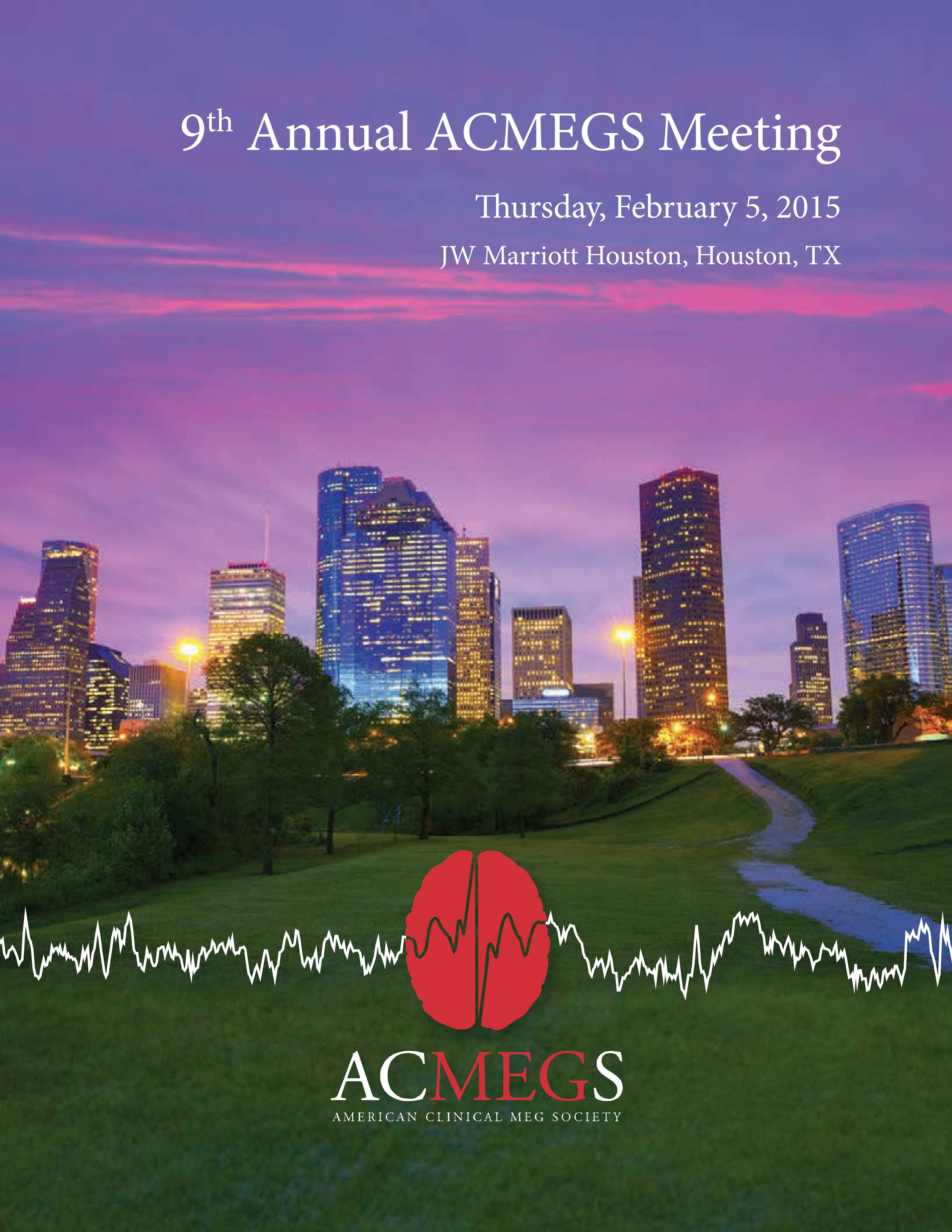
9th Annual ACMEGS Meeting

Thursday, February 5, 2015

JW Marriott Houston, Houston, TX



ACMEGS

AMERICAN CLINICAL MEG SOCIETY

Welcome to Houston!

On behalf of the Program and Course Committees and the ACMEGS Board, I hope that you enjoy your visit to Houston, its climate, food and people.

This is our 9th Annual Conference of the ACMEGS and the fifth joint meeting with the American Clinical Neurophysiology Society (ACNS). The goal of this format is to save ACMEGS members who are also associated with ACNS one trip to a conference, as well as to spark some interest among the members of ACNS who are not so familiar with MEG technology and its clinical applications. After all, MEG is a neurophysiological method, and we have been enjoying a productive synergy with our sister society (ACNS).

This year we changed the format of the meeting slightly by moving the Annual Business Meeting to the late afternoon to encourage interested ACNS members to join us in the morning and afternoon hours for the clinical and scientific presentations.

The past year was another successful year for our Society, during which we improved our administrative issues with the Commonwealth of Massachusetts, reached out to other related professional organizations (i.e. ACNS, AES, ASET, ABRET, etc.), sustained our Center membership and continued to work on enhancing the value of the Society to its members and the value of the MEG Centers to their institutions. To this extent, we also engaged in a conversation with the Research Triangle Institute that performs annual US News & World Report Hospital rankings.

We will have a very interesting scientific program this year with nine presentations delivered by experts in the field of research and clinical MEG, and we are particularly glad to welcome among them Dr. Ernst Rodin from Salt Lake City, Utah.

Our conference aims to provide an informal and friendly atmosphere for discussing and exchanging recent clinically relevant studies that might lead to new clinical MEG indications. In addition, we are dedicated to enabling you, our members, to promote the appropriate use of Magnetoencephalography. We wish to empower you to work closely with national and local health insurance carriers and governmental regulatory bodies to ensure accurate and successful reimbursement.

Welcome to Houston and I hope you will enjoy the conference and our traditional Society dinner at the end of a day filled with lectures and discussions.

Sincerely,

Anto Bagić, MD, PhD
President, American Clinical Magnetoencephalography Society

Michael Funke, MD, PhD
Chair, Meeting Organizing Committee

ACMEGS
AMERICAN CLINICAL MEG SOCIETY

2015 ACMEGS Annual Conference
Thursday, February 5, 2015
JW Marriott Houston • Houston, Texas

8:00am Arrival / Breakfast Reception

9:00am ACMEGS Presidential Address 2014
Welcome and Introduction - Anto Bagic, Pittsburgh

9:15am Current Issues and Controversies in Pediatric MEG — Chair: Gretchen Von Allmen, Houston
- *Anesthetic Management on Quality in Pediatric MEG Patients* - Douglas Rose, Cincinnati
- *Pediatric MEG: The Effect of Head Positioning on SEF* - William Gaetz, Philadelphia
- *Passive Language Mapping with MEG in Pediatric Patients with Epilepsy* - Dave Clark, Austin
- *MEG Localization of Broca's Area Using Verb Generation Tasks* - Elizabeth Pang, Toronto

11:30am Annual ACMEGS Photo Shoot (location TBD)

11:45am Lunch

1:00pm New and Novel Applications of MEG: Results from the Field — Chair: Richard Burgess, Cleveland
- *Complexity Analysis of MEG in Traumatic Brain Injury Patients* - Richard Bucholz, St. Louis
- *Human brain development research with MEG*- Joshio Okada, Boston
- *Neural Synchrony Examined with MEG During Eye Gaze Processing in Autism Spectrum Disorder* - Renee Lajiness-O'Neill, Detroit
- *Abnormal MEG Coherence Imaging in Panic Disorder* - Nash Boutros, Kansas City

2:30pm Coffee Break

3:00pm Update on Educational Initiatives — Chair: Anto Bagic, Pittsburgh
- *Update on MEG Fellowship Curriculum* - Richard Burgess, Cleveland
- *Our Experiences: A Report from MEG Fellows*
  - *Andrew Zillgitt* (Henry Ford Hospital; Detroit, MI)
  - *Michael Watkins* (University of Texas-Houston; Houston, TX)
- *Update on MEG/EEG-Technologist Activities* - Janice Walbert, ABRET & Brian Markley, ASET

3:45pm ACMEGS Lecture 2014
The MEG Slow Dimensions: Sifting Facts from Artifact – Ernst Rodin, Salt Lake City

4:30pm Meeting Adjourn

4:40pm Business Meeting — Chair: Anto Bagic, Pittsburgh
- *President's Report* - Anto Bagic, Pittsburgh
- *Financial Report* - Susan Bowyer, Detroit
- *Public Relations Committee* - Susan Bowyer, Detroit
- *Elections and New Business*

6:00 pm ACMEGS Dinner
Location: Mockingbird Bistro (1985 Welch Street, Houston, TX 77019, ☎ 713-533-0200)

ACMEGS
AMERICAN CLINICAL MEG SOCIETY

Presidential Address
Anto Bagic, Pittsburgh, PA

Presidential Address 2015

Anto Bagić, MD, PhD
(Pittsburgh, PA)

February 5, 2015; Houston, TX

1/37 *Presidential Address* Bagić A, 2015

ACMEGS Year In Retrospect (1/5)

- Society in good standing with Commonwealth of MA.
- Center Members (17): (34 delegated members).
- Individual Members: 13 (7 full + 6 associate).
- Managed by EDI.

2/37 *Presidential Address* Bagić A, 2015

Society In Good Standing With The Commonwealth Of MA

- Administrative Issues
  - All resolved
  - Bylaws updated
  - Collective efforts:
    - ACMEGS Board
    - EDI, Inc. (Milwaukee, WI).
    - Attorney in Boston

3/37 *Presidential Address* Bagić A, 2015

2014 ACMEGS Honor Roll (N=17)

1	Alexian Brothers Neuroscience Institute	Elk Grove Village	IL
2	Atlantic Neuroscience Institute	Summit	NJ
3	Children's Hospital of Philadelphia	Philadelphia	PA
4	Cincinnati Children's Hospital	Cincinnati	OH
5	Cleveland Clinic Foundation	Cleveland	OH
6	Dell Children's Medical Center	Austin	TX
7	Cook Children's Health System	Fort Worth	TX
8	Froedtert Hospital	Milwaukee	WI
9	Henry Ford Hospital	Detroit	MI
10	Meadowlands Hospital	Secaucus	NJ
11	Minnesota Epilepsy Group	St. Paul	MN
12	Nebraska Medical Center	Omaha	NE
13	University of California San Francisco	San Francisco	CA
14	University of Colorado	Denver,	CO
15	University of Pittsburgh Medical Center	Pittsburgh	PA
16	University of Tennessee	Memphis	TN
17	UT Memorial Herman	Houston	TX

(34 delegated "designated" members)

4/37 *Presidential Address* Bagić A, 2015

Individual ACMEGS Members

5/37 *Presidential Address* Bagić A, 2015

The ACMEGS Board Of Directors

Thank You!

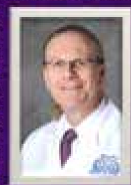
6/37 *Presidential Address* Bagić A, 2015

The ACMEGS Wise Men

ACMEGS Consultant

Michael Longacre
National Account Director
Special Projects - Payer Markets
Assurex Healthcare Inc.
Mason, OH

ACMEGS Board Advisor



Gregory L. Barkley, M.D.
Vice Chair for Clinical Affairs
Comprehensive Epilepsy Program
Henry Ford Hospital
Detroit, MI

Thank You!

7/37 *Presidential Address* Bagić A, 2015

Our First Year And Half With EDI, Inc.

(Since 07/01/2013)

edi

What You Get from an AMC

realize your vision

Ms. Kay Whalen, MBA, CAE
President of EDI, Inc.

ACMEGS Executive Director:
Ms. Megan Hille

ACMEGS
555 East Wells Street, Suite 1100
Milwaukee, WI 53202
Phone: 414-918-9804

ACMEGS

8/37 *Presidential Address* Bagić A, 2015

ACMEGS Year In Retrospect (2/5)

- Continued productive relationship with the ACNS.
- Continued productive relationship with the AAN.
- Informal interactions with Elekta representatives (July 7, 2014: Miikka Putaala, Ted Godfrey).
- *Memorandum of Understanding* – Unrestricted Educational Grant

9/37 *Presidential Address* Bagić A, 2015

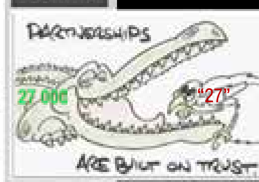
MEG Model Policy

Adopted May 9, 2009

Magnetoencephalography (MEG) Model Policy

AMERICAN ACADEMY OF NEUROLOGY.

SCORE



This 2009 document played a vital role in opening up a door for commercial MEG reimbursement in the USA…

AMERICAN ACADEMY OF NEUROLOGY.

August 22, 2014

Ms. Marilyn Tavenner
Administrator
Centers for Medicare & Medicaid Services
U.S. Department of Health and Human Services
Hubert H. Humphrey Building, Room 445-G
200 Independence Avenue, SW
Washington, DC 20201
Submitted electronically: http://regulations.gov

RE: Hospital Outpatient Prospective Payment and Ambulatory Surgical Center Payment Systems and Quality Reporting Programs; Physician-Owned Hospitals: Data Sources for Expansion Exception; Physician Certification of Inpatient Hospital Services; Medicare Advantage Organizations and Part D Sponsors: Appeals Process for Overpayments Associated with Submitted Data [CMS-1613-P]

Dear Administrator Tavenner:

The American Academy of Neurology (AAN) appreciates the opportunity to provide written comments on the Medicare and Medicaid Programs: Hospital Outpatient Prospective Payment (OPPS) and Ambulatory Surgical Center Payment Systems and Quality Reporting Programs; Physician-Owned Hospitals: Data Sources for Expansion Exception; Physician Certification of Inpatient Hospital Services; Medicare Advantage Organizations and Part D Sponsors: Appeals Process for Overpayments Associated with Submitted Data [CMS-1613-P] published in the *Federal Register* as a proposed rule on July 11, 2014.

