio Castro-Ortiz, MD, Miguel Angel Saiz-Sepulveda, MD

Introduction: Large amplitude EPs in non-epileptic patients are unusual and imply central nervous system (CNS) hyperexcitability due to various causes.

Methods: Retrospective chart review including history, physical examination, and imaging and diagnostic studies of non-epileptic patients with large amplitude somatosensory (SSEPs) and visual (VEPs) EPs during the past six years. Large amplitude EPs were defined as follows: VEPs (N75-P100) > 15 uV; and SSEPs (N20-P25) > 9 uV (7uV in patients over 70 years of age). Recording montage for VEPs was Oz-Cz and SSEPs C3'/C4-Fz.

Results: 53 patients (34 females, 19 males; ages 9-90 years) were identified. No CNS pathology was detected in 8 (sensitivity 85%). The remainder of the patients were diagnosed with new CNS disorders. The etiologies included: vascular (37%); myelopathies (13%); demyelinating (11%); space occupying lesions (8.7%); syringomyelia (8.7%); hydrocephalus (6.5%); B-12 deficiency (4.3%); multisystem atrophy (4.3%), Intracraneal Hypertension (4.3%) and; toxins (2.2%).

Conclusion: This study supports the notion that the presence of large amplitude EPs implies CNS hyperexcitability, with a sensitivity of 85% but relatively low specificity for specific etiologies given the wide number of pathologies. Nonetheless, these results confirm the utility of EP studies in patients with suspected CNS pathology.

F37

Pulse-Train Stimulation Enhances SEP Amplitude

David Pinter, CINM, Jon Dizon, CINM, Ronald G. Emerson, MD

High signal-to-noise ratio SEPs, important for effective intraoperative monitoring, can be difficult to obtain in patients with neurological abnormalities and when mechanical factors prevent adequate current from reaching the stimulated nerve. We describe the use of pulse-train stimulation to enhance SEP amplitude for intraoperative monitoring. In 4 patients with normal, readily elicited SEPs, pulse train stimulation (train = four 200-300 usec pulses, interpulse interval 1 msec) produced 1.5 – 2.5 fold augmentation of cortical SEP amplitudes compared to single pulse stimulation at submaximal intensities. At supra-maximal stimulation intensities, augmentation was negligible. Pulse-train stimulation also produced enhancement of subcortical SEPs in 2 of 3 normals. In 3 patients in whom SEPs to standard single pulse stimulation were not monitorable at baseline due to spinal cord compression, lumbar stenosis and leg edema, pulse-train stimulation elicited well-formed, monitorable cortical SEPs. We speculate that augmentation of SEPs by pulse-train stimulation likely results from temporal summation at one or more sites in the large fiber sensory pathways. Pulse-train stimulation appears to be potentially useful for enhancing the amplitude of SEPs that maybe otherwise difficult or impossible to monitor.

F38

Single vs. Train Stimulation for Identification of Malpositioned Pedicle Screws in Scoliosis Surgery

Gema de Blas, MD, PhD, Ignacio Regidor, MD, PhD, Elena Montes, MD, Lidia Cabañes-Martinez, MD, Carlos Barrios, MD, PhD, Jesús Burgos, MD, PhD, Jaime R. López, MD

Introduction: Pedicle screw placement carries a risk of breaching the pedicle and invading the spinal canal. Unfortunately, pedicle screw electrical single stimuli techniques may not identify malpo-

sitioned screws within the spinal canal (SC). Methods: Prospective study of single (SS) versus pulse-train stimuli (PTS) of pedicle screws in detecting screw SC invasion. 244 thoracic pedicle screws in 13 patients (11 females and 2 males; ages 10-26) were studied. Stimulation thresholds for ¡°safe¡± screw placement was >12 mA for SS and >30 mA for PTS. All patients were also monitored with somatosensory and transcranial motor evoked potentials. Final pedicle screw position was established by postoperative computer tomography. Results: Postoperative pedicle screw position was as follows: Intrapedicular-190; cortical breach without SC invasion-25; mild SC invasion-24; and severe SC invasion-5. SS technique detected 4 (13.8%) malpositioned screws intraoperatively but did not identify 25 within the SC. PTS threshold levels ¡Ü 30 mA correlated with SC invasion in 25 screws. The SS sensitivity and specificity was 14% and 99%; and 86% and 78% for the PTS. No patients suffered new neurologic deficits.

Conclusion: Our results indicate that the pulse-train stimulation technique is more accurate in detecting malpositioned pedicle screws when invading the spinal canal.

F39

EEG Asymmetry with Cerebral Perfusion via Innominate Artery

Steven Tobochnik, T. Sloane Guy, MD, MBA, Sheela Pai, MD, Mercedes Jacobson, MD, Camilo A. Gutierrez, MD

Intraoperative electroencephalogram (EEG) monitoring is increasingly used during aortic arch procedures for early detection of acute neurologic dysfunction. In those procedures involving cardiopulmonary bypass, increased neuroprotection may be gained by using hypothermic circulatory arrest and selective cerebral perfusion. Several techniques for cerebral perfusion exist yet no studies have noted distinct EEG patterns associated with different techniques. In this study, we reviewed EEG records of six aortic arch procedures that used cannulation of the innominate artery to provide selective antegrade cerebral perfusion (ACP). In each case, a transient hemispheric asymmetry was noted within 1-2 minutes of the start of head cooling, which consisted of enhanced suppression over the right compared to left hemisphere. The EEG returned to baseline during passive head rewarming in five cases, while a brief left-sided partial seizure occurred during rewarming in one case. These findings suggest that ACP using cannulation of the innominate artery results in enhanced cooling of the right hemisphere as detected by intraoperative EEG monitoring. Since ACP may be associated with risk of embolism, characterization of this finding is necessary to prevent misinterpretation of ischemia by EEG.

F40

Prognostic Value of Intraoperative Monitoring in Peroneal Nerve Surgery

Paul Kwon, MD, Zhengyong Chen, DABNM, Al Llaguno, MD

Introduction: Outcome following peroneal nerve (PN) repair surgery is variable. An intraoperative index for prognosis is desirable.

Objectives: Investigate the correlation of intraoperative nerve action potentials (NAPs) and compound muscle action potentials (CMAPs) with prognosis.

Methods: Twelve patients underwent PN repair surgery. Preoperative ankle dorsiflexion was 0/5 in nine patients and 1/5 in three. Intraoperative NAPs were recorded on distal branches and CMAPs recorded from tibialis anterior following direct electrical stimulation proximal to the lesion. Postoperative follow-up ranged from 22 to 42 months.

Results: Surgical findings included two neuromas, a ganglion cyst, a synovial cyst and the remaining had scar tissue. Surgical repair procedures depended mainly on the results of the NAPs and CMAPs recordings. Both NAPs and CMAPs were present in all with 1/5 dorsiflexion and three additional patients. All but one of these patient achieved satisfactory recovery with 4+/5 dorsiflexion following external neurolysis. Three patients with 0/5 dorsiflexion but present NAPs attained fair recovery, 2-3/5, following neurolysis. The remaining three patients with absent NAPs and CMAPs received sural nerve grafts, among whom only 1 patient attained fair recovery.

Conclusion: Intraoperative NAPs and CMAPs are valuable for prognosis and pivotal in determining surgical management for PN repair surgery.

F41

Intraoperative Neurophysiological Monitoring in Anterior Lumbar Interbody Fusion (ALIF) Surgery

Ilker Yaylali, MD, PhD, Jung Yoo, MD, Alex Ching, MD, Robert Hart, MD

Background: Somatosensory evoked potential (SSEP) and motor evoked potentials (MEP) are frequently used to monitor neurological function during spinal deformity surgery.

Methods: A retrospective review of all patients undergoing ALIF with IONM from November 2008 to July 2010 was performed. Occurrence of post operative neurological deficit were calculated. Factors including gender, operative time, blood loss and number and levels of interbody fusion were analyzed as risk factors for inter-operational alerts.

Results: A total of 80 consecutive patients who underwent ALIF were studied. All 80 patients had SSEP and 45 patients had MEP as part of the intraoperative neuromonitoring. The remaining 35 patients did not have MEP due to neuro muscular blockade requested by the exposure surgeon. No intraoperative changes in MEP were found. Nine(11.2%) patients experienced intraoperative changes in SSEP; none of these patients had new neurological deficits postoperatively. Increased risk of SSEP changes was seen in patients undergoing fusion of both L4/5 and L5/S1 (p=0.024) and longer surgical duration (p=0.036). No correlation was found between age and positive SSEP changes.

Conclusion: The duration of surgery and concurrent fusion of both the L4/5 and L5/S1 levels were significant risk factors for SSEP changes leading to intraoperative alerts.

F42

Dorsal Column Mapping in Intramedullary Spinal Cord Resection

Emily B Kale, Aatif M. Husain, MD

Introduction: Intramedullary spinal cord tumor resection requires a myelotomy placed along the dorsal median sulcus (DMS) to minimize injury to the dorsal columns. Neurophysiologic mapping of the dorsal columns provides more sensitive localization of the DMS than visual identification.

Methods: Stimulation for dorsal column mapping (DCM) is performed by the surgeon with evoked potential recordings from the scalp and peripheral. After exposure, the surgeon stimulates the dorsal column. Lateralized stimulation of the dorsal column evokes a response that is recorded from the ipsilateral peripheral nerve and contralateral scalp. The point at which the responses transition from one side to the other is the location of the DMS. Details of the monitoring methodology will be discussed.

Results: A review of the outcomes of the 11 out of 91 patients undergoing resection of intramedullary spinal cord tumors with DCM, revealed only 1 (9%) with postoperative worsening of neurologic symptoms. Conversely 80 out of 91 patients under went resection with no mapping and 40(50%) of those experienced postoperative worsening of neurologic symptoms.

Conclusions: DCM was successfully minimized postoperative posterior column dysfunction in the majority of patients. Better definition of the DMS may help reduce the morbidity of this procedure.

F43

Intraoperative Monitoring During Removal of Optical Nerve Schwannoma

Daniel San Juan Orta, MD, Manuel E Cortes, MD, Martha Tena Suck, MD, Adolfo Jose Orozco Garduno, MD, Alejandro Jesus Lopez Pizano, MD, Jonathan Villanueva Dominguez, MD, Maricarmen Fernandez GA, MD

Background: Recently have demonstrated the possibility of obtain VEPs directly from stimulation of the optic nerve (ON) during the resection of central skull base tumors to prevent postoperative visual deterioration. However, this type of intraoperative monitoring (IOM) has been rarely used in clinical practice.

Objective: Describe the first case of IOM during removal of the ON schwannoma to preserve the vision.

Methods: We used Nicolet Endeavour CR to performed scalp VEPs (O1-O2, O1-OZ and O2-OZ) stimulating binocularly with flash goggles and direct epidural ON stimulation delivering a rectangular current pulse (intensity 0.2-5.0mA; duration 0.3ms; rate 4.1Hz).

Results: Thirty year-old women with one year of repetitive amaurotic events on the left eye (LE). The pre-operative ophthalmologic examination showed on the LE; visual acuity 20/80 and proptosis. She underwent surgery through left orbito-zygomatic approach. We recorded scalp VEPs only by direct epidural stimulation and during the resection the amplitude decreased 30-40% and returned to the baseline after the neurosurgeon stopped or changed the resection pathway. In the follow-up, the patient didn´t have any visual loss and the tumor was resected (95%).

Conclusion: Direct epidural electrical stimulation of the ON could be help to prevent vision loss during the resection of ON schwannoma.

F44

Direct Cortical Stimulation for Cranial Nerve Motor Evoked Potentials

Daniel Lai, Viet Nguyen, MD, Leslie Lee, MD, Gary Steinberg, Sungho Cho, Jaime R. Lopez, MD

Objectives: Monitoring motor evoked potentials during intracranial surgery can help reduce post-operative morbidity. To increase the definition of area at risk, the authors recorded and characterized compound muscle action potentials (CMAPs) innervated by cranial nerves following direct focal cortical stimulation.

Methods: This is a retrospective review of thirty-one cases of moyamoya revascularization surgeries. During the operation, an electrode stimulation strip was placed on the cortex, and CMAPs were recorded from muscles of the face, tongue, and hand using needle electrodes. CMAPs recorded from bulbar muscles were analyzed for amplitudes and latencies.

Results: Reliable potentials were obtained in eighteen cases. Genioglossus was the most reproducible CMAP and with the shortest latency. A wide range of onset latencies were observed for each muscle CMAP, and longer latencies corresponded with increased stimulation trains and interstimulus intervals (ISI). Each muscle group had some latency variability without an absolute onset. Stimulus intensity does not appear to correlate with latency. There appears to be a general trend of higher CMAP amplitude with increased stimulus intensity.

Conclusions: Stimulation train and ISI are the primary variables which influence CMAP latency. Increased stimulus intensity generally results in larger amplitudes suggesting increased recruitment without clear latency change.

F45

IOM in Cervical Spine Surgeries: Cost-Benefit Analysis

John P. Ney, MD, MPH

Purpose: Construct a probabilistic cost-benefit model for intraoperative neurophysiological monitoring (IOM) in cervical spinal surgeries.

Methods: A decision-model was based on sensitivity, specificity, IOM cost, prevention rate given IOM alert, post-operative spinal cord injury and C5 radiculopathy rates in pooled estimates from published literature with lifetime post-operative costs (health care+lost wages and benefits) compiled using Markov modeling of spinal injury data. Results from Monte Carlo simulation with 10,000

replications were analyzed for cost outcomes and relationship of input variables to outcomes.

Results: IOM saved $9748 (95% CI $5466-$14029) for the reference case 50 year-old patient with spinal cord injury rate of 0.29%, C5 radiculopathy rate of 4.2%,multimodal IOM 2009 Medicare reimbursement rate of $1,535, 52.4% prevention rate given an IOM alert at 94.3% sensitivity and 95.6% specificity. In linear predictions from the simulated data, IOM remained cost-saving at cervical radiculopathy rates as low as 2.4%(p=0.25) and for IOM sensitivity of 76%(p=0.02), specificity of 90%(p=0.001), and IOM cost of up to $9600 per surgery (95% CI $2100-$16600).

Conclusions: Intraoperative monitoring is net cost-saving for cervical spinal surgeries in a theoretical economic model based on the current published literature.

F46

Analysis of Motor Evoked Potentials to Predict Deficits

Jaime R. Lopez, MD, Scheherazade T. Le, MD, Alexander Ekwueme, Leslie Lee, MD, Viet Nguyen, MD, Sungho C. Cho, MD

The aim of this study is to identify neurophysiologic parameters of transcranial motor evoked potentials (TcMEPs) that predict early motor compromise during spinal surgeries. The ultimate goal is to enhance real-time intraoperative neurophysiologic monitoring (IONM) feedback to prevent irreversible postoperative deficits with earlier treatment intervention. Although a 50% amplitude decrease in somatosensory evoked potentials(SSEPs) correlates with potentially reversible spinal cord injury, there are no corresponding standardized warning criteria for TcMEPs; surgeons may be alerted to a significant change only after the TcMEPs are unobtainable. Eleven true-positive cases in 2011-2012 were identified wherein TcMEPs changes occurred and the patient had a new postoperative motor deficit. TcMEP latency, amplitude, duration, turns, phases, area-under-the-curve(AUC) and intraoperative-spinal-cord-index(ISCI) were measured for each muscle group before complete TcMEP loss. Among the 11 cases there were 18 muscle groups monitored: 5 TcMEPs increased in latency, 10 decreased in amplitude, 8 decreased in AUC, and 9 decreased in ISCI. There was a trend towards a smaller and simpler waveform before complete TcMEP loss. TcMEPs should be obtained more frequently during IONM to increase the sensitivity of detecting impending motor compromise. Further research on TcMEP characteristics is warranted to identify early warning neurophysiologic criteria that precede irreversible corticospinal tract injury.

F47

Focal High Frequency Oscillations With Generalized Seizures

Jeffrey R Tenney, MD, PhD, Hisako Fujiwara, EEGT, Douglas F Rose, MD, Nat Hemasilpin, MS

Background: Absence seizures are characterized by briefly impaired consciousness with diffuse 3 Hz spike and wave discharges on EEG. High frequency oscillations (HFOs) are promising biomarkers of the seizure onset zone. This goal of this study was to use MEG to evaluate whether HFOs occur during childhood absence seizures and where the sources localize.