The American Academy of Neurology (AAN) is the premier national

… more than 27,000 neurologists and clinical neuroscience professionals…

Alzheimer's disease, stroke, epilepsy, Parkinson's disease, migraine, multiple sclerosis, and brain injury.

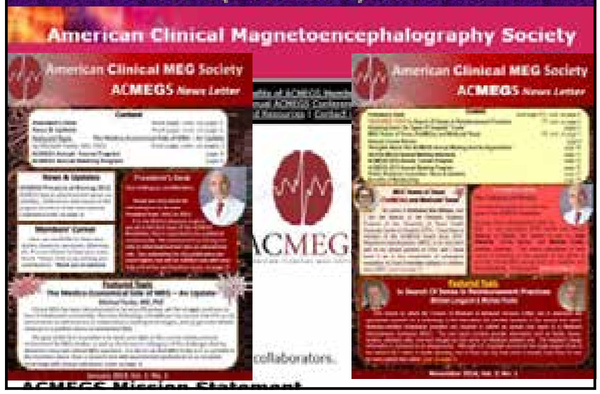
… more appropriate Ambulatory Payment Classification (APC) for Magnetoencephalograpy (MEG) codes 95965-95967 in 2015.…

ACMEGS Year In Retrospect (3/5)

- Continued efforts on increasing the value to our (center) members:
  - Web-based resources (policies, CPGs, cases, jobs, etc.).
  - Addressing individual center member concerns.
  - Assistance to the new sites.
  - Strategic decision not to get on the CMS radar.
  - Inpatient MEG…
  - Newsletter (Check it out and contribute!).

13/37 *Presidential Address* Bagić A, 2015

ACMEGS (Web-based) Resources



ACMEGS Year In Retrospect (4/5)

- Sustained efforts on increasing the value of the MEG centers to their institutions:
  - Improving billing practices.
  - Monitoring insurance situation.
  - Engaging with the RTI (*US News & World Report*).
  - Promoting clinical MEG and ACMEGS at ACNS, AES (ACMEGS boot and Dr. M. Funke had a public presentation), ASET, AAN, and other relevant conferences.

15/37 *Presidential Address* Bagić A, 2015

Engaging with the RTI

16/37 Bagić A, 2015

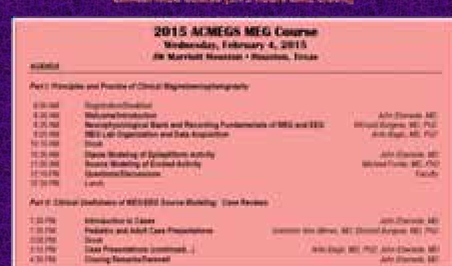
ACMEGS Year In Retrospect (5/5)

- ACMEGS educational efforts and activities:
  - Annual Course (3rd was attended by 28 attendees!)
  - Are we ready for a fellowship concept?
  - Upcoming update on the training opportunities later today (3 MEG Fellows and Dr. Burgess).
  - MEG technologists inquiries: Quo Vadis ASET MEG Section?
  - General web-based resources.
  - Individual help.

17/37 *Presidential Address* Bagić A, 2015

The 4th Annual MEG Course

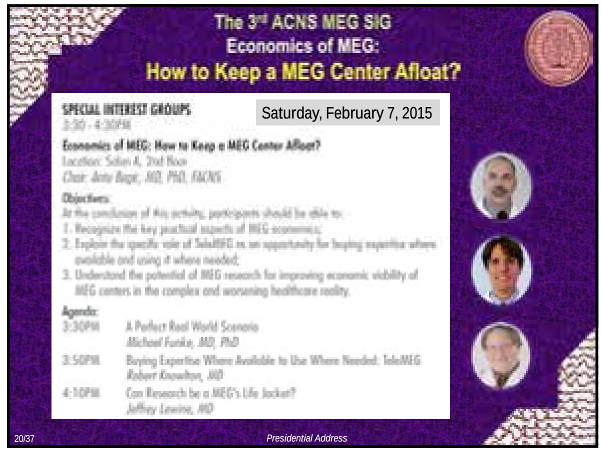
Clinical MEG Course (5.75 hours CME credit)



2015 Faculty

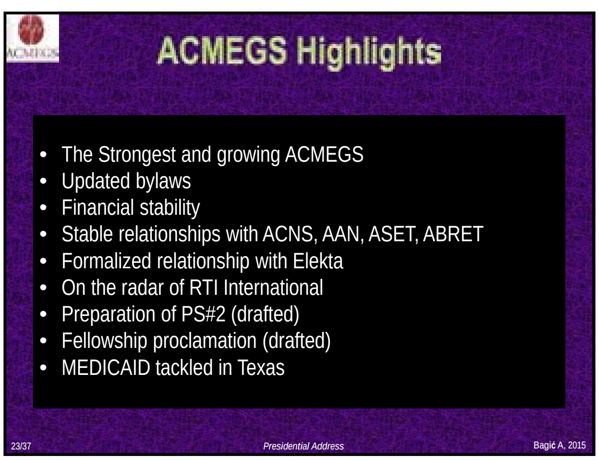
18/37 *Presidential Address* Bagić A, 2015


Crashing the Cultures of the Sole MEG or EEG Source Modeling: Inseperable, Not Only Complementary (SIG)
10:15AM – 12:15PM
Chair: Anto Bagic, MD, PhD
Presentations and Faculty
Combined MEG and EEG Source Modeling is Clearly Advantageous
John Ebersole, MD
MSI is Simply Superior and ESI Gives Me Nothing More
Richard Burgess, MD
Lost Clinician: Experts Disagree, What do I do?
Anto Bagic, MD, PhD
February 9, 2014 (10:15 – 12:15)
Presidential Address


The 3rd ACNS MEG SIG
Economics of MEG:
How to Keep a MEG Center Afloat?
SPECIAL INTEREST GROUPS
Saturday, February 7, 2015
3:30 - 4:30PM
Economics of MEG: How to Keep a MEG Center Afloat?
Chair: Anto Bagic, MD, PhD, FACNS
Objectives:
Agenda:
3:30PM
A Perfect Real World Scenario
Michael Funke, MD, PhD
3:50PM
Robert Knowlton, MD
4:10PM
Can Research be a MEG's Life Jacket?
Jeffrey Lewine, MD
20/37
Presidential Address


MEG/MSI
Special Interest Group
December 7, 2014: 6:00 – 7:30 PM
Convention Center – Room 620, Level 6
Source Localization Results:
Blind Faith, Black Art, or Scientific Method?
Dr. Funke
Dr. Burgess
Dr. Stufflebeam
Dr. Ebersole
Coordinator: Anto Bagić, MD, PhD
University of Pittsburgh, PA
2014 AES Annual Meeting; Seattle, WA
American Epilepsy Society
2014


BIOMAG 2014
19th International Conference
on Biomagnetism
August 24 - 28, 2014
Halifax, Canada
Current Strengths and Emerging Clinical Applications
ISACM Symposium!


ACMEGS
ACMEGS Highlights
The Strongest and growing ACMEGS
Updated bylaws
Financial stability
Stable relationships with ACNS, AAN, ASET, ABRET
Formalized relationship with Elekta
On the radar of RTI International
Preparation of PS#2 (drafted)
Fellowship proclamation (drafted)
MEDICAID tackled in Texas
23/37
Presidential Address
Bagić A, 2015


ACMEGS
What Is Ahead?
Position Statement #2 regarding MEG in PSM (drafted).
Public Announcement of Fellowship Concept (drafted).
Sustain the current efforts on all fronts.
– Escalate efforts on increasing (center) membership.
– Cultivate the relationship with the ACNS, AAN, AES
– Foster the relationship with the ASET, ABRET, ISACM.
Increase our presence at appropriate clinical conferences.
Establish the ACMEGS Resource Center
Facilitate collaborative efforts on clinical research leading to new potential indications for MEG.
24/37
Presidential Address
Bagić A, 2015

Tune Your Travel Plans

- ACNS 2015 Annual Meeting (February 3 - 8, 2015; Atlanta, GA).
- ISACM 2015 (June 23 - 26, 2015; Helsinki, Finland).
- AES 2015 Annual Meeting (December 4 - 8, 2015; Philadelphia, PA).
- ACMEGS 2016 Annual Meeting (March 14, 2016; San Diego, CA).
- ACNS 2016 Annual Meeting (March 13 - 17, 2016; San Diego, CA).
- Biomag 2016 (October 1 - 6, 2016; Seoul, Korea).

25/37 *Presidential Address* Bagić A, 2015

Acknowledgments

- ACMEGS Members (Centers and individuals)
- **ACMEGS Board Members**
- Michael Longacre & Gregory R. Barkley
- Elekta Neuromag Oy
  - Unrestricted Educational Arant
- ACNS
  - Synchronized meetings, CME approval, Sharing posters
- AAN
  - Sustained support in CMS matters
- ASET/ABRET
  - Educational programs for technologists
- EDI, iNc. (Megan Hille, *Executive Director*)

26/37 *Presidential Address* Bagić A, 2015

Let's,

beat

bystander effect (group apathy)

and

participate!

Thank you!

bagica@upmc.edu

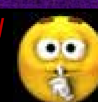
Team work(s)

http://demots.co.uk/web/gfx/a-teamwork.jpg

27/37 *Presidential Address*

Caution

- *Please do not share your institutional reimbursement and billing rates.*



- *Sharing such information could be considered collusion and may have legal ramifications for you and the society.*

28/37 *Presidential Address* Bagić A, 2015

Have a Productive and Joyful Meeting
and
Continue to Promote Clinical MEG, CPGs
and
ACMEGS!

29/37 *Presidential Address* Bagić A, 2015

ACMEGS

AMERICAN CLINICAL MEG SOCIETY

Anesthetic Management on Quality in Pediatric MEG Patients

Douglas Rose, MD

Anesthetic Management on Quality in Pediatric MEG Patients

Douglas F. Rose, M.D.
Cincinnati Children's Hospital Medical Center
February 5, 2015

Need for sedation or anesthesia for MEG: Epilepsy studies in Pediatrics

- High quality recordings with MEG require movement less than a few millimeters and preferably no interfering muscle artifact.
- Both requirements are difficult to achieve while the patient is awake, but can be achieved during sleep
- For adults, cooperative adolescents, and nl children older than 7 years, this can typically be achieved through sleep deprivation the night before.
- However, pediatric patients with intractable epilepsy often function at a cognitive and emotional level several years below their stated age
- Despite sleep deprivation alone, these patients may not be able to fall asleep because of
  - Excessive anxiety
  - Oppositional/combative behavior
- Thus, some kind of additional sedation is often required.

Sedation/Anesthesia for Pediatrics Patients: Strategies

- **Rescheduling of patient's already prescribed bedtime medications for sleep**
  - Melatonin
  - Clonidine
  - Benadryl
  - Held night before to permit sleep deprivation, given instead just before MEG recording begins
- **Conscious sedation – Usually also still requires sleep deprivation to be effective.**
  - Benzodiazepines – suppress epileptiform discharges, beta (+) bandwidth artifact
  - Chloral hydrate – Institution may limit the mg/kg and/or total mg dose allowed, may interact with some AEDs to cause fast activity artifact
  - Clonidine – potential lowering of BP
  - Melatonin – may have limited efficacy in depth and/or duration of sleep
  - Requires presence of nurse assigned to procedure just to monitor vital signs (PulseOx, BP, HR/EKG)
- **General anesthesia (GA)**
  - Inhalation gases
    - Nitrous oxide – usually limited to initial induction of GA
    - Isoflurane, Sevoflurane and others
  - Total intravenous anesthesia (TIVA)
    - Propofol – frontal alpha bandwidth artifact
    - Dexmedetomidine – newer TIVA, low beta bandwidth artifact appears similar to sleep spindles
  - Requires presence of anesthesiologist or nurse anesthetist

Levels of Sedation

Table 1 Levels of sedation

Factors	Minimal sedation	Moderate sedation/analgesia	Deep sedation	General anesthesia
Responsiveness	Normal response to verbal stimulation	Purposeful response to verbal or tactile stimulation	Purposeful response to repeated or painful stimulation	Unarousable even with painful stimulus
Airway	Unaffected	No intervention required	Intervention may be required	Intervention often required
Spontaneous ventilation	Unaffected	Adequate	May be inadequate	Frequently inadequate
Cardiovascular function	Unaffected	Usually maintained	Usually maintained	May be impaired

- Term equivalents for this presentation
  - Minimal sedation – "sleep deprivation only prior to test"
  - Moderate sedation – "conscious sedation"
  - Deep sedation – may be closer "general anesthesia" of dexmedetomidine
  - General anesthesia – may fit some levels of propofol anesthesia

General Anesthesia

- Agents have to be chosen carefully because of epileptogenic activation or suppression
  - Inhalation anesthetics
    - Sevoflurane precipitate seizure-like activity, particularly in
    - Enflurane exhibits periods of suppression with paroxysmal epileptiform discharges in animal models and multiple reports of seizure activity in humans after enflurane anaesthesia
    - Isoflurane and desflurane have anticonvulsant properties
  - Intravenous anesthetics
    - Etomidate increases epileptiform discharges and may induce seizures
    - **Propofol**
    - **Dexmedetomidine**

Review of Literature

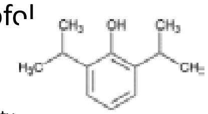
PUBMED review # of articles searched: 20445 MEG or EEG:

- MEG and anesthesia (GA) 39
- MEG and sedation 33
- EEG and GA 5604
- EEG and sedation 1245

- **MEG GA and pediatrics 7: P = 5, D = 1**
- **MEG sedation and pediatrics 5: C = 1**
- EEG GA and pediatrics 985: P = 271, D = 23, C = 8
- EEG sedation and pediatrics 314: C= 50

P=propofol, D=dexmedetomidine, C=chloral hydrate

Propofol



- Mechanism of action:
  - Potentiation of $GABA_A$ receptor activity
  - Sodium channel blocker
  - Endocannabinoid system?
- TIVA
  - Rapid onset anesthesia/rapid recovery from anesthesia
  - Adverse effects:
    - Bradycardia at induction
    - Hypotension after induction
    - Decreased respiration: may require laryngeal mask for MEG studies

Retrospective review

Comparison Midazolam to Chloral Hydrate (premed) + Propofol (MEG)

Subjects and Methods

- Nonprotocol group (31 patients) 5.7+/- 4.3 yrs, 6 M
  - Premedication
  - Oral midazolam, chloral hydrate or fentanyl oralet, IV midazolam or inhalational anesthesia with sevoflurane.
  - Anaesthesia was maintained with propofol, midazolam, fentanyl, alone or in combination.
- Protocol group (17 patients) 4.9 +/- 2.3 yrs, 9 M
  - **Chloral hydrate** as premedication
  - **Propofol** for maintenance of anesthesia.

RESULTS:

- Overall: 25% failure of detection of **interictal epileptiform activity and localization** on the MEG scan.
- Nonprotocol group: 11 scans failed (35.5%). Of these, eight (72.7%) received midazolam orally.
- Protocol group: Only one failure (5.8%) was recorded in the in a patient who received chloral hydrate as sedation supplemented by sevoflurane.

CONCLUSIONS:

- Midazolam premedication resulted in a high MEG failure rate (73%).
- **Chloral hydrate premedication and propofol maintenance resulted in a lower incidence of MEG failure (5.8%).**
- General anaesthesia with a continuous infusion of propofol or sevoflurane appeared acceptable, although, lighter levels of anesthesia might be required to avoid interference with interictal activity of the brain.

Szmuk, P., et al. (2003) Paediatr Anaesth **13**(9): 811-817

Retrospective Study Propofol vs No Anesthesia

Detection of interictal epileptiform discharges in MEG

SUBJECTS AND METHODS:

- 41 epilepsy patients (Age range 1-48 year, ave age 9.9 +/- 9.6; 10 female) MEG while anesthetized.
- Anesthesia group of patients was compared with
  - All other patients with epilepsy who were recorded in the Center without anesthesia
  - ❖ **Subgroup of children with epilepsy who were able to be recorded without the need for anesthesia.**

RESULTS:

- Propofol 38 patients, etomidate 2, sevoflurane 1
- Twenty-nine (71%) had interictal epileptiform activity in MEG recording
- Comparable to percent (63%) found in epilepsy patients studied with MEG without anesthesia.
- ❖ **38 children younger than 18 yr, 28 (74%) had interictal epileptiform activity compared with 80% done without anesthesia**

CONCLUSION:

- **Levels of anesthesia needed to provide unconsciousness and immobility during MEG studies did not significantly alter the likelihood of recording interictal epileptiform spike activity with MEG.**

Balakrishnan, G., et al. (2007) Anesth Analg **104**(6): 1493-1497

MEG using propofol TIVA in pediatric patients with intractable epilepsy: MRI lesional vs nonlesional epilepsy

- METHODS:
  - 28 children (3-14 years; mean, 6.6).
  - IV propofol to record MEG with simultaneous EEG.
  - Evaluated MEG spike sources (MEGSSs).
  - Compared spikes on simultaneous EEG under TIVA with those on scalp video-EEG without TIVA.
- RESULTS:
  - Significant decrease in frequent spikes (10 patients, 36%) on simultaneous EEG under TIVA compared to those (22 patients, 79%) on scalp video-EEG without TIVA (P<0.01).
  - MEGSSs were present in 21 (75%) of 28 patients.
    - ❖ Clustered MEGSSs occurred in 15 (83%) of 18 lesional patients but in 3 (30%) of 10 nonlesional patients (P<0.05).
    - ❖ MEGSSs were more frequently absent in nonlesional (6 patients, 60%) than lesional (one patient, 5%) patients (P<0.01).
    - Thirteen patients with MRI and/or histopathologically confirmed neuronal migration disorder most frequently showed clustered MEGSSs (11 patients, 85%) compared to those of other lesional and nonlesional patients.
- CONCLUSION:
  - ❖ Propofol-based TIVA reduced interictal spikes on simultaneous EEG.
  - TIVA for MEG still had utility in identifying spike sources in a subset of pediatric patients with intractable epilepsy who were uncooperative and surgical candidates.
  - In lesional patients, MEG under TIVA frequently localized the clustered MEGSSs.
  - Neuronal migration disorders were intrinsically epileptogenic and produced clustered MEGSSs under TIVA.
  - ❖ Nonlesional patients often had no MEGSS under TIVA.

Fujimoto, A., et al. (2009) Brain Dev **31**(1): 34-41.

TIVA with propofol affecting spike sources of MEG in pediatric epilepsy patients: focal seizures vs. non-focal seizures

- PURPOSE: Magnetoencephalography (MEG) provides source localization of interictal spikes.
  - Evaluate inhibitory effects of propofol on MEG spike sources (MEGSSs) among different types of seizures in patients who underwent two separate MEG studies with and without total intravenous anesthesia (TIVA) using propofol.
- METHODS:
  - 19 children (1-14 years; mean, 6.2 years) who had MEG **with and without TIVA.**
  - ❖ TIVA was administered using propofol to record MEG with simultaneous EEG.
  - ❖ Analyzed number of spikes of MEG and MEGSSs comparing MEG studies done with and without TIVA.
- RESULTS:
  - Patients: 9 with focal seizure (FS) with/without secondary generalization, 5 with epileptic spasm (ES), and 5 with generalized seizure (GS).
  - ❖ Reduction of MEGSSs occurred in patients with FS under TIVA. (4.5/minute -> 2.0/minute)
  - ❖ Diffuse/generalized spikes in non-FS are not affected by TIVA.
  - ❖ Propofol may decrease focal spikes in the epileptic cortex in FS.
  - Cortical hyperexcitability in non-FS group would be stronger or more extensive than that in the FS group of patients.

Hanaya, R., et al. (2013) Epilepsy Res **105**(3): 326-336

Clinical Practice: Retrospective Review

- Patients with Propofol sedation
  - 90% of recordings readable.
  - In one year 2012, epileptiform discharges seen in 86% of patients
  - Simultaneous EEG was monitored for the presence of low amplitude background and presence of beta activity, both considered to represent excessive sedation. If seen, dose of propofol was titrated down.

Table I. *Details of number of patients scanned in the MEG center between September 2007 to December 2012 at Le Bonheur Children's Hospital.*

Year	2007	2008	2009	2010	2011	2012	Total
Clinical: non-sedation	22	84	58	35	15	91	305
Clinical: with sedation	3	3	40	32	13	49	140
Research	0	0	3	4	1	27	35
Total	25	87	101	71	29	167	480

Birg, L., et al. (2013) Neurodiagn J **53**(3): 229-240

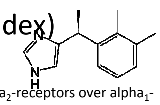
Effect on high frequency oscillations (HFOs)

- ECoG, intraoperative
  - 12 TLE patients with standard grid placement
  - Evaluated spikes, ripples, fast ripples diring induction, maintenance, and emergence
  - There was a decrease in number of channels with spikes, but no change in ripples or FRs
  - There was no change in the durations of HFOs.
  - Amount of HFOs in the presumed epileptogenic areas did not change more than the amount outside the presumed epileptogenic area,
  - spikes paradoxically decreased more within the suspected epileptogenic area.
  - 6 patients showing burst-suppression had lower rates of ripples than6other patients with continuous background activity,
  - No significant difference was found between burst suppression and continuous background activity in four patients, but there was a trend toward showing more ripples during continuous background activity (p = 0.16).
  - ❖ Propofol, known for its antiepileptic effects, reduces the number of epileptic HFOs, but has no effect on spikes.

Zijlmans, M., et al. (2012) Epilepsia **53**(10): 1799-1809.

- [Note: Higher doses of propofol would be used intraoperatively than during non-invasive MEG study, so findings may not be applicable to MEG studies]

Dexmedetomidine (Precedex)



- Relatively selective alpha$_2$-adrenergic agonist
- Chemically related to clonidine, but much greater affinity for alpha$_2$-receptors over alpha$_1$-receptors (1,620:1 compared to 200:1 for clonidine).
- Has activity at a variety of locations throughout the central nervous system.
  - Sedative and anxiolytic effects of dexmedetomidine result primarily from its activity in the locus ceruleus of the brain stem.
    - Stimulation of alpha$_2$-adrenergic receptors at this site reduce central sympathetic output, resulting in increased firing of inhibitory neurons.
  - Presence of dexmedetomidine at alpha$_2$-adrenergic receptors in the dorsal horn of the spinal cord modulates release of substance P and produces its analgesic effects.
- TIVA
  - Requires loading dose for induction; recovery from anesthesia slower than propofol
  - Does not interfere with respiration: nasal canula with O_2 only
  - Bradycardia is a side effect

http://www.medscape.com/viewarticle/524752_2
http://en.wikipedia.org/wiki/Dexmedetomidine

Dexmedetomidine for sedation during **EEG**

Children with autism, pervasive developmental disorders, and seizure disorders

- Subjects and Methods:
  - Retrospective: 42 children, aged 2 -11 years,
  - Dexmedetomidine for sedation during EEG analysis
- MAIN RESULTS:
  - 18 children received oral dexmedetomidine before placement of an i.v..
  - 40 patients received an i.v. loading dose of dexmedetomidine
  - Effective sedation was eventually achieved in all patients.
  - I.V. infusion of dexmedetomidine was started in all patients.
- CONCLUSIONS:
  - **Dexmedetomidine provides effective sedation during EEG analysis in children with autism or PDD.**

Ray, T. and J. D. Tobias (2008) J Clin Anesth **20**(5): 364-368

Effects of dexmedetomidine sedation on the **EEG** in children with epilepsy

- PURPOSE:
  - Compare **dexmedetomidine sedation** on EEG background and epileptiform activity in children to **natural sleep.**
- MATERIALS/METHODS:
  - 16 children undergoing dexmedetomidine sedation for nuclear medicine studies and simultaneous continuous EEG monitoring were studied.
  - ❖ EEG segments during sedation were compared to samples of naturally occurring stage II sleep from the same child.
  - Standard visual EEG analysis, quantification of delta, theta, alpha, beta, and total RMS power, number and location of spike foci, and frequency of spike activity were compared.
- RESULTS:
  - EEG during dexmedetomidine sedation **resembled stage II sleep**.
  - During sedation, statistically significant increases in power of 16% for theta ($P = 0.01$), 21% for alpha ($P = 0.03$), and 40% for beta ($P < 0.01$) were observed, but not for delta ($P = 0.63$) or total EEG power ($P = 0.61$).
  - ❖ Spike frequency increased by 47% during sedation but no new spike foci or seizures were observed.
- CONCLUSION:
  - ❖ Dexmedetomidine sedation elicited an EEG pattern similar to that of Stage II sleep with modest increases in theta, alpha, and beta activity.
  - ❖ Dexmedetomidine does not hinder interpretation of the EEG, suggesting that it may be a uniquely useful agent for EEG sedation in children

Mason, K. P., et al. (2009) Paediatr Anaesth **19**(12): 1175-1183

Dexmedetomidine side effects in Children

Radiographic Studies

- PURPOSE:
  - Determine safety, efficacy, and outcomes of bradycardia, hypotension, and hypertension with dexmedetomidine
- PATIENTS AND METHODS:
  - 669 patients (mean age, 5.7 years +/- 4.5 [standard deviation]; median age, 4.5 years; age range, 0.1-22.5 years
  - Dexmedetomidine given as loading dose then continuous infusion.
- RESULTS:
  - Of 669 studies, 667 (99.7%) were completed successfully.
  - Six children (0.9%) had brief periods of oxygen desaturation below 95%, none of which required airway intervention.
  - Hypotension, hypertension, and bradycardia (all defined as deviations of more than 20% from age-adjusted awake norms), occurred in 58.7%, 2.1%, and 4.3% of patients, respectively.
  - Both hypotension and bradycardia were related to age ($P = .033$ and $P = .002$, respectively); older children tended to experience more of these events.
  - None of these fluctuations required pharmacologic therapy.
- CONCLUSION: Dexmedetomidine offers advantages for pediatric sedation for nuc med imaging.
  - Dexmedetomidine produces a condition similar to natural sleep, with no detrimental effect on respiration.
  - The hemodynamic variability anticipated with dexmedetomidine did not require pharmacologic treatment, and the drug was well tolerated.

Mason, K. P., et al. (2013) Radiology **267**(3): 911-917

Effects of dexmedetomidine on intraoperative motor and somatosensory evoked potential monitoring during spinal surgery in adolescents

- PURPOSE:
  - Evaluate dexmedetomidine as adjunct to an opioid-propofol total intravenous anesthesia (TIVA) technique during posterior spinal fusion (PSF) surgery
- METHODS:
  - Retrospective review of prospectively collected quality assurance data.
  - SSEPs and MEPs were measured before and after the administration of dexmedetomidine in a cohort of pediatric patients undergoing PSF.
  - Dexmedetomidine (IV load followed by infusion) was administered at the completion of the surgical procedure, but prior to wound closure as an adjunct to TIVA which included propofol and remifentanil, adjusted to maintain a constant depth of anesthesia as measured by a BIS of 45-60.
- RESULTS:
  - 9 patients, (age 12 to 17 years)
  - First anesthetized with remifentanil and propofol.
  - There was no statistically significant difference in the MEPs and SSEPs obtained before and at completion of the dexmedetomidine loading dose.
- CONCLUSION:
  - Using the above-mentioned protocol, dexmedetomidine can be used as a component of TIVA during PSF without affecting neurophysiological monitoring.