Methods: Children, aged 6 to 12 years old, with newly diagnosed and untreated absence seizures were recruited and MEG recordings were conducted on a 275 channel CTF magnetometer. Time-frequency analysis using short time fast Fourier transform (STFFT) was completed during absence seizures at 1-20Hz, 20-70Hz, 70-150Hz, and 150-300Hz. Source localization was then completed using a sLORETA algorithm for the first generalized spike and slow wave complex.

Results: Twelve children were recruited and forty-four absence seizures occurred during MEG recording. Time-frequency analysis with STFFT showed significant power density in the 1-20Hz, 20-70Hz, and 70-150Hz bandwidths. Source localized preferentially in the parietal region at 1-20Hz and to the lateral inferior frontal region at 20-70Hz and 70-150Hz.

Conclusions: Using MEG, we have been able to detect focal ictal HFOs in children with untreated absence seizures. These areas could be components of the network responsible for generating absence seizures.

F48

Mantle Cell Lymphoma Presenting as Chronic Inflammatory Demyelinating Polyneuropathy (CIDP)

Reuben Mari Valenzuela, MD, Gregory M Blume, MD

Background: Mantle cell Lymphoma is the rarest of the non-hodgkin's lymphoma (NHL) comprising about 6% of NHL cases. There are only about 15,000 cases in the U.S. Patients are typically in their 60s presenting with fever, night sweats and weight loss. Through our extensive literature search, we found no reported case of mantle cell lymphoma presenting as CIDP.

Objective: To report a rare case of mantle cell lymphoma presenting as CIDP.

Design: This is a case report

Results: A seventy year-old right-handed Caucasian male was admitted with a four-week history of ascending symmetric type weakness, tingling in fingers and dysphagia. MRI of the cervical spine showed multiple lymph nodes and subsequent biopsy was consistent with mantel cell lymphoma. Nerve conduction study was consistent with CIDP. Despite aggressive chemotherapy (R-CHOP), patient succumbed a few months later.

Conclusion: Mantle cell Lymphoma is the rarest of the non-hodgkin's lymphoma (NHL) and to our knowledge there has been no reported case of mantle cell lymphoma presenting as CIDP. The cause is unknown and no inherited predisposition has been identified, although over expression of cyclin gene has been reported. Median survival time is about 3-6 years even with extensive chemotherapy.

F49

Median Motor Axon Properties are Altered in Chronic Stroke Survivors

Cliff Klein, PhD, William Z Rymer, MD, PhD, Morris A. Fisher, MD

The study of axon properties in-vivo may provide important insights into motoneuron adaptation following central nervous system lesions. We compared motor axon properties of the paretic and non-paretic limbs in 20 persons (58±8 y) who had unilateral hemiparesis due to chronic stroke (12±7 y). The median nerve was stimulated while monitoring the threshold current required to evoke a 40% compound muscle action potential of the abductor pollicis brevis. The TROND protocol was applied to record stimulus-response, strength duration time constant, threshold electrotonus, current-threshold relation, and the recovery cycle. There was less accommodation to 100-200 ms subthreshold hyperpolarizing currents in the paretic than the nonparetic axons ($P<0.05$), but this difference disappeared during longer lasting (300 ms) currents. This finding may indicate a slower activation of inwardly rectifying current through hyperpolarization-activated cyclic nucleotide-gated (HCN) channels in paretic axons. In the recovery cycle, refractoriness and the relative refractory period were greater, whereas superexcitability and subexcitability were less, in the paretic compared to the nonparetic axons ($P<0.001$). The altered excitability of the paretic axons during the recovery cycle may reflect reduced sodium conductance due to a compensatory down-regulation of sodium channel expression resulting from heightened motoneuron reflex activity following stroke.

F50

Somatosensory and Motor Evoked Potentials to Predict Post-Operative Neurologic Worsening in Patients who Undergo Spinal Cord Tumor (SCT) Resection: A Retrospective Chart Review

Mesha-Gay Melanie Brown, MD, Steven R. Messe, MD, Michael L. McGarvey, MD

Objective: To determine predictors of neurologic worsening after spinal cord tumor (SCT) resection in patients monitored using somatosensory evoked potentials (SEPs) with/without motor evoked potentials (MEPs).

Methods: We reviewed a consecutive series of 134 SCT resections monitored with SEP or SEP/MEP. Our outcome of interest was > 2 point increase in the National Institute of Health Stroke Scale (NIHSS) comparing postoperative status at discharge to preoperative status. Univariate and multivariate analyses identified predictors of worsened neurologic function.

Results: Persistent SEP changes occurred in 9/134 (7%), persistent MEP changes occurred in 6/31 (19%), and 17 patients (13%) had a worsening of > 2 on their NIHSS. Worsened neurologic status was associated with persistent SEP changes (44% vs 10%, p=0.02) but not MEP changes (33% vs 28%, p=1.0). Sensitivity/specificity were 24%/93% for SEP changes, and 22%/82% for MEP changes. In multivariate analysis only younger age (OR=0.94, 95%CI 0.89-0.98, p=0.005) and intramedullary tumor location (OR 5.2, 95%CI 1.6-16.7, p=0.006) were associated with worsened neurologic status.

Conclusions: Younger age and intramedullary tumor location were the strongest predictors of worsened neurologic status post-procedure. SSEP performed better than MEP but both had a high false negative rate, leading to low sensitivities.

F51

Sleep Characteristics in the Neurological Intensive Care Unit

Brandon Foreman, MD, Jan Claassen, MD, PhD, Carl W. Bazil, MD

Background: Sleep is an important yet understudied physiologic parameter in ICUs. Medical/surgical ICU patients have fragmented, non-restorative sleep. Traditional sleep scoring is not feasible in up to 40%. Sleep in the neurological ICU has not been well described.

Methods: Adult neurological ICU patients undergoing continuous electroencephalography were recruited for a randomized, controlled trial of sleep interventions. A sleep montage including electromyography and flow was used to score sleep per American Academy of Sleep Medicine criteria. We used this data to quantify sleep stages and to examine characteristics associated with sleep not meeting criteria for traditional scoring.

Results: 12 patients were enrolled. Mean age was 57.9; 60% were intubated and 60% were stuporous/comatose. Total sleep times averaged 7 hours over a 19 hour period with mean of 85 awakenings. Slow-wave sleep was 4% of total sleep time and rapid-eye movement sleep was <1%. 70% of studies did not meet scoring criteria; this was associated with worse injury severity, worse neurological exam, intubation, and absent posterior dominant rhythm.

Conclusions: Patients in the neurological ICU have fragmented, non-restorative sleep similar to patients in other ICUs. A substantial proportion have sleep that is not scorable by traditional criteria; these patients have worse neurological status.

F52

Propagation of Seizures in a Case of Lesional Mid-Cingulate Gyrus Epilepsy Studied by Stereo-EEG

Rafeed Alkawadri, Andreas V Alexopoulos

Literature on the propagation of seizures arising from the cingulate gyrus is limited, as cingulate coverage with interhemispheric subdural electrodes is usually challenging and incomplete due to inherent anatomical and vascular limitations. The bulk of the available literature on the connectivity of the cingulate gyrus is based on studies done on rhesus monkeys. Little is available on the connectivity of the cingulate gyrus in humans. We present a case of lesional mid-cingulate gyrus epilepsy confirmed by stereotactically placed intracranial depth electrodes. Hypermotor symptomatology was seen during the first 7 seconds of seizure onset while the seizure was still confined to the mid-cingulate gyrus contacts. Patient had brief contralateral clonic movements as seizure propagated to the primary motor cortex. There was high concordance between the primary propagation contacts as delineated by intracranial EEG

and the contacts with higher coherence values in the connectivity matrix. Interestingly, cingulate-extra-cingulate connectivity and spread to the primary motor, premotor and prefrontal cortex was seen prior to the spread to other cingulate contacts, of which one was less than 18 mm away from the onset contact. This report is one of few in the published literature documenting propagation of seizures arising from the mid-cingulate cortex in humans. As illustrated by this data hypermotor semiology results from direct activation of cingulate cortex. Subsequent seizure propagation in this patient activated an extensive extra-cingulate rather than an intra-cingulate epileptogenic network. Further studies exploring functional, electrophysiological and anatomical connectivity of the cingulate cortex in humans are needed.

F53

Video-EEG of Extra-temporal Lobe Epilepsy in Older Adults

Adriana S. Tanner, MD, Kathryn McDonald, RN, Yvan Tran, MD, Kristina Karanec, DO

Rationale: There is a paucity of clinical and EEG information about extra-temporal lobe epilepsy in elderly patients.

Methods: Retrospective review of adult patients admitted to our Epilepsy Monitoring Unit (EMU) between 2006 and 2011.

Results: We identified six patients with extra-temporal lobe epilepsy. Their mean age was 63.5± 6.8 years. 83% of the patients were women. These patients accounted for 5.7% of elderly patients admitted (105 patients), but only 0.5% of all admissions to the EMU during the study period. Altogether, 33 seizures were recorded in this group: the seizure semiology included hypermotor seizures (69%), Complex Partial seizures (21%), Right face tonic followed by right face clonic seizures (3%), left versive seizures (3%) and complex motor seizures (3%). Auras were rare, and only one unclassified aura was recorded. The location of the interictal epileptiform discharges was: right frontal (16%), right fronto-central (16%), right parieto-occipital (16%), left frontal (16%) and left temporo-parietal (33%).

Conclusions: Elderly patients with extra-temporal lobe epilepsy accounted for a small percentage of patients admitted to our EMU. This may reflect the fact that older adults continue to represent a minority of all admissions to EMUs and that there is a lower frequency of extra-temporal lobe epilepsies

---

F54

Electroencephalographic Features of Dravet Syndrome

Se Hee Kim, MD, Linda Laux, MD, Sookyong Koh, MD, PhD, Anne T Berg, PhD, Douglas R. Nordli Jr., MD

Objective: Patients with Dravet syndrome (DS) have pleomorphic seizure types including both generalized and focal seizures. Methods: Sixty-nine overnight video-EEG in 52 children and adolescents with DS (24 male, 38 female) were reviewed. Background: Interictal abnormalities and clinical electrographic seizures were analyzed.

Results: The median age at the time of the study was 2.9 years (0.8 – 19.4 years). Background activity was normal in 13 (25%) patients while 36 (69.2%) had diffuse slowing. Interictal epileptiform patterns: (1) generalized (33/52, 63.5%); (2) multifocal (25/52, 69.4%); (3) focal (22/52, 42.3%,). Ictal patterns: (1) myoclonic (24/52, 46.2%); absence (10/52, 19.2%); eyelid flutter (6/52, 11.5%); convulsive (generalized, focal) (5/52, 9.6%); partial seizure with intermixed myoclonic jerks (2/52, 3.8%). Thirteen convulsive or partial seizures were captured in 6 patients. Purely focal or generalized convulsive seizures with corresponding focal or generalized ictal pattern were uncommon (3/13, 23.1%). Seizure semiology was more typically complex with both generalized and focal components which could correspond to complex ictal patterns of both generalized and focal features (10/13, 76.9%).

Conclusion: Seizures in DS can be complex with both focal and generalized clinical features and ictal EEG patterns. Those seizures cannot be classified in the current ILAE seizure classification system.

Poster Abstracts—Saturday, February 9

Posters will be on display from 6:45 AM - 1:30 PM and authors will be present between 7:00 AM - 8:00 AM for discussion.

The Poster Hall is located in the Fisher Island, Lummus Island, Hibiscus Island and Bal Harbour Island rooms on Level 3 of the Miami Marriott Biscayne Bay.

Poster numbers indicate the day of presentation (F=Friday, S=Saturday) and the board number where the poster will be displayed.

S1

Autonomic Dysfunction in Adult Onset Alexander Disease: A Case Report and Review of the LIterature

Scott D. Spritzer, DO, Brent P. Goodman, MD

Background: Alexander disease (AxD) is an astrogliopathy, resulting from a mutation in the glial fibrillary astrocytic protein (GFAP) gene. Different clinical subtypes have been described based upon the age at which symptoms begin. Patients with the adult onset form, can develop a, spastic paraparesis, palatal myoclonus, ataxia, and bulbar weakness. Autonomic nervous system (ANS) dysfunction has also been reported as a potential manifestation of adult onset AxD.

Objective: We report a case of adult onset AxD with symptoms of autonomic impairment that underwent formal autonomic testing. Additionally, a literature search was conducted to review the frequency and pattern of autonomic dysfunction in this patient population.

Results: A 51 year-old patient was diagnosed with AxD at the age of 47, following an 8 year history of vertigo, diplopia, and sleep disturbance. The patient developed several autonomic symptoms over his clinical course. Autonomic testing demonstrated OH on tilt-table testing with absent late phase II and IV responses during the Valsalva maneuver, severe cardiovagal impairment, and preserved postganglionic sympathetic sudomotor function. These findings were consistent with central autonomic failure. The most common autonomic symptoms reported in other AxD cases include constipation, urinary incontinence, and sphincter dysfunction. To our knowledge, this is the first report of formal autonomic testing in AxD. Conclusion: Symptoms of ANS impairment can occur in patients with AxD, and can include orthostatic hypotension and bowel/bladder dysfunction. Autonomic testing in our patient suggests impairment in central autonomic pathways.

S2

Safety of Electrical Cardioversion Using Continuous EEG with Uunderlying Cerebral Edema

Tariq Janjua, MD, Eric Nussbaum, MD, Jodie Lowary, CNP

Introduction: During electrical cardioversion procedures it is not clear if there is a risk of seizure in patients with cerebral edema with focal mass effect. The use of continuous EEG can help to monitor for signs of electric changes in real time, allowing for the procedure to be done safely.

Methods: A cardioversion procedure was done on a 76 year old woman with new atrial fibrillation and pervious insertion of a demand pacemaker for sick sinus syndrome. Due to rapid heart rate, cardioversion was selected. She had a left dominant side partial meningioma with associated cerebral edema. A continuous EEG was done during the procedure to observe real time EEG for any risk of seizures. Pacemaker was disabled with a magnet. EEG recordings, radiological images and pacemaker data are presented.

Results: The EEG was read at all times to watch for any signs of electric seizures. Peri-shock EEG showed no ictal changes. Patient converted to sinus rhythm followed by a successful surgical procedure for meningioma.

Conclusion: The ability to watch for seizures with continuous EEG during a cardioversion procedure in patients with a low seizure threshold adds to the safety and security of both the patient and health care providers.

S3

From BICS to BISE

Asma Zakaria, MD, Nishi Rampal, MD

A 54 year old woman with primary CNS lymphoma and recent whole brain radiation therapy presented to the hospital with altered mental status and a witnessed generalized seizure. MRI brain did not show acute pathology or recurrence and cEEG was initiated. Two independent epileptogenic foci evolved over 3 days from rhythmic delta activity to cyclic seizures and then status epilepticus on the left and periodic discharges to cyclic seizures and status epilepticus on the right. This progression of epilepsy evolved into bilateral independent and simultaneous cyclic seizures, culminating in bilateral independent and overlapping status epilepticus. This case is different from the previously described ping pong seizures in that the foci are not time locked to each other. Each focus has its own automaticity and refractory period with seizures occurring independent of any contribution from the contralateral side. Both hemispheres seize simultaneously but independently and eventually progress to status epilepticus which burns out into bilateral independent periodic discharges. To our knowledge this is the first reported case of bilateral independent cyclic seizures (BICS) with progression to bilateral independent status epilepticus (BISE).

S4

Dense Array EEG in Intensive Care Patients

Elaine T Kiriakopoulos, MD, Donald Tucker, PhD, Marie Terrill, PhD, Donald Schomer, MD, Elizabeth Bachman, MPH, Susan T Herman, MD

Background: Critically ill patients are at risk for primary and secondary brain injuries (e.g. seizures and ischemia) causing permanent neurologic disability. Continuous EEG monitoring can detect injuries at a potentially reversible stage. Dense array EEG (dEEG) with electrical source imaging allows for more precise localization of abnormal cortical regions, but has not been used previously in

the ICU. We aimed to demonstrate feasibility of dEEG in ICU patients with acute cerebral injury.