Tobias, J. D., et al. (2008) Paediatr Anaesth **18**(11): 1082-1088

Influence of anesthetic management on quality of MEG scan data in pediatric patients

- Patients and METHODS:
  - Retrospectively reviewed the records of all patients who underwent MEG scanning at our institution with regard to the interaction of anesthetic management and quality of scan data.
  - 19 patients: age range 2-16 yr
    - 6 patients with propofol
    - 13 patients with dexmedetomidine
- RESULTS:
  - High-dose propofol infusions (> or =200 microg.kg(-1).min(-1)) were associated with high frequency artifacts that interfered with the identification of epileptiform discharges.
  - Lower-dose propofol infusions (< or =100 microg.kg(-1).min(-1)) did not produce artifacts but required co-administration of fentanyl to prevent patient motion.
  - Dexmedetomidine infusions were not associated with signal artifacts and prevented patient motion very well in our initial patients and became our standard technique.
- CONCLUSION:
  - Dexmedetomidine infusions are preferable to propofol-based techniques for pediatric MEG scans due to the absence of adverse effect on interictal activity.

Konig, M. W., et al. (2009). Paediatr Anaesth **19**(5): 507-512

Comparison of propofol- and dexmedetomidine-induced EEG dynamics in normal adults: Spectral and Coherence Analysis

- BACKGROUND:
  - EEG patterns observed during sedation with dexmedetomidine appear similar to those observed during general anesthesia with propofol.
  - Occurrence of delta (1 to 4 Hz), propofol-induced alpha (8 to 12 Hz), and dexmedetomidine-induced spindle (12 to 16 Hz) oscillations.
- METHODS:
  - 17 healthy volunteers, 18 to 36 yr of age. dexmedetomidine (n = 9) and propofol (n = 8
  - measured 64-channel EEG
  - Volunteers listened to auditory stimuli and responded by button press to determine unconsciousness.
  - EEG analyzed using multitaper spectral and coherence analysis.
- RESULTS:
  - Dexmedetomidine was characterized by spindles with maximum power and coherence at approximately 13 Hz (mean +/- SD; power, -10.8 +/- 3.6 dB; coherence, 0.8 +/- 0.08),
  - propofol was characterized with frontal alpha oscillations with peak frequency at approximately 11 Hz (power, 1.1 +/- 4.5 dB; coherence, 0.9 +/- 0.05).
  - Notably, slow oscillation power during a general anesthetic state under propofol (power, 13.2 +/- 2.4 dB) was much larger than during sedative states under both propofol (power, -2.5 +/- 3.5 dB) and dexmedetomidine (power, -0.4 +/- 3.1 dB).
- CONCLUSION:
  - The results indicate that dexmedetomidine and propofol place patients into different brain states and suggest that propofol enables a deeper state of unconsciousness by inducing large-amplitude slow oscillations that produce prolonged states of neuronal silence.

Akeju, O., et al. (2014) Anesthesiology **121**(5): 978-989

CCHMC Update: Sedation and Anesthesia for MEG Studies 2006-2014 520 presurgical epilepsy patients

- No sedation – 260 patients
  - Cooperative patients that can be sleep-deprived
    - Usual overnight sleep hours reduced by at least 50% - begin study 7-7:30 am
- Conscious sedation – 132 patients
  - Chloral Hydrate:
    - Not more than 50 mg/kg PO
    - Maximum of 1000 mg total dosage - the criteria usually limit suitable patients to under 6 y/o
    - Sleep-deprived by 50% - begin study 7-7:30 am
    - NPO overnight
    - Nurse monitors pulse oximetry, heart rate, respirations continuously and BP at intervals
    - Onset of sleep within ~45 minutes usually; duration ~1.5 hours; usually able to obtain SEF with electrical stim at end of study
    - Chloral hydrate + vigabatrin AED has produced high frequency artifact – MEG interpretation difficult
    - If patient fails to fall asleep by 1.5 hours, consider reschedule as a GA study
    - Magnetoencephalographer required to have current Pediatric Advanced Life Support (PALS) certification
    - Records kept in EMR for patient evaluation before, and status after, sedation procedure

CCHMC Update: Sedation and Anesthesia for MEG Studies
2006-2014   520 patients

- General anesthesia (deep sedation) – 128 patients
  - Prep: NPO after midnight; no sleep deprivation required
  - Dexmedetomidine TIVA
    - Loading dose 2u/kg over 10 minute
    - Then 2u/kg/hour maintenance
    - For SEF at end of study usually small amt additional med required (Propofol or Fentanyl)
    - After study completed, transport to surgical recovery room for monitoring until awake
  - Anesthesiologist manages all meds and vital sign monitoring
  - Only a few patients did not have epileptiform discharges; also had few or no epileptiform discharges in EMU and no epileptiform discharges on prior 'natural' sleep deprivation MEG study

SUMMARY

- Chloral hydrate PO with prior sleep deprivation can be useful for MEG sleep study lasting 1.5 hours and SEF at end of study (most patients)
  - Dosing at least 50 mg/kg (at our institution, also our maximum)
  - Age range applicable may depend on institution and maximal dose CH
  - Nurse present for VS monitoring and Magnetoencephalographer PALS certified
- GA
  - Propofol TIVA faster induction and emergence than dexmedetomidine TIVA
  - Both require presence of anesthesiologist and equipment/gases
  - Propofol can have bifrontal alpha frequency artifact and delta slowing; dosage may need to be adjusted to reduce artifact if present
  - Effect of propofol on incidence of spikes well studied; may reduce somewhat
  - Effect of dexmedetomidine on incidence of spikes not yet well studied; may increase somewhat
  - Effect of either on detection of activity in high beta and gamma frequencies not yet well studied
  - Both agents have been used successful for general anesthesia for MEG studies for detection of epileptiform discharges with few if any major side effects

References I

- Akeju, O., Pavone, K. J., Westover, M. B., Vazquez, R., Prerau, M. J., Harrell, P. G., . . . Purdon, P. L. (2014). A comparison of propofol- and dexmedetomidine-induced electroencephalogram dynamics using spectral and coherence analysis. *Anesthesiology, 121*(5), 978-989.
- Balakrishnan, G., Grover, K. M., Mason, K., Smith, B., Barkley, G. L., Tepley, N., & Bowyer, S. M. (2007). A retrospective analysis of the effect of general anesthetics on the successful detection of interictal epileptiform activity in magnetoencephalography. *Anesth Analg, 104*(6), 1493-1497, table of contents. [
- Bercovici, E., Pang, E. W., Sharma, R., Mohamed, I. S., Imai, K., Fujimoto, A., . . . Otsubo, H. (2008). Somatosensory-evoked fields on magnetoencephalography for epilepsy infants younger than 4 years with total intravenous anesthesia. *Clin Neurophysiol, 119*(6), 1328-1334. doi: S1388-2457(08)00155-7
- Birg, L., Narayana, S., Rezaie, R., & Papanicolaou, A. (2013). Technical tips: MEG and EEG with sedation. *Neurodiagn J, 53*(3), 229-240.
- Fujimoto, A., Ochi, A., Imai, K., Chan, D., Sharma, R., Viljoen, A., . . . Otsubo, H. (2009). Magnetoencephalography using total intravenous anesthesia in pediatric patients with intractable epilepsy: lesional vs nonlesional epilepsy. *Brain Dev, 31*(1), 34-41. doi: S0387-7604(08)00173-3 [
- Gozal, D., & Gozal, Y. (2008). Pediatric sedation/anesthesia outside the operating room. *Curr Opin Anaesthesiol, 21*(4), 494-498.
- Hanaya, R., Okamoto, H., Fujimoto, A., Ochi, A., Go, C., Snead, C. O., 3rd, . . . Otsubo, H. (2013). Total intravenous anesthesia affecting spike sources of magnetoencephalography in pediatric epilepsy patients: focal seizures vs. non-focal seizures. *Epilepsy Res, 105*(3), 326-336.
- Kirchberger, K., Schmitt, H., Hummel, C., Peinemann, A., Pauli, E., Kettenmann, B., & Stefan, H. (1998). Clonidine and methohexital-induced epileptic magnetoencephalographic discharges in patients with focal epilepsies. *Epilepsia, 39*(8), 841-849.
- Konig, M. W., Mahmoud, M. A., Fujiwara, H., Hemasilpin, N., Lee, K. H., & Rose, D. F. (2009). Influence of anesthetic management on quality of magnetoencephalography scan data in pediatric patients: a case series. *Paediatr Anaesth, 19*(5), 507-512. doi: PAN2983 [
- Mason, K. P., O'Mahony, E., Zurakowski, D., & Libenson, M. H. (2009). Effects of dexmedetomidine sedation on the EEG in children. *Paediatr Anaesth, 19*(12), 1175-1183. doi:

References II

- Mason, K. P., Robinson, F., Fontaine, P., & Prescilla, R. (2013). Dexmedetomidine offers an option for safe and effective sedation for nuclear medicine imaging in children. *Radiology, 267*(3), 911-917. doi: 10.1148/radiol.13121232
- McMorrow, S. P., & Abramo, T. J. (2012). Dexmedetomidine sedation: uses in pediatric procedural sedation outside the operating room. *Pediatr Emerg Care, 28*(3), 292-296. doi: 10.1097/PEC.0b013e3182495e1b
- Munoz-Yunta, J. A., Ortiz, T., Palau-Baduell, M., Martin-Munoz, L., Salvado-Salvado, B., Valls-Santasusana, A., . . . Dursteler, C. (2008). Magnetoencephalographic pattern of epileptiform activity in children with early-onset autism spectrum disorders. *Clin Neurophysiol, 119*(3), 626-634. doi: S1388-2457(07)00652-9 [pii]
- Ray, T., & Tobias, J. D. (2008). Dexmedetomidine for sedation during electroencephalographic analysis in children with autism, pervasive developmental disorders, and seizure disorders. *J Clin Anesth, 20*(5), 364-368. doi: 10.1016/j.jclinane.2008.03.004
- Rezaie, R., Narayana, S., Schiller, K., Birg, L., Wheless, J. W., Boop, F. A., & Papanicolaou, A. C. (2014). Assessment of hemispheric dominance for receptive language in pediatric patients under sedation using magnetoencephalography. *Front Hum Neurosci, 8*, 657. doi: 10.3389/fnhum.2014.00657
- Souter, M. J., Rozet, I., Ojemann, J. G., Souter, K. J., Holmes, M. D., Lee, L., & Lam, A. M. (2007). Dexmedetomidine sedation during awake craniotomy for seizure resection: effects on electrocorticography. *J Neurosurg Anesthesiol, 19*(1), 38-44. doi: 10.1097/01.ana.0000211027.26550.24
- Szmuk, P., Kee, S., Pivalizza, E. G., Warters, R. D., Abramson, D. C., & Ezri, T. (2003). Anaesthesia for magnetoencephalography in children with intractable seizures. *Paediatr Anaesth, 13*(9), 811-817. doi: 1159 [pii]
- Tobias, J. D., Goble, T. J., Bates, G., Anderson, J. T., & Hoernschemeyer, D. G. (2008). Effects of dexmedetomidine on intraoperative motor and somatosensory evoked potential monitoring during spinal surgery in adolescents. *Paediatr Anaesth, 18*(11), 1082-1088. doi: 10.1111/j.1460-9592.2008.02733.x
- Zijlmans, M., Huiskamp, G. M., Cremer, O. L., Ferrier, C. H., van Huffelen, A. C., & Leijten, F. S. (2012). Epileptic high-frequency oscillations in intraoperative electrocorticography: the effect of propofol. *Epilepsia, 53*(10), 1799-1809. doi: 10.1111/j.1528-1167.2012.03650.x

Somatosensory-evoked fields during MEG for epilepsy infants younger than 4 years with total intravenous anesthesia (TIVA)

Subjects and METHODS:

- 26 infants (mean age=2.6 years).
  - 17 patients underwent TIVA
  - 9 patients were tested while asleep, without TIVA.
- Investigated latency, amplitude, residual error (RE) and location of the N20m of the SEF in

RESULTS:

- MEG detected 44 reliable SEFs (77%) in 52 median nerve stimulations.
  - 27 reliable SEFs (79%) with TIVA (propofol)
  - 13 reliable SEFs (72%) without TIVA.
  - TIVA effects included longer latencies ($p<0.001$) and lower RE ($p<0.05$) compared to those without TIVA.
  - Older patients and larger head circumferences also showed significantly shorter latencies ($p<0.01$).

CONCLUSIONS:

- TIVA resulted in reliable SEFs with lower RE and longer latencies.

SIGNIFICANCE:

- MEG can detect reliable SEFs in infants younger than 4 years old.

– When infants require TIVA for MEG and MRI acquisition, SEFs can still be reliably observed.

Bercovici, E., et al. (2008) Clin Neurophysiol **119**(6): 1328-1334

ACMEGS

AMERICAN CLINICAL MEG SOCIETY

Pediatric MEG: The Effect of Head Positioning on SEF

William Gaetz, MD

Somatosensory responses can be evoked by electrical stimulation of a peripheral nerve (e.g., median nerve) or by tactile stimulation of the skin (e.g., on the digits). Clinically, these responses can be used for pre-surgical functional mapping as well as to explore a variety of clinical research questions. Early neural responses are well characterized as a single equivalent current dipole (ECD) and thus the SEF source model can be used to address basic questions about practical aspects of source measurement using MEG. For example, most current MEG devices have been designed to accommodate adult head sizes, yet are commonly used in Pediatric clinical facilities. As a result, SEF measurements in children are likely sub-optimal, as the magnitude of a magnetic field is known to fall off rapidly as a function of distance from source to sensor. In other words, when recording MEG from a child in an MEG helmet designed for adults, the space separating the source and sensor array may cause problems of signal detection.

In this presentation, I will first touch on a few clinical examples detailing the clinical utility of the SEF response in Clinical Pediatrics. Then we will explore how the SEF source parameters change when positioning the head to minimize the distance between the expected SEF source and the inner wall of the MEG dewar. Finally, head motion tracking and motion correction strategies are briefly considered. Overall, these data argue for the continued development of MEG devices specifically designed for use with children.

Pediatric MEG: The effect of head positioning on SEF

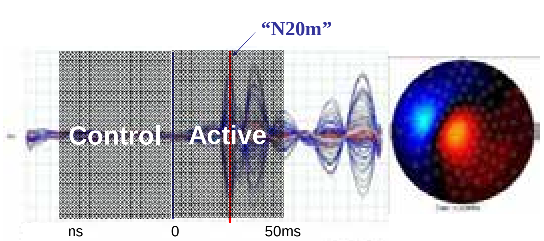
William Gaetz PhD.

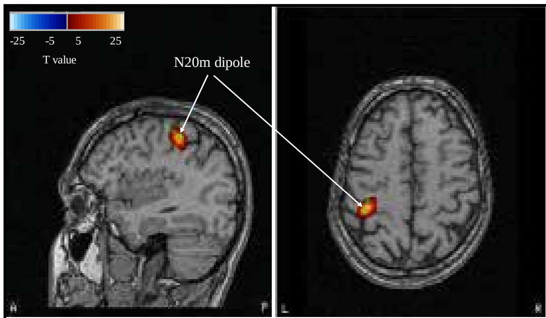
Department of Radiology
Lurie Families' MEG Center
The Children's Hospital of Philadelphia

Talk Outline

- Somatosensory Evoked Fields (SEFs) and paediatrics
  - Why are we interested in the clinical measurement of these responses in kids?
  - What are some of the problems we encounter when doing so?
  - What are some solutions?

Somatosensory Evoked Fields





Difference (pseudo-T)
SAM volumetric images
(Active - Control)

blue = decrease
red = increase

Filter Resolution: 0.5 mm
Source power ($\mu = 0$)
Time = 0.050 s / condition
BW = 15 to 300 Hz

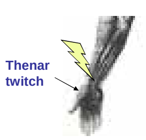
Gaetz and Cheyne (2003); *Neuroscience Letters*

Why record Somatosensory Evoked Fields?

- SEPs are well known responses – long standing use for decades prior to advent of MEG.
- SEFs are often used to indicate central sulcus "hand area" for pre-surgical mapping.
- SEFs for clinical research: e.g., developmental response trajectories may hint at age-dependent changes in E/I balance.

Electrical and Tactile Somatosensory Evoked Fields

Median Nerve Stimulation



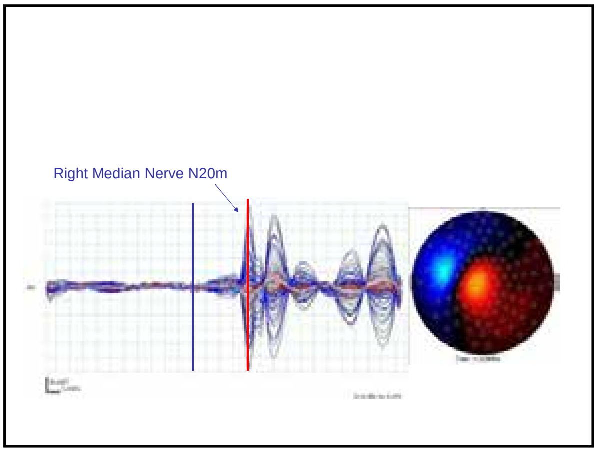
Benefit:
Well studied
Strong SNR

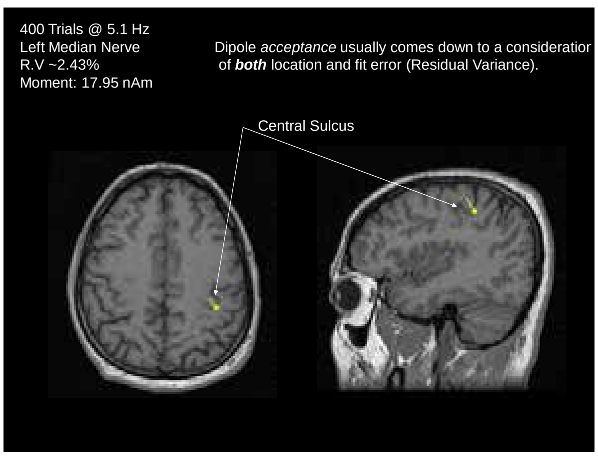
Cost:
- Somewhat painful
- Artifact esp. in ped patients

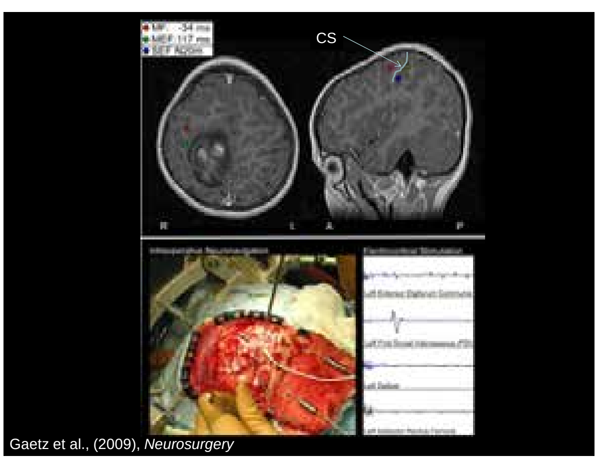
Pneumatic Tactile Stimulation

Benefit:
Well tolerated in peds.
Digit Somatotopy

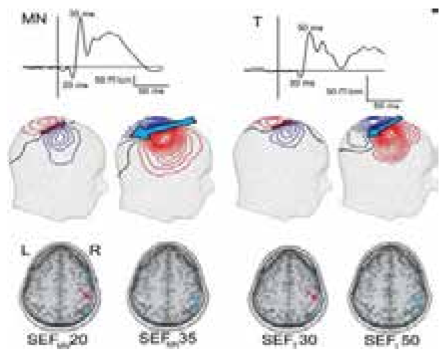
Cost:
- Less well-known
- More time required
- No physiological response
- Contact may vary over time


Right Median Nerve N20m


400 Trials @ 5.1 Hz
Left Median Nerve
R.V ~2.43%
Moment: 17.95 nAm
Dipole *acceptance* usually comes down to a consideratior
of ***both*** location and fit error (Residual Variance).
Central Sulcus


CS
Gaetz et al., (2009), *Neurosurgery*

Electrical and Tactile Somatosensory Evoked Fields

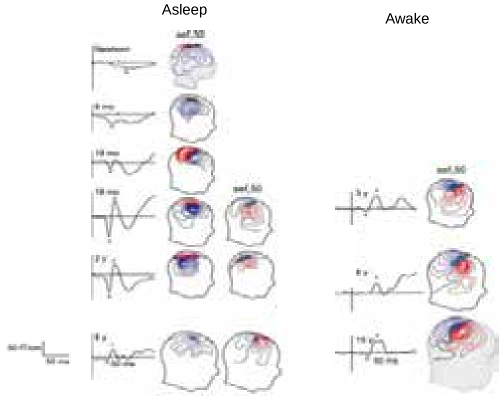


Nevalainen, et al., 2014

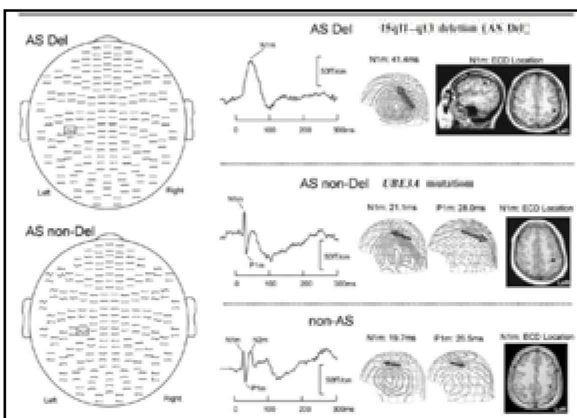
Tactile SEFs and Age

Asleep

Awake



Nevalainen, et al., 2014



Egawa et al., (2008) - NeuroImage

Issues with SEF measurement in Children

Some children in our care cannot remain still MEG. --> General Anesthetic (GA)

The anesthetic is expected to ***somewhat*** attenuate signals from cortex.

A significant portion (~30%) of GA patients 3 years or younger show attenuated SEF activity.

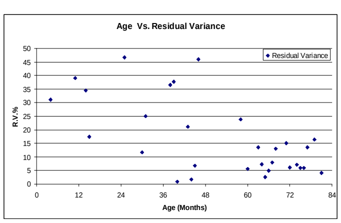
- No "N20m"
- Poor dipole localization
- High residual variance

Yet, in these cases we often observe Somatosensory (SEP) activity using EEG without sedation.

GA patients are :
- Anaesthetized
- Relatively young
  - Body mass by anaesthetics interaction?
  - Young people are generally smaller – Head size??
  - Both?

Retrospective Analysis (HSC)

- Look at previous patient data under GA.
  - 79 GA patients with intractable epilepsy (age 4 months to 7 years).
  - 33 cases had disease confined to a single hemisphere.
  - Observe SEF source parameters (Residual Variance and Moment) for the healthy hemisphere only.



N=33 GA patients
Age range= 4 months to 7 years
Healthy hemisphere only
400 trials @ 5.1 Hz

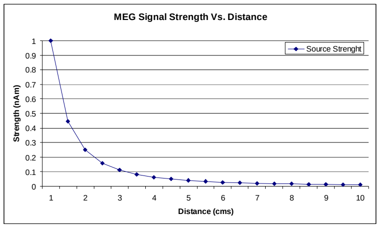
- Residual Variance decreases with age.

3 Patients ***failed to show any SEF signal***: (7, 8 & 39 Months)

Conclusion: Young patients exhibit poorer dipole ***source fits in younger children.***

MEG Physics

- Biot - Savart law: the magnetic field strength falls off with the *square of distance* from a current source.

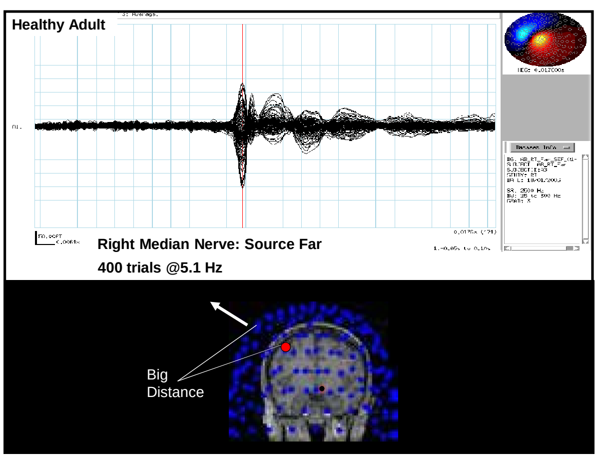


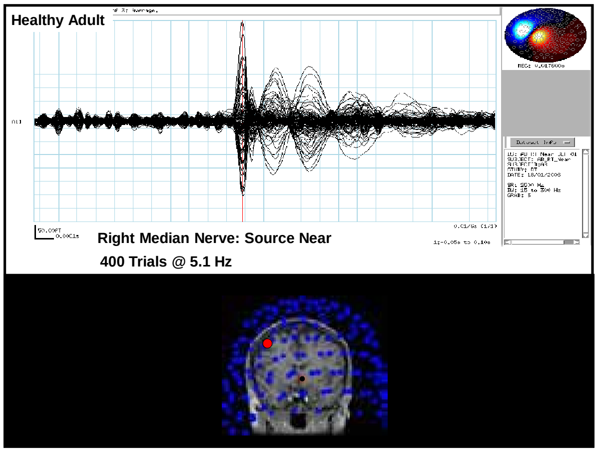
Effect of Head Position on SEFs

Run 1: Position the head centrally - standard SEF.
Run 2: Re-position the head to ***minimize*** the *distance* - and repeat the SEF measurement.

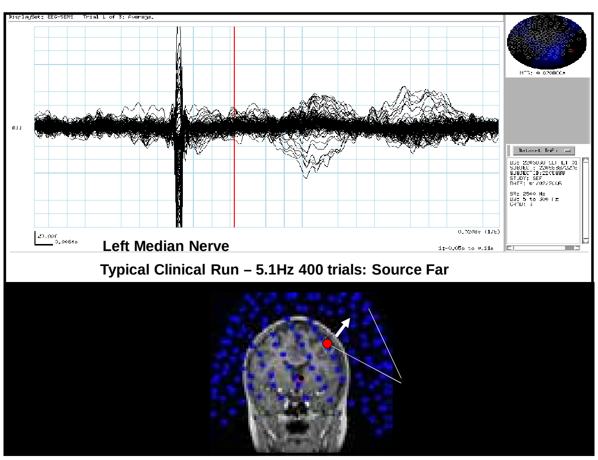
N=17 Pre-surgical functional mapping patients (under GA; age 11 mo. – 13 Yrs.)