Methods: We recorded dEEG using a 256-electrode HydroCel Geodesic Sensor Net (HCGSN, Electrical Geodesics, Inc., Eugene, OR).

Results: Patients (n=16) recorded with dEEG in the ICU setting included cerebral ischemia (5), closed head injury (1), status epilepticus (1), post cardiac arrest (1), intracerebral hemorrhage (1), encephalitis (5), and metabolic encephalopathy (2). Sample recordings and source localization results of triphasic waves and periodic lateralized epileptiform discharges will be presented. dEEG recordings were technically adequate despite the electrically hostile ICU environment. Sensor net placement using saline as electrolyte was rapid, generally requiring less than 5 minutes.

Conclusion: In this pilot study, we have demonstrated feasibility of dEEG recording and source localization in the ICU. Future work will extend recording times to 24 hours and correlate EEG findings with MRI.

S5

An Abbreviated EEG Montage for Rapid Assessment of Electrographic Cerebral Activity In Acutely Hospitalized Adult Patients

Keith Dombrowski, MD, Brian Mace, BS, Saurabh R Sinha, MD, PhD, William Gallentine, MD, Christopher Skidmore, MD, Karl Sanzenbacher, MD, Brad Kolls, MD, PhD

Introduction: Increased continuous EEG utilization has generated an interest in faster acquisition and interpretation of EEG data using limited electrode arrays (LEA) coupled with quantitative algorithms. The aim of the current project was to test a novel LEA, quantify any error rate imparted by the reduction in electrodes, and determine if multiple montages could correct this error.

Methods: Four experienced neurophysiologists reviewed 250 de-identified EEG segments that were reformatted into an 8 electrode array containing a lateral chain and central electrode bilaterally. In phase 1, segments were interpreted in a single AP bipolar montage. In phase 2, fifty frequently misread segments were reinterpreted using five additional montages.

Results: In phase 1, 1000 EEG interpretations were reviewed yielding an approximate sensitivity of 70-75% for seizure, PEDs, and normal with specificities greater than 90%. In phase 2, 150 EEG interpretations were collected with no significant improvement noted in the detection of any EEG finding.

Conclusions: This trial suggests that LEAs contain a base error rate that cannot be corrected with additional montages. The implication of these results suggests that if LEA's are to be leveraged for rapid EEG acquisition and algorithm development, the LEA-specific error rate needs to be established first.

S6

Role of Neuromonitoring in the Acute Phase After External Carotid to Internal Carotid Arterial (ECIC) Bypass Surgery

Tariq Janjua, MD

Introduction: Along with clinical examination and radiological studies, the care of post ECIC bypass patients usually involves blood pressure control, fluids, electrolytes and good medical preventive care in the neuroICU. We describe the role of neurophysiogical monitoring for bizarre behavior in a patient after a 2nd bypass procedure.

Methods: Continuous non-video EEG monitoring was used after an elective left side ECIC bypass procedure in a patient with mild aphasia and bizarre behavior.

Result: A 64 year old patient with a diagnosis of Moyamoya was admitted for an elective left ECIC bypass procedure. A year ago she underwent right side bypass. Folowing the first procedure she developed confusion which resolved with antiepileptic medications. Within 24 hours after the 2nd procedure, she developed confusion and word finding difficulty. cEEG showed diffusely slow background activity associated with left hemispheric sharp activity. She was given levetiracetam 250 mg every morning and 500 mg every evening, oxcarbazepine 600 mg twice daily and phenytoin 250 mg every night with resolution of her partial aphasia and confusion. EEG findings and radiological studies are presented.

Conclusion: Neurophysiological monitoring should be considered in patients after neurovascular procedures like ECIC bypass if there is any change in neurological presentation.

S7

DETECTing Seizures Among Comatose Children: Interim Results

Eric Payne, MD, Kristin McBain, MSc, James S Hutchison, MD, Chris Parshuram, MD, Carter Snead, MD, Rohit Sharma, RET, Xiu Yan Zhao, MSc, Helena Frndova, MSc, Margaret Wilkinson, MSc, Maureen Dennis, PhD, Nicholas S. Abend, MD, William Gallentine, DO, Kendall Nash, MD, Cecil D Hahn, MD,MPH

The prospective multicenter DETECT study aims to characterize the prevalence of and risk factors for electrographic seizures (ES) among comatose children in the ICU and determine the impact of ES on outcome. Critically ill children with coma (GCS <=8) of any etiology receive 48 hours of EEG monitoring on a research and/or clinical basis. Here we report interim results from the first 120 subjects enrolled at The Hospital for Sick Children, Toronto. The median duration of monitoring was 48h (10h-1441h), mean age was 6.3y (1d-17.6y) and 53 subjects were male. ES occurred in 30 subjects (25%), including 24 (20%) who experienced some nonconvulsive seizures (NCS) and 9 (7.5%) who experienced entirely NCS. Electrographic status epilepticus occurred in 9 subjects (7.5%), all of whom experienced mainly NCS. The mean interval between cEEG onset and first ES was 6.3h (5s-32h). Univariate analyses comparing children who did/did not experience ES identified several predictors of ES: younger age (mean 3.5y vs. 7.3y; p=0.0003), re-

cent clinical seizures (p=0.0011), interictal epileptiform discharges (p<0.001), and periodic discharges (p=0.0014). With the addition of 3 U.S. children's hospitals, we anticipate doubling the sample to 240 subjects. Functional and neuropsychological outcomes will be assessed at 6 months post discharge.

S8

SIRPIDs: Prevalence and Outcomes in Critically-Ill Patients

Amanda F Van Straten, MD, Ryan Hakimi, DO, Andrea S Hakimi, DO

Objectives: To determine the prevalence, associated factors, and outcomes as defined by discharge disposition of stimulus-induced rhythmic, periodic, or ictal discharges (SIRPIDs) on long-term video-EEG (VEEG) in critically-ill patients.

Methods: Following IRB approval, we retrospectively reviewed patient characteristics and VEEG findings of all consecutive critical care unit patients that underwent VEEG monitoring between January and September 2012.

Results: The prevalence of SIRPIDs was 10.5% (4 of 38 patients). Factors associated with SIRPIDs versus non-SIRPIDs patients included subclinical status epilepticus occurring at any time during the patients intensive care unit stay (100% vs 17.7%, p=0.003), longer total VEEG recording time (261 vs 51.7 hours, p=0.010), and acute traumatic brain injury (75.0% vs 20.6%, p=0.035). Sex, age, a history of epilepsy, and background rhythm reactivity on VEEG did not correlate with the presence of SIRPIDs. In addition, the presence or absence of SIRPIDs had no bearing on discharge disposition.

Conclusion: This small series suggests that the presence of subclinical status epilepticus, lengthier VEEG recording times, and acute traumatic brain injury correlated with the presence of SIRPIDs. The presence of SIRPIDs did not correlate with outcome.

S9

Early Recognition of Medical Complications in RSE Can Save Lives

Emitseilu Kevin Iluonakhamhe, MD, Asma Zakaria, MD

Three patients with no prior history of seizures who were admitted to our neuro-critical care unit between March 2010 and January 2012 for cryptogenic refractory status epilepticus lasting between 20-81 days. A broad range of anti-epileptic agents such as phenytoin, levetiracetam, valproic acid, topiramate, felbamate, phenobarbital, lacosamide as well as infusions of propofol, ketamine, midazolam, lorazepam and pentobarbital were utilized. High dose steroids, plasma exchange, epilepsy surgery and inhaled anesthetics were employed in some patients. Complications observed included drug induced paralytic ileus, small bowel ischemia, severe metabolic acidosis due to propylene glycol toxicity, lactic acidosis, acute renal failure requiring continuous veno-venous hemodialysis (CVVHD), septic shock, drug induced pancreatitis, cerebral ischemia, ventilator associated pneumonia and critical illness myopathy and neuropathy. These obstacles in the management of status epilepticus are often iatrogenic and contribute to overall morbidity and mortality. Furthermore, some of these conditions and treatments interfere with drug absorption, metabolism and binding, making the management of status epilepticus all the more challenging. Two of the three patients in our cases series survived and eventually returned home. Patients with status epilepticus face a myriad of challenges but foreseeing these impediments and their early aggressive management can improve outcomes tremendously.

S10

cEEG monitoring: A Survey of Neurophysiologists & Neurointensivists

Jay Gavvala, MD, Nicholas S. Abend, MD, Suzette M. LaRoche, MD, Irena Garic, RN, MPH, Susan T. Herman, MD, Jan Claassen, MD, PhD, Cecil D Hahn, MD, MPH, Michael Macken, MD, Stephan Schuele, MD, MPh., Elizabeth Gerard, MD, Critical Care EEG Monitoring Research Consortium

Increasing data is available regarding the utility of continuous EEG monitoring (cEEG) in critically ill adults, yet it remains unclear how this data has translated to clinical practice. We aimed to describe current practice among neurophysiologists and neurointensivists. Ninety-five physicians completed the ongoing online survey. Ninety-eight percent identify their institution as a tertiary care center. Half of the respondents provide EEG interpretation, 36% provide care in the ICU and 14% are involved in both. EEG technologists are available 24/7 at 84.7% of institutions (18.6% in-house, 66.1% on-call). cEEG is commonly utilized to detect non-convulsive seizures in encephalopathic patients with clinical seizures (95%), involuntary movements (88%), cerebral hemorrhage (80%), TBI (78%) and cardiac arrest (76%). Practice was more variable for patients with encephalopathy and tumors, stroke, and metabolic encephalopathy (53-67%). cEEG is used by 63% to monitor burst-suppression and 19% to monitor for vasospasm. Forty-eight percent of physicians monitor comatose patients for 24 hours and 34% monitor for 48 hours; however, 26% would increase the length of monitoring given unlimited resources. Practices are similar among initial respondents from tertiary care centers regarding primary indications for cEEG. However, there is wide variability regarding recommended duration of monitoring and secondary indications for cEEG.

S11

Lateralized Infraslow Oscillations on Scalp EEG in Acute Brain Injury

Nicolas Gaspard, MD, PhD, Nishi Rampal, MD, Ognen AC Petroff, Lawrence J. Hirsch

Background: Infraslow oscillations (<0.5Hz; ISO) are involved in physiological processes and modulate normal cortical activity. With the exception of ISO associated with cortical spreading depressions, very little is known of potential pathological ISO.

Methods: We reviewed 30 consecutive continuous EEG recordings in patients with acute brain injury acquired with DC-coupled amplifiers and Ag/AgCl electrodes. ISO were defined as reproduc-

ible waveforms with a period > 2 second (frequency < 0.5Hz) and a physiologic field.

Results: We identified ISO lateralizing to the side of the injury in 2 frequency bands: 0.2 to 0.5Hz (26 patients) and 0.02 to 0.1Hz (17 patients). Both types of activity exhibited broad fields that usually comprised the field of conventional EEG abnormalities. Periodic discharges and seizures tended to be more frequent in patients with 0.02 to 0.1Hz oscillations (RR 4.6, p-value 0.1 and RR 3.8, p-value 0.18, respectively; Fisher exact test). The amplitude and frequency of periodic discharges were modulated by these activities.

Conclusions: We report a high incidence of lateralized ISO on scalp EEG after acute brain injury. Our data also suggest that they might modulate cortical excitability and indicate an increased risk for seizures. Further work is needed to unravel their pathophysiology and clinical relevance.

S12

Quantitative EEG Measures of Laterality Show High Test-Retest Reliability

Joshua Ewen, MD, Balaji Lakshmanan, MS, Cathy Bachur, BA, Kira Lanier, BA, Brian Caffo, PhD, Eric H.W. Kossoff, MD, Nathan E. Crone, MD, Anne M Comi, MD

Work from our laboratory has demonstrated the utility of quantitative analysis of the EEG signal in determining which infants with port-wine birthmark (PWB) are at the highest risk of developing the intracranial involvement consistent with Sturge-Weber syndrome (SWS). We are further interested in extending these techniques to determine which individuals with known SWS are at high risk for neurological deterioration. As a preliminary step, we sought to assess the test-retest reliability of our quantitative metrics. Nine subjects (ages 5.2-91.5 years; mean=8 years) underwent two EEGs within the same day. Fast-fourier transform assessed amplitude of the signal in each of the four classical frequency bands. A Laterality Score ([L-R]/[L+R]) quantified degree of asymmetry in each of the four frequency bands plus a total of all bands, per the technique in Ewen et al, 2009. Intra-Class Correlation (ICC) was used to evaluate test-retest reliability. The laterality score of the frequency bands (including Total) ranged from 0.94 to 0.97, suggesting high test-retest reliability of these signal analysis metrics.

S13

EEG Source Imaging of Interictal Spikes in a Patient with Focal Cortical Dysplasia

Octavian V. Lie, MD, PhD, Jose E. Cavazos, MD, PhD

Rationale: Postoperative seizures occur in approximately 30-60% of epilepsy surgery patients. EEG source imaging (ESI) of scalp interictal spikes may improve epileptogenic zone localization and postoperative seizure outcome. This study is the first step of a detailed study assessing the accuracy of various ESI methods, an area of limited exploration thus far.

Methods: A patient with pharmacoresistant epilepsy underwent a right fronto-parieto-temporal resection disclosing changes of focal cortical dysplasia ILAE type 2b. Preoperatively, clinical, neuroimaging (structural brain MRI and MEG), and neurophysiological (scalp EEG, extraoperative eletrocorticography, motor and sensory electrical stimulation mapping) information was used to define the resection location and extent. The patient was rendered free of disabling seizures at one year postoperatively (ILAE outcome class 2). We retrospectively averaged 105 interictal spikes with similar scalp topography recorded with conventional 10-20 system electrodes. For ESI, we computed two individual forward models (SMAC and BEM) combined with two inverse methods (LAURA and sLORETA), respectively, applied at the mid-upswing time frame of the averaged spike.

Results: SMAC-LAURA and BEM-sLORETA produced maximal source solutions within the resection volume, and less than 2.5 cm from the resection centroid.

Conclusions: Comparing ESI methodology may lead to an improved noninvasive definition of the epileptogenic zone.

S14

In-Hospital Encephalopathy: An EEG Microstate Analysis

Rani Sarkis, Jong Woo Lee, MD, PhD

Introduction: Acute alteration in mental status (AMS) is present in up to 30% in medical units and is associated with increased health care costs, and increased mortality. EEG microstate analysis examines the brain's temporal evolution of topographic changes, and has been utilized to assess spontaneous conscious cognitive activity. We examine changes in EEG microstate analysis in patients with AMS in the hospital.

Methods: Encephalopathic hospitalized patients age<60 with no known cerebral lesions, a nonfocal EEG, and AMS were compared to a control group with a normal EEG and no AMS. Six second artifact free EEG samples following an eyeblink were analyzed.

Results: A total of 20 control subjects and 16 encephalopathic subjects were identified. Four stable microstates consistent with published literature were observed in controls. These microstates were observed in only 38% of patients. Using a limit of 4 microstates, the average variance explained by the 4 microstates was 73.5±6.5% in controls and 65.3±7.0% in encephalopathic patients ($p<0.001$).

Conclusion: EEG analysis of microstates in inpatients with AMS reveals a decrease in microstate stability, indicating a breakdown of underlying electrophysiological processes. These preliminary findings may provide objective measures to assess hospital encephalopathy, and may be used in future prognostic models.

S15

EEG Abnormalities in Psychopath and Non-Psychopath Violent Offenders

Ana A. Calzada-Reyes, MD, Alfredo Alvarez, PhD, Mitchell Valdes-Sosa, PhD, Lidice Galin-Garcia, PhD

To find electrophysiological differences specifically related to the psychopathy construct and independent of the violent behavior. The current investigation compares the QEEG and the current source density measures of violent psychopath offenders to a non-psychopath violent group. The resting EEG activity and LORETA for the EEG spectral fast bands were evaluated in 58 violent offenders, 31 with and 27 without psychopathy according to the Hare Psychopathy Checklist e Revised. All subjects were assessed using the DSM IV-R criteria. The EEG visual inspection characteristics and the use of frequency domain quantitative analysis techniques are described. QEEG analysis showed a pattern of excess of beta activity on the left parieto-temporal regions and bilateral occipital areas and decrease of alpha band on the left centro-temporal and parieto-central derivations in the psychopath group. LORETA signified an increase of beta activity (17.18 Hz) in psychopath group relative to a non-psychopath group within fronto-temporo-limbic regions. These findings indicate that QEEG analysis and techniques of source localization may reveal differences in brain electrical activity among offenders with psychopathy, which was not obvious to visual inspection. Taken together, these results suggest that abnormalities in a fronto-temporo-limbic network play a relevant role in the neurobiological basis of psychopathy.