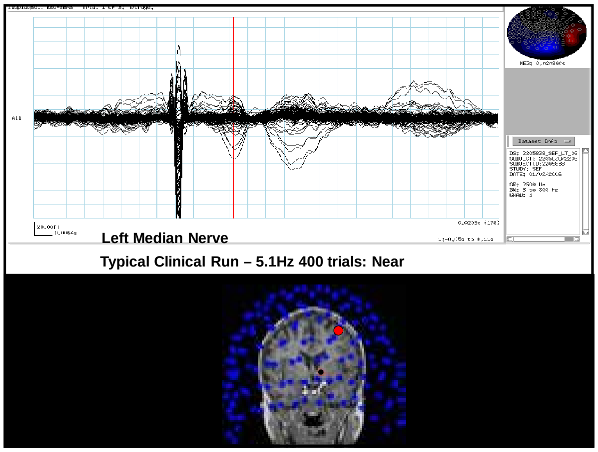
400 trials (5.1 Hz)

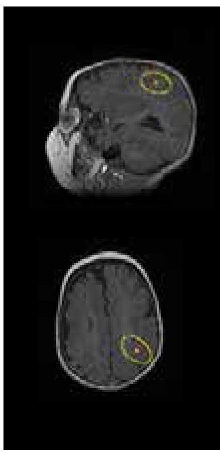




- 11 month old patient



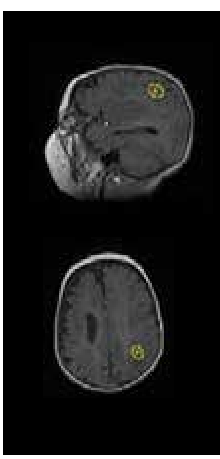




11 Month Old (Centred)

- 95% Confidence Volume

- 39.06% Residual Variance
- Dipole Moment 2.97 nAm



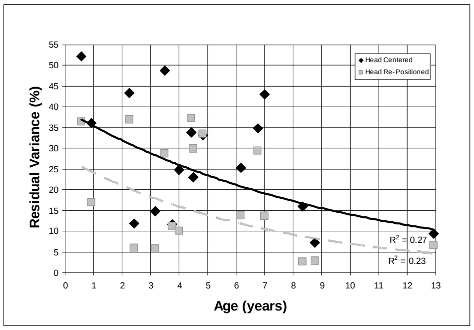
11 Month Old (Repositioned)

- 95% Confidence Volume

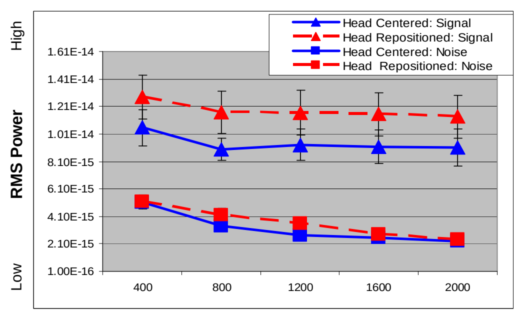
- 16.99% Residual Variance
- Dipole Moment 3.98 nAm

- Repositioning produces a ***smaller*** confidence volume, and a ***more anatomically correct*** location.

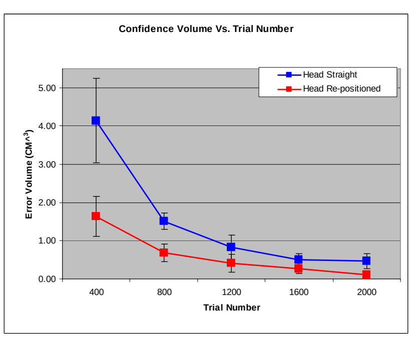
Head Position and Residual Variance



Head Position and SNR



Head Position Confidence Volumes



Why Not Reposition?

- Wilke et al., 2009 fMRI; TMS and MEG (MCA strokes (n=6); periventricular lesions(n=8)).
  - Two types of motor (re)-organization
    - Preserved contralateal MI
    - Ipsilateral MI
  - Sensory impairment was worse for the group with preserved motor function.
  - In all cases, SI was observed contralaterally (fMRI and MEG).

"We conclude that no intrahemispheric reorganization of the somatosensory functions occurred".

  - Motor and somatosensory systems have entirely divergent compensatory profiles.

Conclusions

- In young children, lateralized sources such as the SEF can be better measured with physical adjustments to head position to ***minimize*** the distance between the source and the sensors.
  - Patients under the age of 7 or with a head circumference of < 50 cms should be repositioned prior to standard SEF recording.
  - This procedure is likely to:
    - Improve the accuracy of our paediatric clinical SEF measurements.
    - Significantly improve ("save") dipole results in very young patients.

What about measurements involving young children and bi-laterally active sources:
  - probably best to use a head centred position – although SNR will be sub-optimal.
- These data underscore the importance of developing an MEG specifically engineered for young children.

Acknowledgements

Philadelphia - CHOP
Timothy P.L. Roberts, PhD
J .Chris Edgar, PhD
Luke Bloy, PhD
D.J. Wang, PhD
Erin Simon Schwartz, MD
John Dell
Peter Lam
Rachel Golembski

Toronto – HSC
Elizabeth Pang, PhD
Hiroshi Otsubo, PhD
Roy Sharma
Amrita Hunjan
Bill Chu
Stephanie Holowka

Passive Language Mapping with MEG in Pediatric Patients with Epilepsy

Dave Clark, MD

MEG Localization of Broca's Area Using Verb Generation Tasks
Elizabeth Pang, MD

ACMEGS 2015: Current Issues and Controversies in Pediatric MEG

MEG localization of Broca's Area using verb generation tasks

Elizabeth W. Pang, Ph.D.
Division of Neurology / Department of Paediatrics
Hospital for Sick Children / University of Toronto
Toronto, Ontario, Canada

February 5, 2015

SickKids

UNIVERSITY of TORONTO

WHY is language mapping so important?

- Neurosurgery in areas within or adjacent to critical language areas may result in permanent post-surgical language impairment or loss.
- Lateralization and localization of language helps in risk determination – this information can change the course of treatment or preclude the surgical intervention entirely.
- Pre-surgical functional mapping is very important in patients with mass lesions or epileptogenic foci since these often are associated with atypical language lateralization.
- Developmental plasticity: Early left-hemisphere insult was associated with increased atypical (right or bilateral) language representation (55%) compared to individuals with late left-hemisphere injuries (16%) (Milner & Rasmussen, 1977)

Pre-operative Gold Standard

For language lateralization:

– intracarotid amytal test (IAP / WADA) (Wada & Rasmussen, 1960)

Generally reliable, but has a few drawbacks:

– Invasive, time consuming, increased risk of stroke, results may be confounded due to unusual cerebral vascular perfusion effects; in event of mixed language dominance, cannot sort out neural involvement

For language localization:

– electro-cortical stimulation (via wake up test / implanted electrodes)

Limitations:

– very invasive, dependent on cooperativity, no pre-operative information, cannot test entire network -- limited by size of craniotomy

fMRI: becoming the new gold standard

Offers lateralizing information via a laterality index:

– most common method is to count the number of activated voxels above a particular statistical threshold and calculate laterality index

Advantages:

– non-invasive, no side-effects, available pre-operatively, equipment available at most institutions, full-brain coverage

Limitations of fMRI:

– can be used for relative localization (i.e., planning the surgical route), but cannot be used for precise localization

Advantages of MEG

- MEG can lateralize and localize neural areas.
- **MEG is a more direct marker of neuronal activity.** This is especially relevant in children as the BOLD-fMRI signal increases with age (Schapiro et al 2004).
- Development of different stages of language processing are probably reflected as **changes in the timing** of neural activation.
- More **paediatric / patient friendly** due to quieter environment and less physical constraints.

Challenges of using MEG to localize expressive language:

- head movement
- muscle artefacts
- each response is slightly different

Developing a MEG expressive language task

- tasks that work with fMRI:
  - verb generation
  - verbal fluency
  - reading
  - picture naming
  - etc.
- differences in paradigm design between MEG and fMRI:
  - no blocking
  - no visual scanning allowed
  - not limited to visual tasks

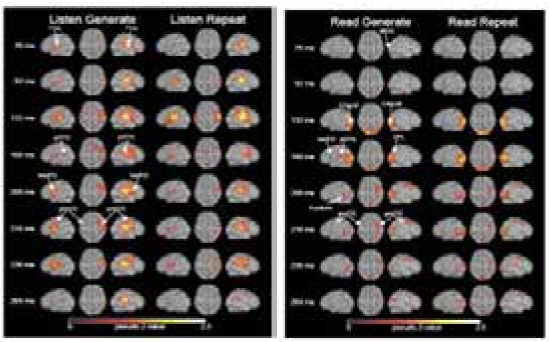
MEG Expressive Language Study #1:

Subjects:
- control adults (aged 20-42 yrs) (6 males, 2 females), all right-handed

Language Task: overt expressive language tasks
- in the visual modality:
  - read a noun
  - generate a verb
- in the auditory modality:
  - repeat a noun
  - generate a verb

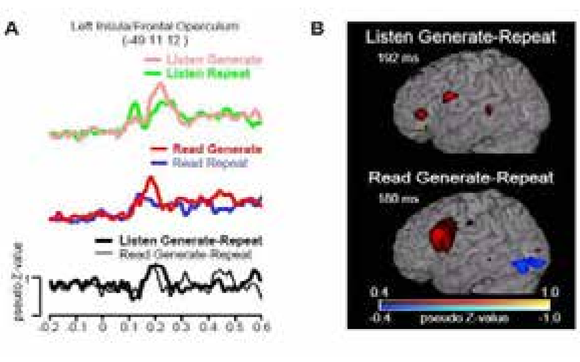
Research questions:
- Which is better for activating Broca's area: auditory or visual presentation?
- Is it sufficient to simply produce language or do you need to do something more "generative"?

Results



Herdman, et al., Cerebral Cortex (2007)

Results (cont'd)



Herdman, et al., Cerebral Cortex (2007)

Summary of Study #1

These findings show that auditory and visual word presentations during verb generation and repeat tasks produce a fast sequence of activity:

- immediate large activations within bilateral primary sensory cortices (75-130 ms), followed by association cortices between 130 – 170 ms, followed by inferior frontal and premotor areas between 150 – 240 ms.

From a practical perspective,

- visual tasks seem to produce clearer activations
- "generative" nature of task is important.

Next Questions:

Can we create an expressive language task that will

- work for young children and for the pediatric clinical population?
- and not require an overt response?

MEG Expressive Language Study #2: pictures instead of words

Subjects:

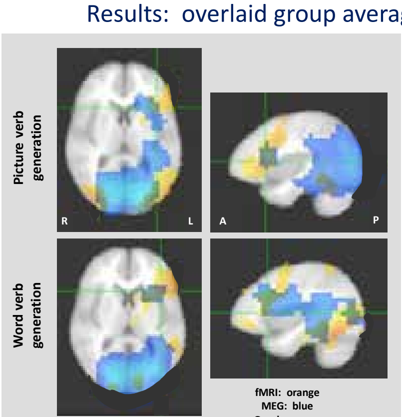
- 10 teenagers (14 – 18 years; 5 males, 5 females), all right-handed

Same tasks used in both MEG and fMRI:

- picture verb generation
- word verb generation

- **covert** expressive language task with **vigilance** task

Results: overlaid group average data



Pang, et al., Neuroscience Letters (2011)

Results (cont'd)

- We used a voxel count approach to compare MEG and fMRI activations.

At p<0.005 on fMRI:

- **for picture verb generation:**
  - the number of overlapping voxels identified by both modalities was 80±15 resulting in a 79.6% overlap.
- **for word verb generation:**
  - the number of overlapping voxels was 61±13 voxels resulting in 50.2% overlap.

Pang, et al., Neuroscience Letters (2011)

Summary of Study #2

- Our MEG tasks identified left inferior frontal and left dorsolateral pre-frontal regions involved in verb generation.
- There is a clear area of overlap between MEG and fMRI, but the extent of the overlap is quite different.
- This underlines the basic differences in the mechanisms underlying MEG and fMRI.
- From a practical perspective,
  - we can do this in the MEG,
  - picture stimuli work just as well, or better than, word stimuli.

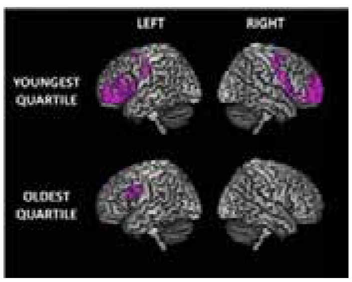
New questions:

- Can we use the picture verb generation task to track the development of expressive language from childhood into adolescence?
- If language representation is "atypical" in a young patient's brain, is this a function of disease or development?
  - evidence that early insult leads to inter-hemispheric shifts while later insults lead to intra-hemispheric re-organization
- Can we use this task in the clinical population to localize expressive language for pre-surgical planning?