S16

Significance of Rapid Bilateral Eye Blinking During Partial Seizures

Prasuna Latha Velur, MD, Giridhar Kalamangalam, MD, MBBS, Dphi

Rationale: Rapid bilateral eye blinking (RBEB) is commonly seen during generalized absence seizures, and is the defining character of seizures in the Jeavons syndrome. Complex partial seizures (CPSs) in focal epilepsy are usually associated with the opposite semiology – fixed staring – though we have also observed RBEB in CPSs. In this pilot study we surveyed the electroclinical associations of RBEB in a cohort of patients with proven focal epilepsy exhibiting CPSs.

Methods: We retrospectively reviewed scalp video-EEG monitoring seizure data on all patients with proven partial epilepsy and CPSs encountered over a 12-month period (year 2009). Seizures with RBEB were identified by obvious excessive eyeblink artifact on the EEG that lasted ≥ 10 seconds at any time during the seizure. The total number of eye blinks for the duration of the seizure, as well as the 10-second epoch with the highest number of eye blinks (peak blink rate) was noted.The ictal EEG of each seizure (whether with RBEB or not) was classified as having a lateralized or nonlateralized onset pattern.

Results: 156 partial seizures (n=156) were recorded in 41 patients (N=41) that fit the above criteria. Ninety-one seizures (n1=91) had clearly lateralized ictal EEG onsets; the remainder (n2 = 65) had nonlateralized onsets. Four patients had 8 seizures (m=8) that showed RBEB; in each such seizure, RBEB was confirmed by peak eye blink rates during the seizure significantly higher than at baseline ($p < 0.01$). Assuming N and n as population figures, the probabilities of a random seizure having lateralized versus nonlateralized onset were n1/N ≈ 0.58 versus n2/N ≈ 0.42. Thus, the probability of m seizures with RBEB all having nonlateralized onset was (0.42)8 ≈ 0.001.

Conclusions: (i) RBEB, a semiological feature often seen in seizures arising in generalized epilepsy syndromes, may occur rarely in seizures in focal epilepsy (4/41 ≈ 10% of patients and 8/156 ≈ 5% of seizures, in this series). (ii) There is a statistically significant association between seizures with RBEB and a diffuse (nonlateralized) ictal EEG pattern at onset. This observation argues for direct bihemispheric propagation of seizures with RBEB, or alternatively, preferential spread of such seizures to regions (e.g. the orbital and medial frontal cortices) that secondarily project to widespread brain areas. Functional imaging studies demonstrate medial and orbital frontal activation with spontaneous eye blinking (Tsubota et al, Exp Eye Res 69(1):1-7, 1999; Yoon et al, Neurosc Lett 381(1-2):26-30, 2005). These brain areas are also among those that display an early increase in the BOLD signal during generalized spike-wave paroxysms in simultaneous EEG-fMRI studies (Benuzzi et al, Epilepsia 53(4):622-30, 2012). (iii) It is plausible that in CPSs with RBEB, seizure spread occurs early to 'eye-blink eloquent' brain areas that results in both rapid eye blinking as well as a diffuse ictal EEG. The further associations of RBEB with epilepsy syndrome and treatment responsiveness will be investigated in a larger study that confirms these preliminary findings.

S17

EEG as a Biomarker: Pre-Hypsarhythmia Predicts West Syndrome

John J. Millichap, MD, Sookyong Koh, MD, PhD, Douglas R. Nordli Jr., MD

Objective: Assess serial EEG as a biomarker to predict West syndrome (WS).

Background: Retrospective studies describe a specific EEG background, pre-hypsarhythmia (focal or multifocal epileptiform discharges) that appears 3-6 weeks prior to hypsarhythmia.

Design/Methods: A longitudinal prospective cohort study. Subjects with neonatal hypoxic ischemic encephalopathy (HIE) were followed prospectively with serial monthly EEG from ages 3- 7 months. EEGs analyzed and assigned a type: Type 0: Normal for age. Type 1: background normal or mildly abnormal; focal or multifocal epileptiform discharges < 50% of non-REM sleep record; Type 2 (prehypsarhythmia): background abnormal; focal or multifocal epileptiform discharges > 50% of non-REM sleep record; Type 3 (hypsarhythmia): chaotic, high-voltage (>200 uV) epileptiform discharges.

Results: Twelve subjects have been enrolled thus far and followed until 9 months old. Six subjects (50%) had abnormal EEGs at 3 months old. Of those 6, two subjects (33%) had pre-hypsarhythmia. Of those 2 with pre-hypsarhythmia, 2 (100%) developed infantile spasms. The remaining subjects are seizure-free with Type 0-1 EEG.

Conclusions: These preliminary results suggest that a specific EEG pattern, prehypsarhythmia, appears prior to the development of hypsarhythmia. Identification of an EEG biomarker to predict WS may allow for pre-emptive treatment and prevention of epilepsy.

S18

DIAGNOSTIC OUTCOME OF EXPLORATORY INTRACRANIAL EEG RECORDING

Ricky W. Lee, MD, Greg A. Worrell, MD, PhD, Elaine C. Wirrell, MD, Gregory Cascino, MD, W. Richard Marsh, MD, Nicholas M Wetjen, MD, Elson L So, MD

Noninvasive investigations of extratemporal lobe epilepsy sometimes result in indeterminate seizure lateralization. Therefore, bilateral exploratory intracranial electrode implantation (EXPIEEG) is sometimes performed, before more electrodes are implanted for precise seizure localization. Between 1997 and 2010, ten patients underwent EXPIEEG at our institution. Four (40%) were found to have lateralized seizure onset. Compared with non-lateralizing EXPIEEG, there was a trend for lateralizing EXPIEEG to have less implanted electrode strips (5.3 strips vs. 9.5 strips; p=0.05), but not significantly less electrode contacts (38 electrodes vs. 72 electrodes; p=0.092). 75% of patients with regional but not lateralized scalp seizure onset (e.g. bifrontal, or midline frontocentral) had lateralizing EXPIEEG, vs. only 16.7% of the patients with generalized or indeterminate scalp seizure onset, but the difference did not reach significance (p=0.19). Furthermore, focal seizure semiology, symmetry of implantation or positive SPECT finding had no association with the yield of EXPIEEG. Due to the small sample size in this study, brain MRI and interictal scalp EEG findings did not yield meaningful data. In conclusion, this study showed that the yield of EXPIEEG is low (<50%). The small number of patients in our study limits our ability to determine factors that contribute to the yield.

S19

Triphasic waves: EEG, clinical and imaging characteristics

Peter W. Kaplan, MB, FRCP, Raoul Sutter, MD

Objectives: To characterize triphasic waves (TWs) and clinical/neuroradiological correlates in encephalopathic patients.

Methods: 9-year cohort study of consecutive encephalopathic patients with semiquantitative assessment of electrographic TWs characteristics (frequency, amplitudes, location, direction of time lag), background activity and reactivity to stimulation/arousal. Clinical conditions, neuroimaging, and outcome (Glasgow Outcome Score (GOS) and death) were assessed.

Results: 105 adult patients with TWs were identified. EEGs were performed because of mental status change (100%), emergence of delirium (16%) and suspected seizures (13%). 59% of patients had infections, 50% renal insufficiency, 25% dementia, and 18% respiratory failure. Intensive care was required in 81%. Neuroradiological studies revealed white matter lesions in 60%, cerebral atrophy in 55%, ischemic stroke and intracerebral hemorrhage in 14% each. 84% had ≥2 of the major clinical and/or neuroradiological abnormalities. Outcome was unfavorable (GOS1-3) in 68% and mortality 20%. Background activity and TWs characteristics were not associated with outcome. Absence of background reactivity was associated with death (OR4.3, 95%CI 1.5-12.5, p=0.007).

Conclusions: In contrast to earlier studies with a high mortality, death occurred in our cohort in 20%. The only EEG feature associated with death was the absence of background reactivity;increased frequency, lag direction and TW amplitudes were not.

S20

Triphasic Waves: EEG, Clinical and Imaging Characteristics

Raoul Sutter, MD, Peter W. Kaplan, MB, FRCP

Objectives: To characterize triphasic waves (TWs) and clinical/neuroradiological correlates in encephalopathic patients.

Methods: 9-year cohort study of consecutive encephalopathic patients with semiquantitative assessment of electrographic TWs characteristics (frequency, amplitudes, location, direction of time lag), background activity and reactivity to stimulation/arousal. Clinical conditions, neuroimaging, and outcome (Glasgow Outcome Score (GOS) and death) were assessed.

Results: 105 adult patients with TWs were identified. EEGs were performed because of mental status change (100%), emergence of delirium (16%) and suspected seizures (13%). 59% of patients had infections, 50% renal insufficiency, 25% dementia, and 18% respiratory failure. Intensive care was required in 81%. Neuroradiological studies revealed white matter lesions in 60%, cerebral atrophy in 55%, ischemic stroke and intracerebral hemorrhage in 14% each. 84% had ≥2 of the major clinical and/or neuroradiological abnormalities. Outcome was unfavorable (GOS1-3) in 68% and mortality 20%. Background activity and TWs characteristics were not associated with outcome. Absence of background reactivity was associated with death (OR4.3, 95%CI 1.5-12.5, p=0.007).

Conclusions: In contrast to earlier studies with a high mortality, death occurred in our cohort in 20%. The only EEG feature associated with death was the absence of background reactivity;increased frequency, lag direction and TW amplitudes were not.

S21

Clinical, Imaging and EEG Correlates in Encephalopathy

Raoul Sutter, MD, Peter W. Kaplan, MB, BS, FRCP, Robert D. Stevens, MD

Objectives: To identify associations among encephalopathy EEG patterns, clinical and neuroradiological abnormalities, and outcome in encephalopathy patients.

Methods: 5-year cohort study of EEGs of 154 encephalopathic patients that were classified into five predefined patterns: persistent background slowing (theta, theta/delta, delta) with or without episodic transients (i.e., triphasic waves [TWs] or frontal intermittent delta activity [FIRDA]). Associations among EEG patterns, clinical pathologic conditions, blood tests, neuroimaging, and outcome were evaluated. Glasgow Outcome Score >3 at discharge was defined as favorable and 1-3 as unfavorable outcome.

Results: In multivariable analyses, theta pattern was associated with brain atrophy (OR 2.6, p=0.020); theta/delta with intracerebral hemorrhages (OR 6.8, p=0.005); FIRDA with previous cerebrovascular accidents (OR 2.7, p=0.004); TWs with liver or multi-organ failure (OR 6, p=0.004; OR 4, p=0.039); and delta activity with alcohol/drug abuse, and HIV-infection (OR 3.8, p=0.003; OR 9, p=0.004). TWs were associated with death (OR 4.5, p=0.005); theta/delta with unfavorable, and FIRDA with favorable outcome (OR 2.5, p=0.033; OR 4.8, p=0.004).

Conclusions: Well-defined EEG patterns in encephalopathy are associated with specific pathological conditions and outcome. Prospective studies are needed to clarify the contributions of respective pathologic conditions to specific EEG encephalopathy patterns.

S22

EEG and MRI Patterns in Encephalopathy

Angela Wabulya, MD, Ronald P. Lesser, MD, Rafael Llinas, MD, Peter W. Kaplan, MB, FRCP

Introduction: Using histology and EEG, Gloor et al reported paroxysmal synchronous discharges (PSDs) in cortical grey (CG) and "sub cortical" grey (SCG) matter pathology, non-paroxysmal polymorphous delta activity (NPDs) in white matter(WM) pathology, a combination of PSDs and NPDs in CG, SCG and WM pathology and cortical background slowing (BGS) in all patients with encephalopathy.

Methods: Retrospective case control study; we blindly reviewed EEG and MRI data acquired within 4 days of each other of 47 cases (encephalopathy on EEG) and 41 controls (normal EEG). Age range: 18-89 years with 46 women and 39 men.

Results: 75% of cases with PSD+BGS had SCG lesions; while 25% had CG lesions. 0% of cases had PSD+BGS and SCG+CG compared to 25% reported by Gloor etal. 72% of cases had WM lesions vs. 48.8% controls. 4.6% cases had NPD; of these 50% had WM and BGS+NPD while the remaining 50% had NPD without WM lesions. 93.6% of cases had EEG BG slowing (BGS).

Conclusion: EEG PSD were found with either CG or SCG MRI abnormality. A combination of CG and SCG MRI abnormality was not necessary to predict EEG PSD. Findings in NPDs were similar to what Gloor et al described.

S23

Alternating Low Frequency EEG Pattern in Critically Ill Patients

Hiba Arif, MD, Suzette M. LaRoche, MD

Background: We describe the occurrence of a novel EEG pattern in critically ill patients consisting of regular intervals of generalized, high amplitude delta activity alternating with regular intervals of low amplitude theta frequencies.

Methods: Patients with an alternating low frequency (ALF) EEG pattern were identified and compared to all patients undergoing continuous EEG monitoring over a 3-year period.

Results: An alternating low frequency (ALF) pattern was noted in 30 patients. Electrographic seizures were seen in 33.3% (10) with ALF compared to 18.1% of the general cEEG population. Lateralized periodic discharges (LPDs) were present in 73.3% (22) vs.8.6% and generalized periodic discharges (GPDs) in 56.7% (17) vs. 11.2%. Subarachnoid hemorrhage (SAH) was the primary diagnosis in 36.7 vs. 16.5%. At discharge, poor outcome (expired or discharged to hospice, long term care or subacute nursing facility) was seen in 53% of both patient groups.

Conclusions: In this small cohort of patients, electrographic seizures and periodic discharges were seen more frequently in patients with ALF compared to the general cEEG population and SAH was more prevalent. However, there was no difference in outcome between patient groups. Further study regarding the clinical significance of this pattern is warranted.

S24

Myopathy Masquerading as Bilateral Anterior Interosseous Syndrome

Elizabeth Ann Mauricio, MD, Elliot Dimberg, MD, Devon I. Rubin, MD

Background: We report a patient who presented with suspected bilateral anterior interosseous neuropathies (AION) but electrodiagnostic studies identified a myopathy, consistent clinically with inclusion body myositis.

Case Report: A 79 year old woman was referred for EMG following orthopedic consultation for bilateral AIONs. She had a 3 year history of difficulty flexing the distal thumb, index, and middle fingers (right > left). A cricopharyngeal myotomy was performed for dysphagia several years prior; she had no other complaints. Examination demonstrated bilaterally severe weakness in flexor digitorum profundus (digits 2 and 3) and flexor pollicis longus, mild weakness in FDP digits 4 and 5 and the flexor digitorum superficialis, and normal strength in other muscles.

Results: Median and ulnar nerve conduction studies were normal. Needle examination revealed scattered fibrillation potentials and

short duration, polyphasic motor unit potentials in multiple upper and lower extremity muscles. The flexor digitorum profundus demonstrated a combination of rapid and reduced recruitment in different areas of the muscle. The findings were consistent with a chronic myopathy.

Conclusion: This case demonstrates a unique pattern of weakness clinically mimicking AIONs in a patient with a chronic myopathy. The importance of a careful needle EMG to confirm a myopathy is exemplified.

S25

Gadolinium-Induced Refractory Nonconvulsive Status Epilepticus

Nasheed Jamal, MD, Saumya Gill, MD, Claude Wasterlain, MD

Intrathecal administration of high-dose gadolinium can lead to neurological complications, including encephalopathy, seizures, rigidity, and optic nerve atrophy. Here, we present a case of refractory nonconvulsive status epilepticus following accidental injection of gadolinium via an external ventricular drain prior to an MRI brain scan. The patient is a 59 yo male, who was status post right temporal craniotomy for microsurgical resection of large right tentorial meningioma post endovascular coil embolization of feeder vessels to right tentorial meningioma, who received an intrathecal administration of 10mL volume of gadolinium via an external ventricular drain and subsequently became hypertensive and unresponsive. Continuous EEG revealed nonconvulsive status epilepticus (NCSE) likely from an epileptogenic zone at the right temporal region (T4) with some spread to the right frontal region (F4). His seizures continued for 7 weeks despite a treatment regimen that, in various combinations, included: levetiracetam, phenytoin, valproic acid, midazolam, propofol, pentobarbital, ketamine, lacosamide, topiramate. Phenobarbital infusion and subsequent prolonged burst suppression finally halted the seizures. At discharge, the patient was able to follow simple commands intermittently. This case thus demonstrates that CNS infusion of high-dose gadolinium may lead to refractory nonconvulsive status epilepticus.

S26

Early Use of Newer Anti-Seizure Agents in the Treatment of Status Epilepticus (SE)

Uma Menon, MD, Fawad Khan, MD, Vivek Sabharwal, MD, Arash Afshinnik, MD, Eugene Ramsay, MD

Background: Limited information is available regarding the use of newer anti-seizure agents Levetiracetam and Lacosamide as initial treatment of status epilepticus. Availability of parenteral forms with good pharmacokinetics and safety profile make the newer agents logical choices for early rather than later use.

Methods: We conducted a retrospective analysis of the agents used for convulsive and non-convulsive status epilepticus in our institution during one month, to assess the utility and efficacy of Levetiracetam and Lacosamide combined as initial agents for status epilepticus. Propofol and Ketamine were used when appropriate and other anti-seizure agents were infrequently used.

Results: In the ten patients identified, 5 with convulsive and 5 with non-convulsive SE, the combination of Levetiracetam and Lacosamide was used as first line agents after Benzodiazepines failed. Five patients required additional Propofol and two required Ketamine. The combination resulted in resolution of SE in all patients except one with withdrawal of care. No significant drug adverse effects were observed.

Consclusions: Although status epilepticus can be appropriately and favorably managed with different combinations of anti-seizure agents, our goal was to evaluate the use of Levetiracetam and Lacosamide in combination as a good alternative to the older agents as an effective first line treatment.

S27

Psychogenic Non-Epileptic Seizures: Comparison of Clinical Manifestations between Afro-American and Caucasian Female Patients

Abuhuziefa Abubakr, MB, FRCP, Ilse Wambacq, PhD, Hanna Goerres, BS

Rationale: Psychogenic non-epileptic seizures (PNES) represent an important alternative diagnosis for refractory epilepsy. In various series, the frequency of PNES range between 10-40%. However the frequency and clinical manifestations are not well characterized in AAF (Afro-American females). Therefore we compared various clinical findings between women of AAF and Caucasian descends admitted to the EMU.

Methods: Retrospective chart review of all patients admitted to the EMU between January 2010 and December 2011 were included. Female patients 18 years or older with the diagnosis of PNES were selected. The demographic information, age of onset, seizures duration, frequency and clinical characteristic were evaluated.

Results: There were 18 AAF with mean age of 44.9 years (age range (18-72 yrs) and 27 Caucasian females (CF) with mean age of 37.6 years (age range 18-83yrs). There were 13 AAF (72%) and 23 CF (85%) under 50 years of age. Six AAF and twelve CF were younger than 35 yrs at seizure onset; seven AAF and eleven CF were between 35-50 yrs at seizure onset and five AAF and four CF were older than 50 yrs at seizure onset. The duration of PNES was less than one year in 6 AAF and 9 CF, between 1-5 yrs in 6 AAF and 8 CF and more than 5 yrs in 6 AAF and 10 CF. Daily / Weekly seizures occurred in 50% of AAF and 78% of CF which is significantly different between the two groups ($P < 0.05$). Sixteen of AAF (88%) and 18 CF (66%) were on 1-2 AEDs, but only 4 CF were on > 3 AEDs. There were 13 AAF (72%) and 15 CF (55%) with seizures lasting < 5 minutes. However, seizures with the duration between 5-10 minutes occurred in 17% of AAF and 26% of CF and seizures with duration >10 minutes in 11% and 19% respectively. The clinical manifestation of seizures with limpness and unresponsiveness occurred in 5 AAF and 7 CF. However, predominant motor manifestations occurred in 12 AAF and 19 CF and this was the most frequent clinical manifestation in both groups. Pelvic thrust-

ing occurred in 4 AAF and 9 CF and it was twice as frequent in CF. Vocalization occurred in 2 AAF and 4 CF.

Conclusion: The clinical manifestations of PNES between AAF and CF were similar with exceptions that in AAF the seizures were significantly less frequent and there was a tendency

---

S28

A Case of Very Prolonged Todd's Paralysis for 15 Days

Hyun Joo Sophie Cho, MD, Robert L. Beach, MD, PhD

Background: Todd's paralysis is a condition characterized by transient post-ictal focal neurological deficits. The mechanism underlying Todd's paralysis remains uncertain. The longest duration of reported Todd's paralysis was 36 hours.

Objectives: Here we describe the case of 58 year-old-man who presented with prolonged Todd's paralysis lasting for 15 days.

Methods and Results: A 58 year-old man with history of complex partial epilepsy from right frontal lobe metastatic lung cancer was admitted to our hospital for breakthrough seizure. On exam, he had global aphasia, right side face, arm and leg flaccid paralysis and right hemianopsia. 2 MRIs of the brain were negative for acute stroke in 10 days. Continuous EEG showed marked asymmetry, with reduced amplitude on the left, but no seizure or epileptiform discharges. SPECT obtained on day 7 showed relative increased perfusion within the left parietal lobe. The aphasia, right hemianopsia and facial palsy started resolving on day 10. Right arm and leg paralysis started improving on hospital day 15.

Conclusions: This is the first case in which Todd's paralysis lasted for 15 days. The mechanism of this prolonged Todd's paralysis is unclear. We speculate that our patient had mismatched cerebral perfusion and metabolic activity by altered cerebrovascular autoregulation.

S29

Epilepsy Associated with Perisylvian Migrational Defects: Widely Divergent Clinical Outcomes

Olimpia Carbunar, MD, MS, Alma Bicknese, MD

Perisylvian migrational defects have been linked to multiple genes, although most cases still do not have an identified etiology. It is often assumed incorrectly that patients will have poor outcome with intractable seizures and developmental delay. We present two cases with similar perisylvian migrational defects, but with different clinical phenotypes and different EEG patterns. Our first patient had oral motor dysfunction with pseudobulbar signs, developmental delay and refractory seizures. Our second patient had normal development and exam, and mild epilepsy that went into remission. Migrational defects involving the insular cortex are associated with dysarthria and difficulties with palatal and lingual movements. There is a high association with epilepsy and it may be difficult to control. However, others lack dysarthria and may have mild or no epilepsy. Clinicians and epileptologists should aware of the wide clinical spectrum of these disorders and their clinical implication when discussing outcome with patients and their families.

S30

Comorbidities May Help Predict Outcomes in Status Epilepticus

Lukas Clark, MD, Makoto Kawai, MD, Amit Verma, MBBS

This case-control study examined whether common co-morbid conditions > help predict outcomes in status epilepticus. Patients with either > partial or generalized status epilepticus were selected based on EEG > review of 710 continuous recordings at The Methodist Hospital from > January 2008 through January 2012. In all, 59 patients were included > (23 men, 36 women, mean age=59.6), of which 51 were noted to be in > partial status epilepticus, and another 8 were in generalized > non-convulsive status epilepticus. Prognosis at the time of discharge > was classified with the Glasgow-Pittsburgh Cerebral Performance > Category (CPC). These CPC scores were compared with control populations using the Kruskal-Wallis test for statistical significance. > Hepatic dysfunction in 7 patients (with and without renal > insufficiency) correlated with poorer outcomes compared to controls. > (median CPC = 5 (5,5), mean = 4.71, compared with median CPC in controls = 2 (2,3), mean = 2.38). > Generalized status epilepticus patients also faired worse than their > partial status epilepticus counterparts (p=0.00299). Other factors, > including renal insufficiency (p=0.075), heart failure (p=0.492), > intracerebral hemorrhage (p=0.991), stroke (p=0.607), tumor (p=0.585), > hypertension (p=0.974), diabetes mellitus (0.273), urinary tract > infection (p=0.18), and obesity > (p=0.516) failed to achieve statistical significance.

S31

Hippocampography & Lesional Dominant Neocortical TLE Surgery

Marcus Ng, MD, FRCPC, Ronan Kilbride, MD, Mirela Simon, MD, Emad Eskandar, MD, Andrew J. Cole, MD, FRCPC

Symptomatic lesions account for a large proportion of temporal lobe epilepsy (TLE) cases. Not uncommonly these lesions are located outside the hippocampus in temporal neocortex. When epilepsy is medically refractory, the treatment of choice is surgery. Resection usually includes the mesially located hippocampus, especially if the hippocampal electrocorticogram ("hippocampogram") demonstrates epileptiform abnormalities. However hippocampectomy in the dominant hemisphere entails considerable risk to neuropsychological function, such as memory, in addition to the general hazard of more extensive neurosurgery. We sought to determine the relation between seizure freedom and findings on electrocorticography in dominant temporal neocortical lesional epilepsy cases and to determine whether hippocampal recording is helpful in determining extent of hippocampal resection in patients with neocortical lesions. In a retrospective chart review, we found 6 patients who underwent hippocampography during surgery. We will present the hippocampography, radiology, pathology, extent of

resection, and Engel score for each patient. We will interpret these findings in relation to the role of hippocampography and hippocampectomy in achieving seizure freedom for these patients.

S32

Continuing Challenges in Diagnosing Frontal Lobe Epilepsy

Rajbeer Singh Sangha, Robert L. Beach, MD, PhD

Introduction: Frontal Lobe epilepsy is difficult to diagnose due to the unusual presentations and lack of a post ictal period that characterizes temporal lobe epilepsy.

Results: 33 y/o female with vigorous motor seizures was admitted after multiple EEGs couldn't were negative. VEEG captured clinical events, only be clearly identified as seizures after AEDs were stopped and multiple events with stereotypical semiology, but poorly localizing rhythmic theta or alpha activity were captured. She presented several years later to a different epileptologist, with spells of unresponsiveness, "shaking and thrashing". Suspicion for psychogenic non-epileptic seizures prompted VEEG monitoring. After 2 vigorous spells she classified as nonepileptic and her AEDS were reduced. Later the patient presented in complex partial status. On VEEG, suspicious poorly localizing activity in the left frontal central head region suggested insular onset. Her AEDs were modified with improved control.

Conclusion: Despite multiple VEEG recordings, diagnosis of frontal lobe epilepsy can be markedly difficult. The diagnosis may require characterization of stereotypical behavior from multiple events in the absence of clear ictal electrographic abnormalities.

S33

Reorganization of the Background ECoG Underlies Afterdischarge

Giridhar Kalamangalam, MD, DPhil

Afterdischarges (ADs) are runs of focal epileptiform activity following cortical electrical stimulation (CES). AD occurrence depends on stimulus intensity, brain location and pre-existing low-frequency power in the background electrocorticogram (ECoG); how ADs occur following CES however remains unclear. We reviewed extraoperative CES ECoG data following 1079 bipolar stimuli in four patients undergoing presurgical subdural grid evaluation. Thirty-eight ADs lasting ~ 8 seconds were analyzed for spectral content, and compared to those from length-matched segments of baseline ECoG and following subthreshold stimuli. A consistent relation of AD-to-baseline spectra was found, in one of three ways: (i) a "condensing" relation, with AD and baseline spectral peaks appearing at the same locations, but AD peaks showing less dispersal; (ii) condensation plus harmonics, with additional peaks appeared at selected harmonics and (iii) harmonic-dominant, with high harmonic content and minimal condensed peak power. Spectra of epochs following subthreshold stimuli differed from baseline in a similar though less prominent fashion. Thus ADs, rather than representing de novo change, fundamentally arise from a "reorganization" of background brain rhythms, comprising "condensation" of pre-existing peaks and a process generating higher harmonics of the condensed peaks. Possible underlying neurophysiological mechanisms and a quantitative model for this phenomenon are proposed.

S34

Oxygen Saturation in Epileptic and Non-Epileptic Convulsive Spells

Gowri Lakshminarayan, MD, Bruce J. Fisch, MD

Patients with psychogenic non-epileptic seizures (PNES) that present with attacks in the emergency department are frequently misdiagnosed, resulting in the administration of antiepileptic medication, occasionally with intubation and general anesthesia. We hypothesized that oxygen monitoring might be helpful to emergency physicians and first responders in distinguishing epileptic from non-epileptic attacks. To test this hypothesis we performed an unblinded, retrospective study of the variability of oxygen saturation in the pre-ictal (30 sec before onset), ictal, early post-ictal (30 sec) and late postictal (up to 3 min) phases in patients undergoing video EEG monitoring during convulsive epileptic (12 patients, 15 seizures) and non-epileptic spells (7 patients, 10 attacks).

Results: The lowest desaturation in the PNES group was 86% (in 2 of 10 attacks; nadir > 90% in 6 of 10). All epileptic seizures had a nadir below 90% with 10 of 15 <= 80% (lowest recorded 64%). In 8 of 10 PNES attacks the lowest saturation was in the ictal period, vs 12 of 15 epileptic seizures with the nadir during the postictal period (example below).

Conclusion: The temporal pattern and degree of 02 desaturation may be diagnostically useful in distinguishing epileptic from PNES convulsive attacks.

S35

PESSt and H Reflex in 24 Patients with Urinary Incontinence Before Lumbosacra Trauma. Comparative Study at Three Months.

**Teresa Maria Montes de Oca Domingo, Juan Manuel Rojas de Dios, Javier Enrique Garcia Cordero, Idalme Padron Lopez, Gladys Maya Morales, Olga Gonzalez Perez*

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 of nerve tibials in patients with urinary incontinence transitory posttraumatic of column lumbosacra without section medullar in initial stages and study comparative at three months. Methods: Study of H Reflex and PESSt with analysis of central conduction in 24 patients, between 24-42 years of age with urinary incontinence transitory posttraumatic of column lumbosacra without section medullar in initial stages (21 days of trauma).

***Travel Fellowship Award Recipient:** The Travel Fellowship Award is given to the most outstanding poster submitted by a Fellow who is the First Author.

Results: H Reflex was absent bilateral in 18 patients and slowed down in the rest. PESSt and TCC was absent in 21 patients. The comparative study in 3 months, demonstrates H Reflex absent in 12 patients and the PESSt in 15 patients.

Conclusion: The alterations in initial stages of this pathology could evaluate the susceptibility of urodynamic in the incontinence urinary in the course of affections medullar

S36

Simultaneous EEG and fMRI Correlation of Focal Slowing in Temporal Lobe Epilepsy

Rohit A. Marawar, MD, Hsiang J Yeh, BS, Christopher Carnabatu, BS, John M. Stern, MD, Gautam Tammewar

Objective: Characterize the focal abnormality of temporal lobe epilepsy (TLE) with fMRI through EEG evidence of continual dysfunction instead of episodic epileptiform discharges.

Background: Simultaneous EEG and fMRI (SEM) studies in TLE have correlated spikes with fMRI, but spike yield and significant results are limited.

Methods: 11 Left TLE, 8 Right TLE and 14 controls underwent SEM. We measured power spectral analysis in delta band in selected electrodes representing temporal lobes bilaterally. Average power of 2 second epochs was convolved and correlated to yield fMRI maps which were then coregistered on MRI images.

Results: Controls' temporal delta activity showed extensive activation of bilateral temporal, occipital and parietal lobes with minimal activation of frontal lobes and thalamus without significant difference between left and right sided electrodes. Subtraction images of control greater than LTLE showed activation of bilateral anterior temporal lobes while LTLE greater than control showed activation of thalamus.

Conclusions: Temporal region delta generation is a function of diffuse network in normal subjects. This network is disrupted in LTLE with a shift of functional activity from temporal lobes to thalamus.