Expressive Language Study #3: tracking typical and atypical development

- **Subjects:**
  - typically developing children aged 5 -18 years
  - children with epilepsy or tumours requiring pre-surgical language mapping
- **MEG Language Task:**
  - **covert** expressive language task with **vigilance** task
    - picture verb generation (preferred task)
    - picture naming (if child is unable to perform verb generation task)

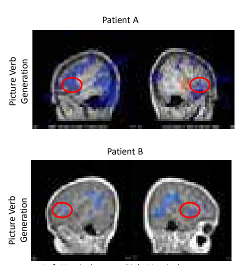
Results: typically developing children



Kadis, et al., J Int Neuropsychological Society(2011)

Results: atypical language

- thus far, 21 children with:
  - epilepsy, tumours, arteriovenous malformations
- Patient A:
  - 11 year old girl
  - localization related epilepsy arising from right hemisphere posterior temporal and parietal regions
  - MEG demonstrated bilateral frontal regions for expressive language, consistent with fMRI
- Patient B:
  - 11 year old girl
  - large periventricular left hemisphere AVM
  - MEG demonstrated bilateral frontal regions for expressive language



Final summary

- Language is a complex function that involves a network of neural areas.
- MEG is a promising tool that allows the localization of expressive language function to Broca's area.
- We have validated our MEG task against fMRI and find a high degree of overlap, especially with picture verb generation.
- We have used this task in typically developing children and can see activation of classic Broca's area. We have used this task in children with clinical conditions where language is expected to be atypical, and we find atypical localizations, consistent with fMRI.

Complexity Analysis of MEG in Traumatic Brain Injury Patients

Richard Bucholz, MD

ACMEGS

AMERICAN CLINICAL MEG SOCIETY

Human Brain Development Research in MEG

Joshio Okada, MD

(presented by Christos Papadelis, MD)

Human brain development research with babyMEG

Yoshio Okada
Director, MEG Program, Division of Newborn Medicine, Dept. Medicine, Boston Children's Hospital
Fetal and Neonatal Neuroimaging and Development Science Center, Div. Newborn Medicine
Clinical Professor, Harvard Medical School

February 2, 2015

Collaborators

- **Tristan Technologies (babyMEG construction)**
  Douglas Paulson, Anthony Mascarenas, Kevin Pratt, Bill Power, Menglai Han, Paul Miller, Jose Robles, Anders Cavellini, Kosal Sang

- **MGH/Martinos Center (real-time MEG software)**
  - Matti Hamalainen, Christoph Dinh, Martin Luessi, Seok Lew, Limin Sun, Aapo Nummenmaa

- **Boston Children's Hospital (system integration, research)**
  - Ellen Grant (Director, FNNDSC), Christos Papadelis, Banu Ahtam, Chiran Doshi, Tapsya Nayak
  - Phillip Pearl (Chief, Epilepsy Div, Dept. Neurol.), Tobias Loddenkemper, Jurriaan Peters, Alexander Rotenberg, Chellamani Harini

New MEG Facility: Main Hospital Building Boston Children's Hospital, Boston, MA

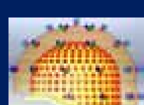
Boston Children's MEG Facility

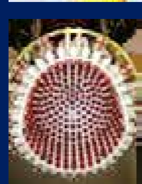
Based on A2.07

Boston Children's MEG Facility

Based on 2010.02.27

BabyMEG: whole-head 384-ch pediatric MEG system





- •2-layer sensor array
- •270 channels/inner
- channels/outer
- 9 reference channels
- 7-8 mm gap
- Helmet – up to 3-4 yrs
- 100% helium recycler

Okada 2/5/2015

BabyMEG sensors are closer to the scalp than VectorView

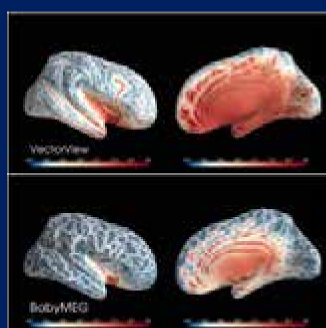
Band	Magnetometers	Gradiometers
1 Hz - 40 Hz	5.31 fT/Hz$^{1/2}$	395 fT/cmHz$^{1/2}$
45 Hz - 115 Hz	3.20 fT/Hz$^{1/2}$	276 fT/cmHz$^{1/2}$

Band	Inner Layer	Outer Layer
1 Hz - 40 Hz	12.7 fT/Hz$^{1/2}$	5.31 fT/Hz$^{1/2}$
45 Hz - 115 Hz	7.47 fT/Hz$^{1/2}$	3.20 fT/Hz$^{1/2}$

Luessi, Hamalainen and Okada, 2013

Okada 2/2015

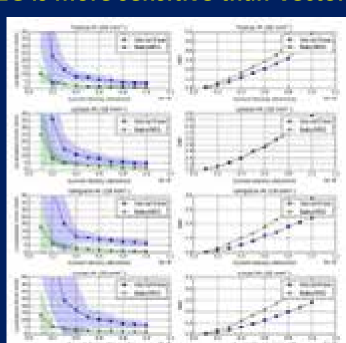
BabyMEG is more sensitive than VectorView



Luessi et al 2013

Okada 2/2015

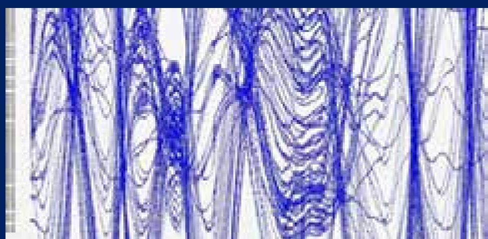
BabyMEG is more sensitive than VectorView



Luessi et al 2013

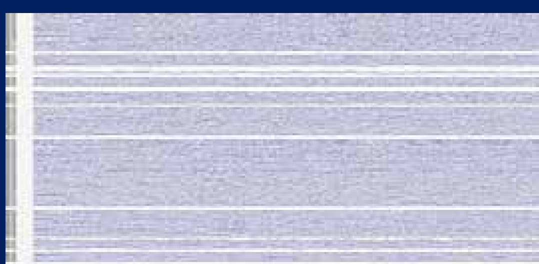
Okada 2/5/2015

Rejection of environmental noise with signal space projection



Hamalainen and Okada

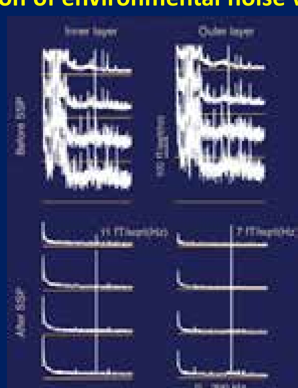
Rejection of environmental noise with SSP



Same gain before and after SSP application

Hamalainen and Okada

Rejection of environmental noise with SSP



Hamalainen

Status Report as of 1/23/2015

Hardware

•100% magnetometers
•97% of the channels work (369 out of 384 channel)
•Magnetometers operate without saturation in the MSR (with a shielding factor comparable to the 1-layer Elekta MSR)
•System noise with gain of 100x:
  •~5 fT/√Hz (inner); ~3 fT/√Hz (outer), to be confirmed
  •11 and 7 fT/√Hz noise in the preliminary data were obtained with gain of 1x.

•Closed-loop helium recycler has been running since 12/2/2014
•Recycler update in 2/2015 with a more powerful cryocooler

Okada 2/5/2015

Status Report as of 1/23/2015

Software (Hamalainen et al)

•BabyMEG software runs on a new device-independent platform – MNE-X
•Real-time MEG (rt-MEG) software, open-source, is being implemented
•Calibration software for gain, position and orientation of magnetometers
•Head digitization software available
•Real-time head position monitor and motion correction (beta version)
•On-line SSP for real-time noise rejection nearly ready
•Online normalized averaging available for >1 event
•Real-time current source imaging software available (beta testing)
•Functional connectivity analysis software in MNE exported to MNE-X

Okada 2/5/2015

Predicted performance

Real-time MEG capabilities
Basic
•Single-trial detection of evoked sensory responses
•Detailed analysis of cortical organization

Clinical
•High-resolution imaging of epileptiform activity
•High-resolution imaging of eloquent areas for neurosurgery
•Possible applications in rehabilitation
•Analysis of functional abnormality in connectivity in ASD

Okada 2/5/2015

Acknowledgements

NSF grant 0958669 from Major Research Instrumentation Program
Donation from Boston Investment Conference
Financial support from Boston Children's Hospital
Financial support from CH Neurology Foundation

Okada 2/5/2015

Neural Synchrony Examined with MEG During Eye Gaze Processing in Autism Spectrum Disorder

Renee Lajiness-O'Neill, MD

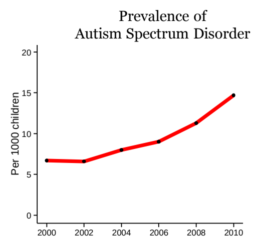
Gaze processing deficits are a seminal, early, and enduring behavioral deficit in autism spectrum disorder (ASD); however, a comprehensive characterization of the neural processes mediating abnormal gaze processing in ASD has yet to be conducted. This study investigated whole-brain patterns of neural synchrony during passive viewing of direct and averted eye gaze in ASD adolescents and young adults (MAge = 16.6) compared to neurotypicals (NT) (MAge = 17.5) while undergoing magnetoencephalography. Coherence between each pair of 54 brain regions within each of three frequency bands (low frequency (0 to 15 Hz), beta (15 to 30 Hz), and low gamma (30 to 45 Hz)) was calculated. Significantly higher coherence and synchronization in posterior brain regions (temporo-parietal-occipital) across all frequencies was evident in ASD, particularly within the low 0 to 15 Hz frequency range. Higher coherence in fronto-temporo-parietal regions was noted in NT. A significantly higher number of low frequency cross-hemispheric synchronous connections and a near absence of right intra-hemispheric coherence in the beta frequency band were noted in ASD. Significantly higher low frequency coherent activity in bilateral temporo-parieto-occipital cortical regions and higher gamma band coherence in right temporo-parieto-occipital brain regions during averted gaze was related to more severe symptomology as reported on the Autism Diagnostic Interview-Revised (ADI-R). The preliminary results suggest a pattern of aberrant connectivity that includes higher low frequency synchronization in posterior cortical regions, lack of long-range right hemispheric beta and gamma coherence, and decreased coherence in fronto-temporo-parietal regions necessary for orienting to shifts in eye gaze in ASD; a critical behavior essential for social communication.

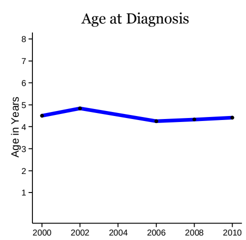
Neural Synchrony Examined with MEG During Eye Gaze Processing in Autism Spectrum Disorder

RENEE LAJINESS-O'NEILL
DEPARTMENT OF PSYCHOLOGY
EASTERN MICHIGAN UNIVERSITY

CENTER FOR HUMAN GROWTH & DEVELOPMENT
UNIVERSITY OF MICHIGAN

Prevalence Rates Compared to Age at Diagnosis





What to Study?

- Gaze processing deficits – A seminal, early and enduring behavioral deficit in autism spectrum disorder (ASD).
- A comprehensive characterization of the neural processes mediating abnormal gaze processing in ASD has not been conducted.
- Exploring whole-brain patterns of **neural synchrony** during gaze processing has the potential to illuminate a biomarker prior to the onset of the behavioral symptoms, leading to earlier identification.

Typically developing 16 month old

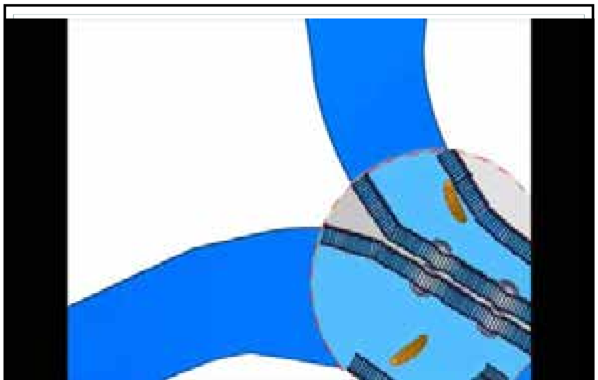
Autism Speaks Video Glossary

Features of Autism Spectrum Disorder

Autism Speaks Video Glossary

Gaze Processing
Potential Endophenotypic Candidate

- Given its central phenotypic prominence in ASD, **gaze processing** clearly surfaces as a strong and enduring **endophenotypic candidate**.
- To more broadly understand the relationship between a core and enduring behavioral deficit in ASD and its neuropathology, we hypothesized *a priori* that processing eye gaze information was likely to more precisely **characterize aberrant beta and gamma band oscillatory activity** and potentially aberrant connectivity.



Neural Synchrony
The Mind Project, Auth. Robert Shufflebeam

Methods

- Whole-brain patterns of neural synchrony during direct and averted gaze while undergoing MEG.
- Coherence between each pair of 54 brain regions (27 in each hemisphere).
- Power spectra for activity at all active sites were also calculated and used to quantify differences in low frequency, beta, and gamma power.
  - Low frequency (0-15 Hz)
  - Beta (~15-30 Hz)
  - Low Gamma (~30-45 Hz)

Participants

- 10 ASD (Mean Age 16.6) and 8 NT (Mean Age 17.5) adolescents and young adults.
- The groups did not differ significantly in age ($U(16) = 1.79$, $P = 0.76$), gender ($\chi^2 = 0.11$), or Full Scale IQ, with both groups generally performing in the Average to Above Average range on the Wechsler Abbreviated Scale of Intelligence (WASI; $U(16) = 40$, $P = 0.74$).

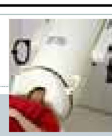
Participants (Cont.)

- Recruited from and underwent MEG procedures at Henry Ford Hospital (HFH), Department of Neurology, Neuromagnetism Lab.
- Participants were diagnosed with Pervasive Developmental Disorder (PDD) (recently revised to ASD) based on the Diagnostic and Statistical Manual of Mental Disorders-Fourth Edition-Text Revision (DSM-IV-TR) diagnostic criteria. Diagnoses were confirmed with the Autism Diagnostic Interview-Revised (ADI-R).