S37

Interventional Neuroradiology Intraprocedural Clot Series

David S. Gloss, MD, Brian Alkire, CNIM

We performed an IRB approved retrospective chart review at Barrow Neurologic Institute from 2006-2012, looking for intraprocedural clot formed during interventional neuroradiology procedures (including coils, stenting, vasospasm procedures), which included intraoperative monitoring. We found 15 such cases. In 6, SSEPs dropped significantly in amplitude before the clot was seen on angiogram, allowing recognition of the clot and intervention more quickly than if there were not monitoring. In another seven, it is unknown if the SSEP change happned before being seen on angiogram. In all 15 cases, there were significant amplitude drops in SSEPs. In addition, in two of the cases, there was a significant increase in latency. In 13 of the 15 cases, there was at least partial recovery after intervention on the clot; for example, injecting abciximab directly on the clot. In 6 cases, the baseline waves returned, in 7, there was partial recovery. In 2 cases, the waves remained absent. This retrospective series needs to be verified with a prospective cohort, but it suggests that monitoring interventional radiology procedures is a safe and effective way to determine the occurrence of intraprocedural clot formation, which may allow for intervention before would have otherwise been noticed on the angiogram.

S38

Multi-Myotomal MEPs Can Improve the Identification of Segmental Spinal Cord Injury During Surgical Instrumentation

Jose Fernandez, Dr, Alfredo Traba, Dr, Oscar Riquelme, Dr, Azucena Garcia, Dr, Jose Luis Gonzalez, Dr

Motor evoked potentials (MEPs) have gained increased recognition as an essential component of multimodality intraoperative monitoring (IOM) during spinal surgeries with deformity correction. Although the critical period of MEPs monitoring have been traditionally considered the deformity correction, several reports have shown spinal cord injury during instrumentation. In this context multi-myotomal MEPs recordings may be usefully. We report two cases of abrupt MEPs loss after screws were placed at T7 and T9 pedicles. MEPs were bilaterally recorded from abductor pollicis brevis, mid - axillary chest at upper and mid thoracic levels, external oblique abdominis, vastus lateralis, tibialis anterior and abductor hallucis muscles. In both patients instrumentation of the pedicle resulted in loss or reduced amplitud of lower limbs MEPs. External oblique abdominis MEPs were loss only after T7 instrumentation. Abductor pollicis brevis and mid axillary chest MEPs were stable and unchanged throughout the surgical course. Pedicle screw removal resulted in total or partial reversal of MEPS changes. There were no postoperative neurologic deficits. These results suggest that multi-myotomal MEPs monitoring during segmental instrumentation can detect reversible spinal cord injury, minimizing postoperative deficits.

S39

Critical Intraoperative Neurophysiologic Monitoring (IONM) Changes Associated with Patient Positioning Maneuvers

Leslie H. Lee, MD, S. Charles Cho, MD, Viet Nguyen, MD, John Ratliff, MD, Jongsoo Park, MD, Griffith Harsh, MD, Jaime R. López, MD

Introduction: Positioning maneuvers during surgical cases can place the patient at risk for spinal cord and/or peripheral nerve injury. Initial transition of the patient from the supine to prone position, as well as passive neck flexion or extension, are potentially high risk portions of the procedure, especially during spine surgeries. The role of IONM in helping to prevent such injuries is emphasized.

Methods: We present a series of six cases where critical IONM changes were identified and resolved following modification of patient positioning in cervical spine (5) and intracranial (1) procedures.

Results: Significant IONM changes were observed during the initial prone positioning onto the surgical table in four spine cases, while in another case changes occurred with passive neck extension while supine. During an intracranial surgery IONM changes were observed during tumor resection that resolved with modification of neck positioning. In all cases prompt identification of IONM changes enabled rapid assessment and repositioning of the patient, which largely resolved all neurophysiologic changes and correlated with no new sustained postoperative deficits.

Conclusion: This series highlights the importance of appropriately instituting IONM early, prior to patient positioning, to facilitate the prompt identification of potentially reversible changes that may indicate impending positioning-related injuries.

S40

Neurophisiologic Detection of the Spinal Cord Lesion Level

Lidia Cabanes-Martinez, MD, Gema de Blas, MD, PhD, Elena Montes, MD, Nelson Cuellar, MD, Jesus Burgos, MD, PhD, Carlos Correa, DMV, Jaime R. López, MD

Introduction: In our experience, most of the spinal cord injuries in spinal surgeries have compressive mechanical causes. Localization of the lesion level would allow immediate decompression, improving the chances of recovery.

Material And Methods: In five experimental pigs the thoracic spinal cord was exposed in three segments via bilateral laminectomies. Four sublaminar epidural catheters were placed at T3, T3, T11 and L1. The following techniques were performed: spinal cord to spinal cord evoked potential (EP), D-wave recordings and somatosensory epidural EP. Then, the spinal cord was severed at the T8 level, and the neurophysiologic protocol was repeated.

Results: In all cases, the cord to cord EP was absent when stimulating the two levels above the lesion and recording at the two distal levels. The epidural sensory EP was normal in the two distal levels, and absent in the proximal levels. D wave was present in all cases at the two levels proximal to the lesion, and absent in the distal ones.

Conclusions: It is possible to identify the level of the spinal cord lesion by neurophysiologic techniques. The cord to cord, D-wave and sensory epidural potentials recorded at different levels allow us to exactly identify the injury level.

S41

Reversible Intraoperative Neurophysiologic Monitoring (IONM) Changes Associated with Surgical Retraction

Leslie H. Lee, MD, S. Charles Cho, MD, Viet Nguyen, MD, Gary K. Steinberg, MD, PhD, Steven D. Chang, MD, Robert Dodd, MD, PhD, Stephen Ryu, MD, Jaime R. López, MD

Introduction: Inadvertent retraction-related injuries are a known risk of intracranial surgical procedures. Retraction in the vicinity of critical neural tissue and vascular structures may result in compression, stretch, or steno-occlusive ischemic injuries that are unexpected, and may not be recognized until the postoperative period. The role of intraoperative neurophysiologic monitoring (IONM) in helping to prevent such injuries is highlighted.

Methods: We present a series of eight intracranial surgical cases where IONM changes occurred in association with retractor placement and positioning, procedures that include resection of tumor (3) and vascular malformations (2), and aneurysm clipping (3).

Results: In the three aneurysm cases significant IONM changes occurred following retractor placement, but prior to any planned intervention. In all remaining cases changes occurred during the interventional period that also coincided with the placement or positioning of retractors. Most commonly transcranial motor evoked potentials were primarily affected. In all cases prompt identification of these IONM changes led to rapid surgical assessment and eventual removal or repositioning of retractors, which resolved neurophysiologic changes and correlated with no new sustained postoperative deficits.

Conclusion: This case series highlights the importance of IONM in the early identification of potentially reversible changes that may correlate with impending retraction-related injuries.

S42

Impact of Technique Employed in Motor Evoked Potential Studies of the External Anal Sphincter on Obtaining Reliable Responses

Hos Loftus, MD, Benjamin Cohen, MS, CNIM

Background: The best method to obtain motor evoked potentials in the external anal sphincter (EAS) has not been established in literature. This review was performed to examine the relationship between the technique and responses.

Methods: The data from a series of 16 cases which has EAS included in transcranial motor evoked potentials for spine surgeries were analyzed retrospectively.

Results: 6 cases had a single EAS channel among either right or left side channels; 3 had the same channel checked with stimulation of both sides; 7 had independent right and left channels. Of first group, 4 had responses initially; 2 were initially unobtainable but later emerged. Of second group, 1 had no responses; 1 had no responses initially but they emerged bilaterally; 1 had responses only on one side. Of third group, 2 had no responses; 2 had no responses initially, which later emerged bilaterally; 1 had responses on one side and the other later emerged; 1 was absent initially, only one side emerged later. Of 10 cases that had responses checked bilaterally, 4 had responses only on one side for part of, or the entire, study.

Conclusion: Checking EAS motors bilaterally may be more helpful in obtaining responses.

S43

Epidural Recording: New Technique for Malpositioned Pedicle Screw Detection

Nelson Cuallar, MD, Gema de Blas, MD, PhD, Lidia Cabanes-Martinez, MD, Jesus Burgos, MD, PhD, Miguel Anton, MD, Eduardo Hevia, MD, Carlos Barrios, MD, PhD

Introduction: Available methods for screw monitoring fail to detect approximately 15% of the malpositioned screws. We have developed a new method based on spinal cord recording in response to pedicle screw electrical stimulation.

Patients and Methods: We studied 123 thoracic screws from 6 patients with idiopathic scoliosis. Following the classic single pulse stimulation of the screws, we performed train stimulation using 4 stimuli, 0.2 sec duration, 500 Hz rate, with a decreasing intensity from 30mA. The response was recorded with two epidural catheters placed cranial and caudally. Additionally, the placement of the screws was guided by intraoperative imaging techniques. After surgery, a CT scan was performed.

Results: 6 screws were removed due to malpositioning according to conventional screw neurophysiologic monitoring techniques. 99 screws (84.6%) showed correct placement by CT imaging, while 18 (15.4%) were invading the canal. Of these, 7 (39%) showed an epidural response with a threshold below 15mA, indicating proximity or contact with neural structures. 11 screws showed normal thresholds. None of these patients have had postoperative symptoms.

Conclusions: The data from this technique suggests an approximately 40% improved rate in detecting malpositioned pedicle screws.

S44

SSEPs: A Comparison of Cervical Spine Dysfunction

Vivian Hoang, MD, MBA, Michael Dorsi, MD, Langston Holly, MD, Marc R. Nuwer, MD, PhD

Cervical stenosis leading to myelopathy is one of the leading causes of spinal cord dysfunction. Many patients with cervical spine disease often have to proceed to surgical procedures to alleviate their symptoms. At our institution, many of the surgical cervical spine procedures are monitored intra-operatively by somatosensory evoked potentials (SEP) obtained from all four extremities. We are interested in the difference in SEP latencies of cervical myelopathy patients as compared to other cervical procedure patients. Using cervical radiculopathy patients who receive surgery as a comparison group, we analyzed the absolute latencies of the lower extremities' SEP with adjustment for a patient's height. This data was then compared to normative data for absolute lower extremity latencies adjusted for height. Our preliminary data from 109 cervical myelopathy patients and 25 cervical radiculopathy patients indicates that the myelopathy patients had mildly greater absolute latency (mean = 46.32) as compared to the radiculopathy patients (mean = 42.07). When these latencies were adjusted for height and compared to normative data, the latencies from the myelopathy patients were more often prolonged or at the higher limits of normal. As a next step, we plan to assess whether these SEP findings correlate with MRI and functional status.

S45

IOM for Intracranial Aneurysms: The Michigan Experience

Kinshuk Sahaya, MD, Aditya S Pandey, MD, Brian R. Bush, BS, Daniela Minecan, MD

Objective/ Methods: Present the association between neurological outcome of patients with intra-cranial aneurysms (ruptured/unruptured) and intraoperative monitoring (IOM) during endovascular/transcranial repair. Standard IOM practices were followed. The cases associated with IOM changes were retrospectively mined from our database.

Results: 406 subjects underwent 470 procedures. Total 331 (70.4%) procedures were performed on patients with unruptured aneurysms. Endovascular procedures were performed in 56.8% cases. Somatosensory evoked potentials (SSEP) were used in all patients and EEG, brainstem EP (BAEP) in 99.14% and 23.19% respectively. EMG, visual EP (VEP) and motor EP (TcMEP) were rarely used. Changes occurred in 4.4% of procedures. Majority (85.7%) were detected on SSEPs followed by BAEP. 11 (52.4%) were reversible, while 10 were not or only partially reversible. 10 patients experienced immediate post-operative deficits, while 11 had none. Amongst patients without deficits, the changes were either reversible (81.8%) or partially reversible (18.2%). Amongst patients with deficits, 60% of changes were irreversible, 20% partially reversible and 20% reversible.

Conclusion: SSEPs remain the most effective modality to detect changes during aneurysm repair. Irreversible changes exclusively occurred in patients with immediate post-operative deficits while reversible changes were higher in patients without post-operative deficits. Partially reversible changes were comparable in both groups.

S46

Contralateral Response to Lumbar Pedicle Screw Stimulation

Parastou Shilian, DO, Adel Olshansky, MD, Andres A Gonzalez, MD

Goal: To report an intriguing finding observed during pedicle screw triggered electromyography (tEMG).

Background: Lumbosacral surgery often involves placement of screws into the pedicle wall to provide stability and/or to correct deformity. Neuromonitoring including tEMG is used to assure the correct placement of pedicle screws and to avoid injury to the nearby neuronal and vascular structures. Case description: Patient underwent posteriolateral fusion with instrumentation, L2 to L5. Following screw placement tEMG was performed. Right L2 screw stimulation, an EMG response was seen in the right vastus medialis with threshold of 9mA. Stimulation of the left L2 pedicle screw stimulation showed no responses ipsilaterally, but an EMG response

was seen in the right vastus medialis with the same threshold of 9mA. Electrodes were checked for connection to appropriate side. Intraoperative radiograph showed the tip of the right L2 pedicle screw projecting medially and appearing to abut the tip of the left L2 pedicle screw. Right L2 screw was repositioned with pedicle screw stimulation having a threshold above 20 mA bilaterally.

Discussion: A contralateral EMG response was seen with pedicle screw stimulation with the same threshold as the ipsilateral breached pedicle screw. This observation should raise the possibility of a hardware bridge.

S47

Median Nerve SEPs in Carotid Surgery: Does Reference Choice Matter?

Stephen Fried, MD, Diane Smith, CNIM, Alan D. Legatt, MD, PhD

Median nerve somatosensory evoked-potential (SEP) monitoring is commonly used during carotid endarterectomy surgery in order to permit selective shunting in only those patients who are determined to have inadequate collateral flow following carotid cross-clamping. The N20 component is recorded from the CPc (contralateral centroparietal) electrode; either CPi (ipsilateral centroparietal) or FPz (forehead) can be used as the reference. Due to the distribution of the subcortically-generated N18 component, the CPc-FPz derivation might record both the N20 and N18 components, and might therefore inadequately detect hemispheric ischemia following carotid cross-clamping. We compared SEPs recorded using these two derivations during 38 carotid endarterectomies, in order to assess their ability to detect neurophysiologic changes after carotid cross-clamping. Although, as expected, the baseline N20 component was significantly larger when recorded with the CPc-FPz derivation than with the CPc-CPi derivation (3.1 μV vs. 2.4 μV in the hemisphere ipsilateral to the clamped carotid, $p<0.001$), there was no significant difference in the post-clamp amplitude decline between the two derivations (8.7% vs. 8.6%, $p=0.82$). We conclude that CPc-FPz is an acceptable derivation for recording post-clamp hemispheric SEP changes during carotid endarterectomy surgery, and may be advantageous because it provides a larger-amplitude SEP than the CPc-CPi derivation.

S48

Comparison of Continuous 32 Channel EEG Monitoring and SSEPs Monitoring with Burst Suppression during Carotid Endarterectomy (CEA) in Predicting Surgical Outcome

Anishee Shah, MD, Robert Zelaya, Technician, Evgeny Tsimerinov, MD, Abirami Muthukumaran, MD, Lilit Mnatsakanyan, MD

Objective: To re-present with the addition of 16 patients the reliability of different monitoring methods during carotid endarterectomy.

Background: Carotid endarterectomy (CEA) is a commonly performed procedure. Various intraoperative monitoring (IOM) modalities are used to detect early signs of ischemia during the critical portions of a surgical procedure. Continuous EEG (cEEG) can detect the earliest changes while burst suppression during clamping (sEEG) offers theoretical benefit of neuroprotection. Somatosensory evoked potentials (SSEPs) have been shown to be more sensitive and specific in detecting changes but with a time lag in detecting the changes during acquisition of the data. To date, there are no established guidelines for the best IOM approach during CEA.

Methods: 38 individuals with symptomatic and asymptomatic carotid stenosis who underwent carotid endarterectomy at Cedars Sinai Medical Center. Neurological function and intraoperative neurophysiologic monitoring data were obtained from medical records and retrospectively reviewed. Subjects were divided into 2 non-randomized groups based on surgeons' preference, either cEEG or burst-suppression EEG with SSEP monitoring during clamping. Primary outcome was measured by comparing the neurological functions at baseline, post-anesthesia recovery, and discharge. Intraoperative changes were identified and correlated to primary outcome for analysis.

Results: Both cEEG and sEEG+SSEPs were shown to reliably monitor electrophysiologic changes during CEA in our study.

S49

Long-Term Evolution of Pedicle Screws Placed in the Spinal Canal During Scoliosis Surgery

Sergio Garcia-Urquiza, MD, Lidia Cabañes, MD, Nelson Cuallar, MD, Maria Soledad del Cura, MD, Elena Montes, MD, Ignacio Regidor, MD, PhD, Carlos Barrios, MD, PhD, Gema de Blas, MD, PhD, Jesus Burgos, MD, PhD

Introduction: According to the literature, 10-15% of thoracic pedicle screws are malpositioned, even when using current methods of intraoperative neurophysiologic monitoring (IONM). This occurs more frequently at the convexity of the apex of the curve. Patients and methods: Between 2005 and 2009 we performed postoperative CT scans on 28 patients who underwent scoliosis surgery. All screws were placed by the hand-free technique, and three methods (palpation, radioscopy and IONM) were performed to asses proper screw placement. In 8 patients, we found 11 screws invading the spinal canal to more than 4mm, all of them asymptomatic. We carried out radiological, neurophysiological, and clinical follow-up for an average period of 46 months, until the end of the child's growth period.

Results: The 11 screws within the canal were placed at the convexity of the apex. The triggered-EMG mean threshold was 12.48 mA (9.5-18 mA). Postoperatively, we did not find any change on the screw placement. Patients stayed asymptomatic, their neurological examination remained normal and their somatosensory evoked potentials showed no changes.

Conclusions: In our series, patients with pedicle screws placed within the canal were asymptomatic in the immediate postoperative period and did not develop long- term clinical symptoms.

S50

Complementary Nature of MEG/EEG & SISCOM in Epilepsy Surgery

Michael A. Stein, MD, Travis R. Stoub, PhD, Marvin A. Rossi, MD, PhD

MEG/EEG source localization and SISCOM are functional neuroimaging modalities that can provide localizing information in planning epilepsy surgery when the standard evaluation including MRI, continuous video-EEG monitoring, and neurocognitive evaluation is non-diagnostic. Although others have compared their relative sensitivities (Knowlton RC et al., 2008, Seo JH et al., 2011), this study presents a case series (n=35) analyzing coregistered MEG/EEG and SISCOM data with emphasis on their complementary nature. Since MEG/EEG and SISCOM provide similar but also unique information, we argue that using both in conjunction adds localizing power in planning for epilepsy surgery which should lead to improved outcomes. Both tests have high spatial resolution. Advantages of SISCOM are that it is an ictal measure, and it can localize deep sources. MEG/EEG has advantages of being a direct measure of neuronal function, and having high temporal resolution. When used together MEG/EEG-SISCOM provides information on both ictal and interictal localization with high spatial and temporal resolution. We also show how the shortcomings of one modality can be compensated for with information from the other. Finally a model incorporating MEG/EEG-SISCOM into planning for intracranial electrode placement that minimizes the extent of necessary electrode coverage and hence associated morbidity and mortality is presented.

S51

Does a Focal Irritative Zone in MEG Always Correlate with the ECoG Interictal or Ictal Onset Zone?

Angela Y Peters, MD, Paul House, MD, Michael E. Funke, MD, Pegah Afra, MD

Background: MEG, a reliable indicator of the irritative zone (IrZ), is currently used for planning of intracranial grid and strip placement. The correlation of focal IrZ as demonstrated by MEG to a focal ictal onset zone (IOZ) as demonstrated by intracranial EEG (ICEEG) is unclear.

Methods: Two patients with non-lesional neocortical epilepsy with focal MEG- IrZ underwent ECoG with grids and strips. Please see table-1 for localization of IrZ by MEG, and IrZ/IOZ by scalp-EEG/ICEEG, and table-2 for grid locations.

Results: Both patients had diffuse/regional-IOZ that were inclusive but not limited to the MEG-IrZ. Patient-1 had a large resection with complete removal of IOZ with Engel class-Ia outcome at 3 years. Patient-2 had MEG guided review of MRI pointing to a focal cortical dysplasia underwent initial lesionectomy resulting in complete removal of MEG-IrZ. She had a transient Engel-Ia outcome followed by Engel III. Shortly thereafter she had ICEEG showed a regional IOZ that was only partially resectable due to intervening eloquent cortex resulting in Engel III outcome.

Conclusion: These cases suggest that focal IrZ by MEG does not always predict a focal IOZ. Therefore MEG cannot be used to determine extent of coverage needed by intracranial grid and strips.

S52

Presurgical Functional Mapping of M1 and S1 with Dense-Array EEG

Kyle Morgan, BS, Don Tucker, PhD, Phan Luu, PhD, Mark Dow, MS

To minimize post-surgical functional deficits, presurgical localization of critical functions is required to guide surgical resections. Functional localization can be obtained using non-invasive technologies, such as fMRI, or invasive intracranial EEG (icEEG) recordings. Data from fMRI do not have the temporal resolution required for mapping of rapidly evolving functional processes and icEEG recordings cannot cover the entire brain. Dense-array EEG (dEEG) has both the ability to spatially and temporally localize activity from the entire cortical surface. In this study, we present localization results for primary motor (M1) and somatosensory (S1) functions using dEEG and compare these results against fMRI data from the same four subjects. To localize the dEEG data, individual head models are constructed. These head models accurately describe the geometry of the different head tissues and their conductivities, the location of potential source generators, and the EEG sensor positions on the scalp surface. Results show that with dEEG and accurate head models, M1 and S1 can be reliably localized in all subjects and that M1 and S1 can be separated. The results are consistent with the fMRI findings but they also provide millisecond-by-millisecond mapping of the functional time course to reveal when M1 and S1 are functionally activated

S53

Hypersomnia in Patients with Epilepsy and Comorbid Obstructive Sleep Apnea Hypopnea Syndrome

Douglas McKay Wallace, MD, Carolina Valdes, MD, Adriana Escandon-Sandino, MD, William K. Wohlgemuth, PhD

Background: Both obstructive sleep apnea-hypopnea syndrome (OSAHS) and anti-epileptic drugs (AED) are known to cause sleepiness, but the independent contribution of each in epilepsy patients is unknown. Our aim was to characterize predictors of hypersomnia in patients with epilepsy and OSAHS.

Methods/Design: Subjects were patients from the Miami VA Epilepsy clinic with comorbid OSAHS. Demographics, seizure characteristics/treatment, and polysomnography (PSG) variables were extracted from the medical record on PSG date. Hypersomnia was assessed with Epworth Sleepiness Scale (ESS) on the PSG night. In this cross-sectional analysis significant correlations between ESS and patient demographic/treatment factors were used to identify variables associated with hypersomnia. Linear regression analysis with these variables was performed to predict hypersomnia.

Results: Seventy-seven individuals (95% male, age 56±13 years) met study criteria. Mean apnea-hypopnea index and ESS were 34 ± 29 events/hr and 10.8 ± 5.3, respectively. Age, Charlson

comorbidity index, body mass index (BMI) and topiramate use were significantly correlated with hypersomnia. In a linear regression model with these 4 variables and AHI, only topiramate use (p=.01) and BMI (p=.005) predicted hypersomnia.

Conclusions: Topiramate and BMI, but not AHI, predict hypersomnia in epilepsy patients with untreated OSAHS. These factors may influence choice on AED in this patient population.

S54

Quantification and Localization of Ictal Onset EEG Sources on LTM Recordings

Pedro E. Coutin-Churchman, MD, PhD, Marc R. Nuwer, MD, PhD

Ictal video-electroencephalography (EEG) is commonly used to establish ictal onset-zone location, but surface topography does not necessarily correspond to actual source locations. EEG Source localization (ESL) of interictal spikes has been extensively studied and has been shown to have reasonable correspondence with epileptogenic foci. Ictal activity is more challenging but if it sources can be localized they should have a greater sensitivity and specificity. In this work we present the preliminary results of ESL during the first second of ictal onset activity on all seizures recorded during several days of LTM recording in 10 patients using two different strategies: frequency domain and time domain analysis. The results are compared to the standard consensus based on interictal ESL, interictal MEG, PET-MRI and clinical assessment during Epilepsy surgery workout.

S55

Hidden Value of EKG when Video-EEG Calls the Diagnosis into Question

Divya Singhal, MD, Heather McKee, MD, Meriem Bensalem, MD

Rationale: To report two cases of atypical spells presenting with bradycardia and normal EEG and highlight the relevance of concomitant electrocardiographic (EKG) recording when diagnosis remains an enigma despite continuous video EEG monitoring (vEEG). Background: vEEG is considered to be the gold standard of diagnosing epilepsy. However, when clinical evidence points to potential epilepsy, veeg alone may not be sufficient to preclude the diagnosis. Arrythmogenic seizures can present with normal background EEG when there is a deep frontal lobe focus and conversely, vEEG with concomitant EKG recording can aid in diagnosis of non-epileptic neurogenic arrhythmias.

Case reports: 41-year-old lady with intractable partial epilepsy underwent vEEG: stereotypic dystonic posturing episodes occurring out of sleep with concomitant bradycardia were documented on vEEG. Diagnosis of simple partial arrhythmogenic seizures was made and they responded to antiepileptic medication adjustment. 30-year-old gentleman presented with episodes of non-positional paroxysmal syncope refractory to antiepileptic medication. vEEG demonstrated bradycardia with no EEG change. MRI brain and Cervical-spine was pursued and revealed a Chiari I malformation. Posterior fossa decompression led to resolution of these cerebellar fits of Hughling Jackson.

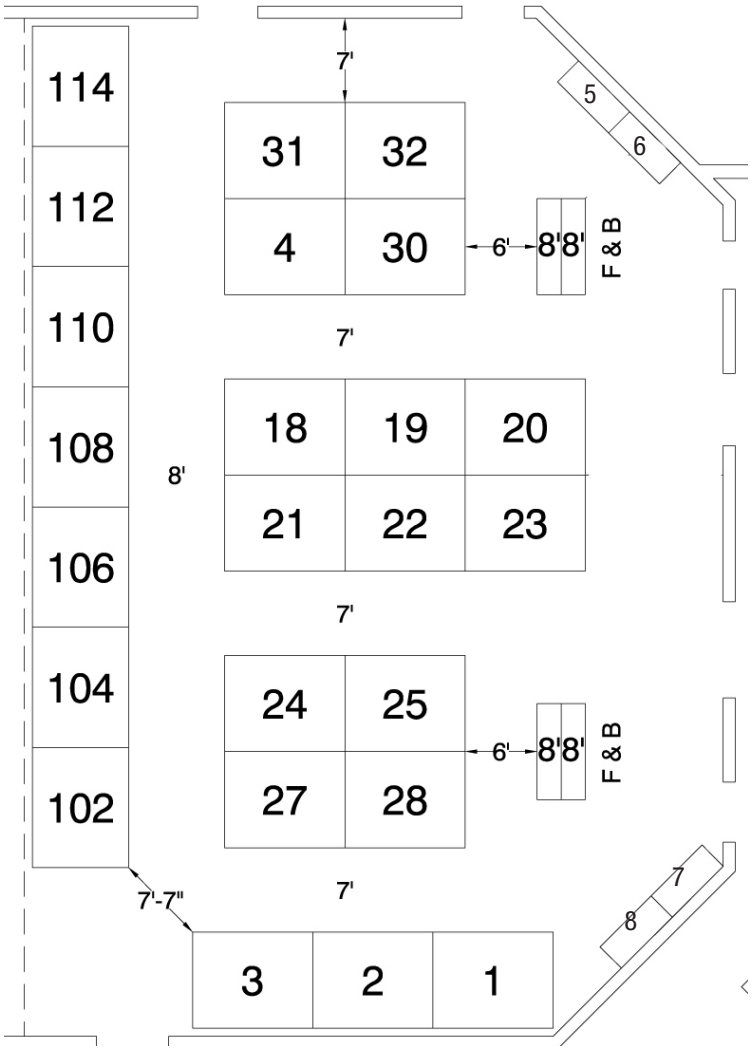
Conclusions: These two cases of arrhythmogenic spells emphasize the importance of astute observation of EKG changes in presence of normal EEG.

Exhibit Hall Information

EXHIBIT HOURS

Friday, February 8 - 10:00 AM - 4:00 PM
Friday, February 8 - 6:45 PM - 8:00 PM (Welcome Reception)
Saturday, February 9 - 10:00 AM - 1:30 PM

EXHIBIT HALL FLOORPLAN



Booth #	Company Name
30	Advanced Brain Monitoring Inc.
5	American Board of Clinical Neurophysiology, Inc. (ABCN)
5	The American board of Registration of EEG and EP Technologists (ABRET, Inc.)
6	American Clinical MEG Society (ACMEGS)
7	American Society of Neurophysiological Monitoring
8	ASET--The Neurodiagnostic Society
25	Blackrock Neuromed
20	Cadwell Laboratories, Inc.
21	Compumedics USA Inc.
19	Demo Medical Publishing
106	DigiTrace EEG Services
31	doctorPal
24	Electrical Geodesic, Inc. (EGI)
110	Grass Technologies
18	Ives EEG Solutions
108	Lippincott William & Wilkins
104	Moberg Research
4	Natus Neurology Inc.
3	NeuraLynx
112	Nihon Kohden America
2	Persyst
23	PMT Corporation
32	Professional Risk Management Services
1	Rhythmlink International
27	Ripple
22	Rochester Electro-Medical, Inc.
102	Terason
28	UCB

Exhibit Hall Information

EXHIBITORS

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 indicating sponsorship or approval of their products by the ACNS.

The following organizations will exhibit at the ACNS 2013 Annual Meeting in the Grand Ballroom at the Miami Marriott Biscayne Bay.

Booth #30
Advanced Brain Monitoring, Inc.
2237 Furaday Ave, Suite 100
Carlsbad CA 92008
Phone: 760.720.0099
www.advancedbrainmonitoring.com

Advanced Brain Monitoring offers three distinct B-Alert wireless EEG headsets which all deliver medical-grade, dependable signals in portable, easy-to-use and comfortable-for-hours designs. The new B-Alert X24 combines unprecedented power and flexibility with two interchangeable, pre-configured sensor strips that assure individually comfortable fit and accurate site placement across subjects. Please visit us to discuss your application ideas and opportunities for working together.

Booth #5
American Board of Clinical Neurophysiology, Inc. (ABCN)
2509 W. Iles Ave
Suite 102
Springfield, IL 62704
Phone: 217.726.7980
Fax: 217.726.7989
www.abcn.org

Booth #5
The American Board of Registration of EEG and EP Technologists (ABRET, Inc.)
2509 W. Iles Ave, Suite 102
Springfield, IL 62704
Phone: 217.726.7980
Fax: 217.726.7989
www.abret.org

Booth #6
American Clinical MEG Society (ACMEGS)
One Regency Drive P.O Box 30
Bloomfield, CT 6002
Phone: 860.243.3977
Fax: 860.286.0787
www.acmegs.org

Booth #7
American Society of Neurophysiological Monitoring
22 North Carroll Street, Suite 300
Madison, WI 53703
Phone: 608.310.5579
www.asnm.org

Booth #8
ASET--The Neurodiagnostic Society
P.O. BOX 36
East Boothbay, ME 04544
Phone: 207.350.4087
Fax: 877.207.2235
www.aset.org

Booth #25
Blackrock Neuromed
391 Chipeta Way, Suite G
Salt Lake City, UT 84108
Phone: 859.221.1043

Blackrock Neuromed's Cervello Elite EEG Monitoring System is a multi-channel data acquisition system specifically designed for real-time monitoring and recording of human brain and peripheral nerve electrical activity. The sophisticated system will simultaneously record, monitor, and analyze up to 256 channels of single unit action potentials through application of Blackrock's high-density Utah Array. The Cervello Elite is compatible with individual stiff-wire electrodes, microelectrode and microwire arrays, planar silicon probes, subdural ECoG grids, and epidural and scalp EEG electrodes, providing clinicians unsurpassed flexibility in patient-monitoring options. Flexible I/O options also facilitate interfacing to third-party equipment including behavior, stimulus, and video systems.

Booth #20
Cadwell Laboratories, Inc.
909 North Kellogg Street
Kennewick, WA 99336
Phone: 800.245.3001
Fax: 509.783.6503
www.cadwell.com

Cadwell has been focused on the development of useful and innovative devices for physiatrists, neurologists, neurophysiologists and technologists who want the very best devices to provide superior patient care since 1979. Based in Kennewick, WA, our products include the Cascade family for IONM, the Easy family for routine, ambulatory, LTM, and critical care EEG and PSG for in-lab and HST and the Sierra family for EMG, NCV and EP for both research and practice environments.

Exhibit Hall Information

Booth #21
Compumedics USA Inc.
6605 W. WT Harris Boulevard
Charlotte, NC 28269
Phone: 704.714.3200
Fax: 704.714.3298

Compumedics USA, Inc. is pleased to provide comprehensive solutions for Epilepsy evaluation and monitoring. Our NEUVO LTM and Grael EEG Systems with our CURRY-SACN 7 Neuroimaging Suite meet your requirements for routine and ambulatory clinical recordings as well as extended long-term EEG monitoring and Neuro-ICU monitoring. With capabilities unavailable from any other company to address the interest in ultra-high density and extended frequency-range recording for HFO, source localization and source imaging. SEE MORE! DO MORE!

Booth #19
Demos Medical Publishing
11 West 42nd Street, 15th Floor
New York, NY 10036
www.demosmedpub.com

Demos Medical is a publishing leader in clinical neurology and related disciplines. Visit us at the 2013 ACNS Annual Meeting to preview our list of premier print titles and exciting new digital products including Pediatric EEG Interactive DVD, Handbook of ICU EEG Monitoring, Neurology Video Textbook, Inherited Metabolic Epilepsies, Pediatric Neurocritical Care, and classics including EEG on DVD: Adult, Intraoperative Neurophysiology, Handbook of EEG Interpretation, Practical Approach to Neurophysiologic Intraoperative Monitoring, Nonconvulsive Status Epilepticus, and much more.

Booth #106
DigiTrace EEG Services
200 Corporate Place, Suite 5
Peabody, MA 01960
Phone: 978.536.6101
Fax: 978.536.6401

DigiTrace EEG products and services are used by dozens of comprehensive epilepsy centers throughout the U.S. In addition, there are over 40 SleepMed Service Centers around the country where physicians can refer their patients for ambulatory EEG testing. We are noted for unique capabilities including our lightweight head-mounted preamplifier that minimizes motion artifact, on-line spike and seizure detection, multiple day monitoring capabilities and high-resolution synchronized video. Ask about our newest offerings: DigiView 3.5 and DigiLink.

Booth #31
doctorPal
21444 Harper Avenue
St. Clair Shores, MI 48080
Phone: 248.894.7262

HIPAA-compliant, mobile billing app, developed by physicians for physicians. Create, Charge, Capture at point-of-care.

Booth #24
Electrical Geodesic, Inc. (EGI)
1600 Millrace Drive, Suite 200
Eugene, OR 97403
Phone: 541.687.7962
Fax: 541.687.7963
www.egi.com

EGI brings next-generation clinical EG systems, tools and workflows to hospitals and clinics worldwide. EGI's EEG Systems feature EGI's revolutionary Geodesic Sensor Net for unprecedented patient comfort and rapid application, removal, and cleanup. Your choice of systems with 32 channels for routine 10-20 EEG, or dense array systems with 64, 128, or 256 channels for source estimation and other applications requiring high spatial resolution. Visit EGI's booth to see this in action!

Booth #110
Grass Technologies
600 East Greenwich Avenue
West Warwick, RI 02893
Phone: 401.828.4000
Fax: 401.822.2430
www.grasstechnologies.com

Grass Technologies offers a wide range of instrumentation for PSG, EEG, LTM, Neuromonitoring – from lab-based to ambulatory recorders – at affordable prices. Systems feature the world-renowned accuracy, dependability and performance of Grass amplifiers, and powerful software. We also offer the new S12X Cortical Stimulator. A full line of electrodes, transducers, etc. is also available – visit our Online Store.

Booth #18
Ives EEG Solutions
25 Stoney Ave, Suite 118
Newburyport, MA 01950
Phone: 978.358.8006
Fax: 978.358.7825
www.iveseegsolutions.com

Never have to remove EEG Electrodes again for imaging purposes. The Ives Imaging Friendly™ Electrode System provides a comprehensive selection of three separate imaging friendly EEG electrode types to record the patient's EEG in the EMU or cEEG in the ICU.

Exhibit Hall Information

Booth #108
Lippincott William & Wilkins
16522 Hunters Green Parkway
Hagerstown, MD 21740
Phone: 410.528.4444
Fax: 410.510.1666
journals.lww.com

Lippincott William & Wilkins Woldters Kluwek Health is a global provider of information, business intelligence and point-of-care solutions for the healthcare industry. Major brands include Lippincott Williams & Wilkins, Lippincott's Nursing Solutions, medical books, journals, and electronic media. Please visit our booth to browse our comprehensive product selection.

Booth #104
Moberg Research
224 S. Maple Street
Ambler, PA 19002
Phone: 215.283.0860
Fax: 215.283.0859
www.moberg.com

The Component Neuromonitoring System (CNS Monitor) is the only EEG system specifically designed for the needs of the ICU. It is a full-function video/EEG monitor with remote review of EEG and other physiology using Persyst software. Only the CNS Monitor can collect data from over 25 other monitoring and therapeutic devices and accurately synchronize it in real time with quantitative EEG trends. The future of integrated neurophysiological monitoring is available today with the CNS Monitor.

Booth #4
Natus Neurology, Inc.
1501 Industrial Road
San Carlos, CA 94070
Phone: 800.255.3901
Fax: 650.802.0401
www.natus.com

Natus is a global market leader providing Nicolet and XLTEK diagnostic and monitoring medical equipment for neurological and vascular markets, supplies and accessories, and integrated connectivity solutions.

Booth #3
NeuraLynx, Inc.
105 Commercial Drive
Bozeman, MT 59715
Phone: 915.545.3191
Fax: 406.585.4542

Neuralynx, Inc. is an internationally recognized provider of electrophysiology data recording systems and solutions for neuroscience research, as well as for practical human medical data recording. The Neuralynx ATLAS is the premier clinical single-unit system available on the market today. Neuralynx, Inc. specializes in the development of cutting edge electrophysiology data recording systems, optogenetics systems, and experimental accessories while providing quality, long term customer guidance and support.

Booth #112
Nihon Kohden America
90 Icon Street
Foothill Ranch, CA 92610
Phone: 949.580.1550
Fax: 949.580.1555
www.nkusa.com

Nihon Kohden's Neurology product and portfolio includes instrumentation for Epilepsy Monitoring, Electroencephalography, EEG & PSG Ambulatory Recording, Polysomnography, Wireless EEG & PSG, Home Sleep Testing/PSG, Electromyography, Evoked Potentials, Intra-operative and cEEG ICU monitoring. Nihon Kohden's instrumentation offers the flexibility and expandability needed to meet the changing demands of today's neurodiagnostic field.

Booth #2
Persyst
12625 High Bluff Drive, Suite 213
San Diego, CA 92130
Phone: 858.461.4542
Fax: 858.461.4531
www.persyst.com

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

Exhibit Hall Information

Booth #23
PMT Corporation
1500 Park Road
Chanhassen, MN 55317
Phone: 952.470.0866
Fax: 952.470.0865

For over three decades PMT® has been designing and producing quality electrodes and microwires for epilepsy procedures and research. Our unmatched signal clarity, product quality, and dependable customer service makes us an industry leader. With a large sales force spread throughout the U.S. and distributors around the world, we can be on-site to work with you to define products to match your specific requirements.

Booth #32
Professional Risk Management Services
1401 Wilson Blvd.
Arlington, VA 22209
Phone: 703.907.3888
www.tnpinsurance.com

The Neurologists' Program (TNP) is a comprehensive medical professional liability insurance program designed specifically for neurologists. Benefits include: extensive risk management resources such as the Risk Management Consultation Service helpline, access to experienced defense attorneys and discounts including early career, part-time, loss-free and more! Visit www.tnpinsurance.com for more information.

Booth #1
Rhythmlink International, LLC
1256 First Street South Extension
Columbia, SC 29209
Phone: 803.252.1222
Fax: 803.525.1111
www.rhythmlink.com

Rhythmlink International, LLC designs manufacture and distributes neurodiagnostic, intraoperative monitoring, critical care and polysomnography devices and provides custom packaging, private labeling, custom products and contract manufacturing solutions to its customers. Rhythmlink is recognized as a leader within its field at providing the important physical connection between patients ad the diagnostic equipment. Based in Columbia, SC, Rhythmlink was established in 2002 and is owned and operated by CEO Shawn Regan and COO Michael O'Leary. Rhythmlink's advancements and improvements in the areas of technology, business development and corporate branding have been recognized both on a national and international scale.

Booth #27
Ripple
2015 South 1100 East
Salt Lake City, UT 84106
Phone: 801.413.0139
Fax: 801.413.2874
www.rppl.com

Ripple provides neurophysiology data acquisition systems for neuroscience and clinical research. Our systems are compact, portable, and heavily optimized for real-time, closed-loop applications with hundreds of channels of EMG, EEG, ECoG, and microelectrode data. Our software is cross platform, and can be run on Windows, Mac OS, and Linux.

Booth #22
Rochester Electro-Medical, Inc.
4212 Cypress Gulch Drive
Lutz, FL 33559
Phone: 800.328.5544
Fax: 800.545.0845
www.rochestermed.com

For over 50 years, Rochester Electro-Medical has been your best course for affordable, high quality Neurodiagnotic Products and Customer Service with a personal touch. Featured products include NEW customizable lines of Singe, Paired and RIBBON Subdermal Needle Electrodes that offer unbeatable value and performance. Contact us for information on our full range of Disposable Products, Electrodes, Needles, Sensors and Accessories.

Booth #102
Terason
77 Terrace Hall
Burlington, MA 01803
Phone: 781.270.4143
Fax: 781.270.4145

Booth #28
UCB
7 Flagg Street
Cambridge, MA 02138
Phone: 617.803.9195
Fax: 770.970.8913

Notes

Annual Course Outline

Tuesday, February 5, 2013		
8:00 AM – 5:00 PM	Registration Open	Bayview Ballroom Foyer, Level 2
9:00 AM – 5:00 PM	Intraoperative Monitoring: Part I	Bayview Ballroom, Level 2
Wednesday, February 6, 2013		
6:30 AM – 5:00 PM	Registration Open	Bayview Ballroom Foyer, Level 2
7:00 – 8:30 AM	EP Reading Session	Bayview Ballroom Foyer, Level 2
	Neonatal EEG	Watson Island, Level 2
9:00 AM – 5:00 PM	EEG Course: Intracranial EEG	Watson Island, Level 2
	Intraoperative Monitoring Part II	Bayview Ballroom, Level 2
5:00 – 7:00 PM	EEG and MEG Source Modeling	Bayview Ballroom, Level 2
Thursday, February 7, 2013		
6:30 AM – 5:00 PM	Registration Open	Grand Ballroom Foyer, Level 3
7:00 – 8:30 AM	EMG and EEG Technology	Bayview Ballroom, Level 2
	Business in Clinical Neurophysiology	Grand Ballroom Salon F, Level 3
9:00 AM – 5:00 PM	Intensive Care Unit, Electroencephalography	Grand Ballroom Salon F, Level 3
9:00 AM – 12:00 PM	Electromyography	Hibiscus Island, Level 3
	Video-EEG	Bayview Ballroom, Level 2
1:00 – 2:30 PM	Applied Autonomic Neurophysiology	Bayview Ballroom, Level 2
2:30 – 5:00 PM	Electrophysiological and Pathological Findings in Neuromuscular Disease: A Case-Based Approach	Bayview Ballroom, Level 2
5:00 – 7:00 PM	Billing and Coding in Clinical Neurophysiology	Bayview Ballroom, Level 2
	Seizure Detection	Grand Ballroom Salon F, Level 3

Annual Meeting Outline *for Course outline see inside back cover*

Friday, February 8, 2013		
7:00 AM – 5:00 PM	Registration Open	Grand Ballroom Foyer, Level 3
7:00 – 8:00 AM	Continental Breakfast	Grand Ballroom Salons A – E, Level 3
8:00 – 10:00 AM	Opening Session	Grand Ballroom Salons F – K, Level 3
10:00 AM – 10:30 AM	Coffee Break	Grand Ballroom Salons A – E, Level 3
10:30 AM – 12:30 PM	Symposium: Memory & Language	Grand Ballroom Salons F – K, Level 3
	Symposium: Neurophysiologic Evaluation	Watson Island, Level 2
	Symposium: Intraoperative Neurophysiologic Monitoring	Bayview Ballroom, Level 2
12:30 – 1:30 PM	Lunch	Grand Ballroom Salons A – E, Level 3
	Professional Development Mentoring Program	Biscayne Island, Level 3
1:30 – 3:30 PM	Symposium: Challenges of Nonlesional Focal Epilepsy	Grand Ballroom Salons F – K, Level 3
	Symposium: Controversial EMG Topics	Watson Island, Level 2
	Symposium: Challenges in Intraoperative Neurophysiologic Mapping	Bayview Ballroom, Level 2
3:30 – 4:00 PM	Coffee Break	Grand Ballroom Salons A – E, Level 3
4:30 – 5:30 PM	Symposium: Neuromonitoring of the Pediatric Patient	Bayview Ballroom, Level 2
	Symposium: Brain Stiumlation	Grand Ballroom Salons F – K, Level 3
5:30 – 6:45 PM	Neurophys Bowl	Grand Ballroom Salons F – K, Level 3
6:45 – 8:00 PM	Welcome Reception – Visit Exhibits	Grand Ballroom Salons A – E, Level 3
Saturday, February 9, 2013		
7:00 AM – 5:00 PM	Registration Open	Grand Ballroom Foyer, Level 3
7:00 – 8:00 AM	Continental Breakfast	Grand Ballroom Salons A – E, Level 3
8:00 – 10:00 AM	Opening Session	Grand Ballroom Salon F, Level 3
10:00 – 10:30 AM	Coffee Break	Grand Ballroom Salons A – E, Level 3
10:30 AM – 12:30 PM	Symposium: High Density EEG	Grand Ballroom Salon F, Level 3
	Symposium: Advances in the Neurophysiological Assessment of Concussion	Bayview Ballroom, Level 2
	Workshop: Neuromuscular Ultrasound	Grand Ballroom Salons A – K, Level 3
12:30 – 1:30 PM	Lunch	Grand Ballroom Salons A – E, Level 3
12:15 – 1:30 PM	Fellowship Director's Lunch	Watson Island, Level 2
1:30 – 3:30 PM	Symposium: New Directions for Quantitative EEG	Grand Ballroom Salon F, Level 3
	SIG: Sleep & Epilepsy	Bayview Ballroom, Level 2
	SIG: Epilepsy Networks	Grand Ballroom Salons G – K, Level 3
3:30 – 4:00 PM	Coffee Break	Grand Ballroom Foyer, Level 3
4:00 – 5:30 PM	SIG: Intraoperative Monitoring	Bayview Ballroom, Level 2
	Workshop: EEG-Video: Expert Consensus	Grand Ballroom Salon F, Level 3
	SIG: The Coming of Age of Magnetoencephalography	Grand Ballroom Salons A – K, Level 3
5:30 – 6:00 PM	Annual Business Meeting	Grand Ballroom Salon F, Level 3
Sunday, February 10, 2013		
7:00 AM – 12:00 PM	Registration Open	Bayview Ballroom Foyer, Level 2
7:00 – 8:00 AM	Continental Breakfast	Bayview Ballroom, Level 2
8:00 – 10:00 AM	SIG: Intensive Care Unit, Electroencephalography	Bayview Ballroom, Level 2
	Workshop: Advanced Nerve Conduction	Dodge Island, Level 3
	SIG: Models of Professional Care in IOM	Watson Island, Level 2
10:00 – 10:15 AM	Coffee Break	Bayview Ballroom, Level 2
10:15 AM – 12:15 PM	Symposium: Continuous EEG Monitoring	Dodge Island, Level 3
	Symposium: Encephalopathy; Electrophysiologic, Clinical & Imaging Correlations	Bayview Ballroom, Level 2
	Symposium: The Current State of Safety in the EMU	Watson Island, Level 2

American Clinical Neurophysiology Society
555 E. Wells St. Suite 1100 | Milwaukee, WI 53202 | Phone: 414-918-9803 | Fax: 414-276-3349 | email: info@acns.org