Autism Diagnostic Inventory-Revised

- Means and standard deviations for the ADI-R domain scores include: M_{Social} (SD) = 19.25 (4.59);
- $M_{Communication}$ (SD) = 15.50 (4.34);
- $M_{Repetitive}$ (SD) = 6.63 (1.69).

MEG Data Acquisition



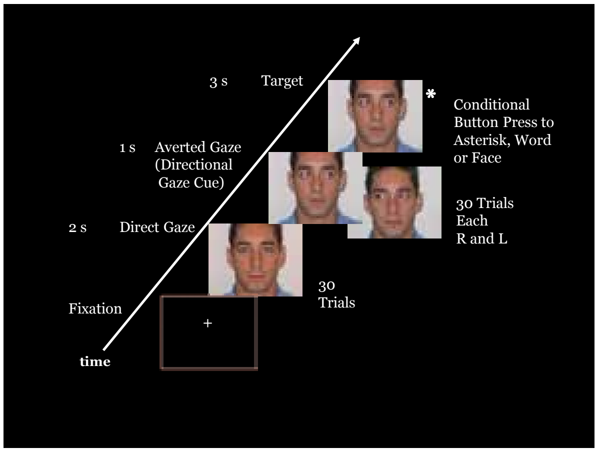
- 148 channel whole head MEG system (4D Neuroimaging, Magnes WH2500) with magnetometer type sensors
- Band-pass filtered 0.1 to 100 Hz
- Sampled at 508.63 Hz
- Timing recorded as pulse codes on a trigger channel

Post-processing

- Post-processing – noise artifacts removed using independent component analysis (ICA)
- Data were band-pass filtered 1 to 50Hz
- 2-s epochs of MEG data – average evoked responses for stimuli requiring conditional button press (asterisk, face, or word).
- Epochs had baseline of 500 ms before stimuli onset to 1,500 ms after stimuli onset.

Gaze Cueing Paradigm

- 14-min trials
- 5 conditions – direct gaze, averted gaze, and gaze cues to peripheral stimuli (asterisk, word, face).
- Direct and averted conditions – subject passively viewed
  - Conditional button press required to gaze cues to peripheral stimuli
  - Central character engaged in random gaze shift to R or L for 1 s
  - A target appeared for 3 s
  - Sixty targets in each gaze cueing condition – 30 congruent, 30 incongruent
- Goal for peripheral conditions was to measure voluntary shifts to eye gaze rather than reflexive responses



MEG Coherence Analysis

- Synchronization of neuronal activity was quantified by calculating coherence between cortical sites.
- The MRI was segmented and the brain surface was represented by a cortical model of approximately 4,000 dipoles each having an x, y, and z orientation at each site.
- Sites were distributed to represent the same volume of cortical gray matter. This model was then morphed to fit the digitized head shape collected during the MEG acquisition.
- To calculate coherence, the MEG data were first divided into 80 segments each containing 7.5-s segments of data and cortical activity in each segment was imaged on to the MRI using the MR-FOCUSS imaging technique.
- Using the time sequence of imaged activity, coherence between active cortical model sites was calculated for each data segment and then averaged for the completed study.
- Connectivity was quantified by a histogram of the number of sites to which the site had the same level of coherence. Statistical analysis of cortical coherence levels (0 to 1) were used to quantify differences in network connectivity between groups.

MEG Coherence Analysis

- Power spectra for activity at all active sites were also calculated and used to quantify differences in low frequency, beta, and gamma power.
- For the 'lower' frequency band, delta, theta, and alpha bands were collapsed for comparison with the alert working brain, specifically beta and gamma frequencies.
- A region-of-interest (ROI) tool implemented in MEG Tools was used to identify 54 regions in the brain (27 in each hemisphere).

Group Differences

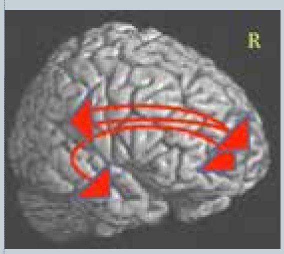
- For each frequency band (low, beta, and gamma) within each condition (direct and averted gaze), a *t*-test was conducted to assess group difference in average coherence values for each pair of brain regions (N = 1,431).
- False discovery rate (FDR) was used to adjust for multiple testing. The Benjamini-Hochberg algorithm was used to control the FDR at 0.10. From each *t*-test, a *z*-score was computed according to the method of Efron to summarize the difference in coherence values between ASD and NT. Positive *z*-scores indicate higher coherence in the ASD group.
- A series of chi-squares were computed to determine if the number of intra-hemispheric and inter-hemispheric cortical differences within the low (0 to 15 Hz), beta (15 to 30 Hz), and low gamma (30 to 45 Hz) frequency bands were statistically different between the groups.
- Kendall Tau correlation coefficients were computed to examine relationships between ASD clinical symptoms as reported on the ADI-R and neural oscillatory activity (coherence).

Results – Behavioral Data

- No significant between group differences noted in error rates in responding to the conditional button press during the intervening task condition with respect to accuracy ($t(16) = 0.70$ $P = 0.51$), suggesting that both groups were equally engaged in the task.
- Reaction times were not statistically different ($t(16) = -0.11$ $P = 0.92$).

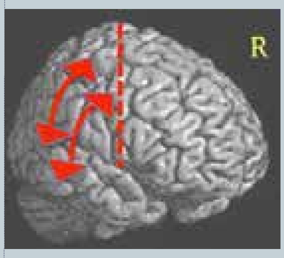
Coherence Imaging-DIRECT GAZE

- 91 of the 1,431 pathways were found to be significantly different between the groups
- **NT** - higher coherence was observed between **frontal, temporal, and parietal regions**. Higher coherence was particularly evident ***between bilateral frontal (middle, inferior, and orbitofrontal) gyri and right superior temporal, pre- and postcentral gyri***.



Coherence Imaging-DIRECT GAZE

- ASD - **higher coherence** was noted **between left occipito-parietal** (angular, middle, and superior occipital gyri) **and bilateral occipito-parietal regions** (inferior, middle, superior occipital gyri, and supramarginal regions).

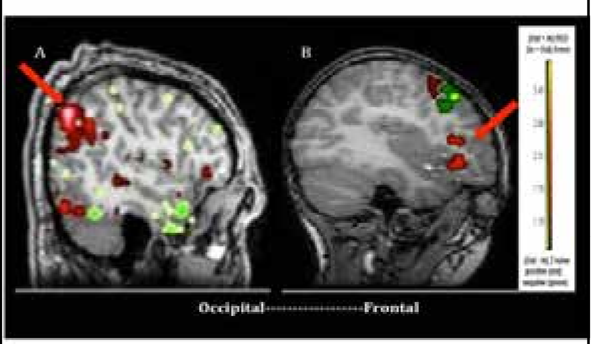


Coherence Imaging-AVERTED GAZE

- 390 of the 1,431 pathways were found to be significantly different between the groups
- **NT** demonstrated significantly **higher coherent activity across all frequencies** between **bilateral frontal (inferior, middle, superior, orbitofrontal gyri) and right frontal (inferior, middle, superior, and precentral gyri), superior temporal, and parietal (postcentral gyrus) regions**, consistent with known neuroanatomical substrates critical for responding to shifts in eye gaze

Coherence Imaging-AVERTED GAZE

- **ASD** participants displayed **higher coherence between left parieto-occipital (angular, inferior, and middle occipital) and bilateral temporo-parieto-occipital regions (inferior, middle, superior temporal, occipital, angular gyri).**



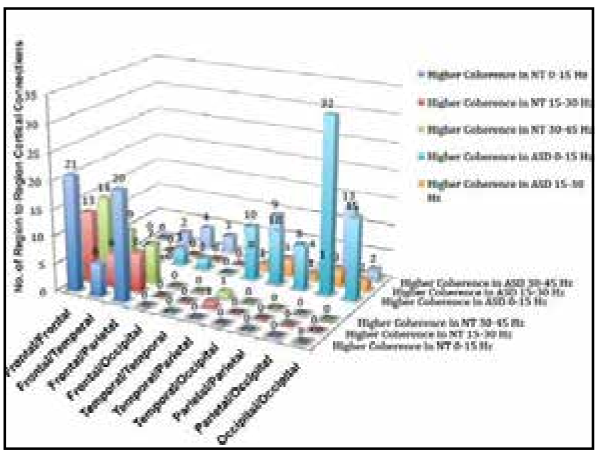
Subset of regions of network of greatest average coherence in a representative ASD and NT participant

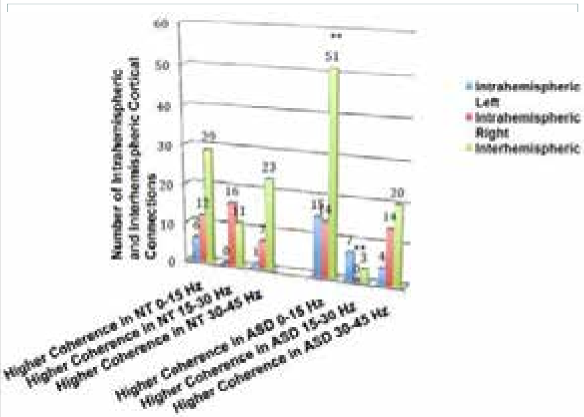
Group Differences within Frequencies during Averted Gaze Conditions

- Of the 390 pathways, significant differences in coherence were found within specific frequencies in 233 of the pathways
- 127 in the low frequency band (0-15 Hz)
- 37 in the beta frequency band (15-30 Hz)
- 69 in the low gamma frequency band (30-45 Hz)

Frequency Band Differences

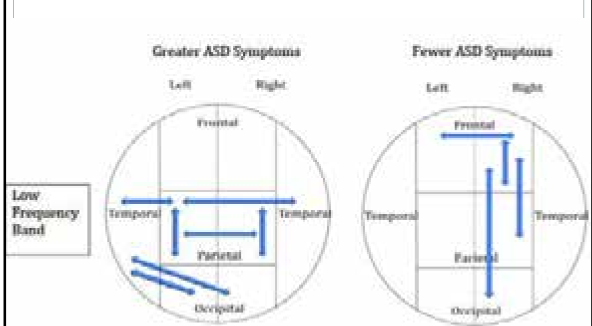
- Low frequency band - **ASD** participants displayed **higher coherent activity between left parieto-occipital regions and right temporo-parieto-occipital regions** and significantly **lower coherence between bilateral frontal and right fronto-temporo-parietal regions.**
- Beta band - **ASD** participants demonstrated **higher coherence between left parieto-occipital regions and bilateral temporo-occipital and left parietal regions.**
- Gamma Band - **ASD** participants **showed higher coherence between bilateral temporo-parieto-occipital regions as well as bilateral parietal and orbitofrontal regions.**
- Beta and Gamma - **ASD** participants **showed lower coherence between bilateral frontal, fronto-temporal, and temporo-parietal regions** compared to NT
- NT subjects displayed significantly higher coherence between bilateral frontal, fronto-temporal, and fronto-parietal regions across all frequency bands.

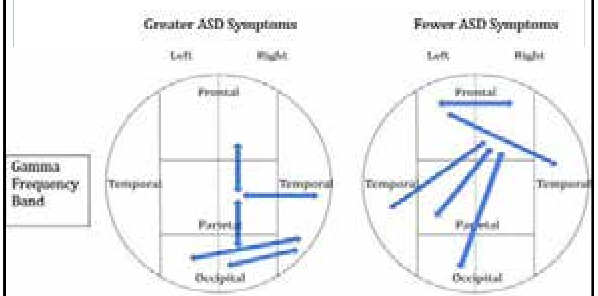


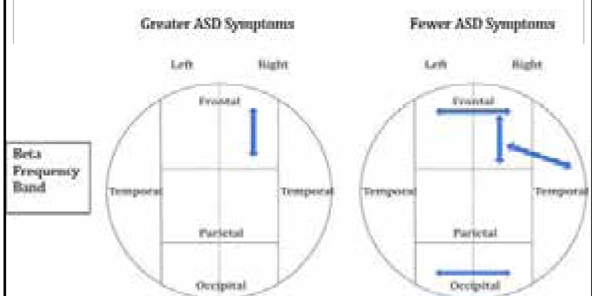


Relationship Between Oscillatory Activity and Clinical Symptomatology

- This preliminary examination of the relationship between neural coherence and ASD symptomatology further revealed a unique pattern of relatively mutually exclusive findings…







Discussion

- One of the hallmarks of ASD is a failure to detect and/or respond in a typical manner to information conveyed by eye gaze.
- Our study characterized whole-brain patterns of synchrony in ASD compared to NT during direct and averted eye gaze processing while undergoing MEG.

Results revealed:

- (1) higher coherence and synchronization in temporo-parieto-occipital brain regions across all frequencies in ASD, particularly within the low frequency range;
- (2) a higher number of low frequency cross-hemispheric coherent connections; and
- (3) a near absence of right intra-hemispheric synchrony in the beta frequency band in ASD.

- Finally, ASD participants demonstrated higher gamma power in inter-hemispheric connections between the left and right parietal lobes and intra-hemispherically between the right parietal lobe (particularly angular gyrus) and temporal regions.

Discussion

- Angular gyrus has been implicated in a number of processes including reading and number comprehension, numerical processing, visual attention, and social cognition.
- It is a cross-modal region where sensory information from the visual, auditory, and tactile senses converge allowing for a combined and integrated percept. It is essential for the manipulation of mental representations and reorienting of attention.
- Heightened connectivity in these regions without appropriate regulatory or contextual feedback from frontal regions may result in exquisite sensory sensitivity, acceleration of letter, number, and word recognition with limited comprehension or applied skills, or an over-allocation of attention to information without a clear appreciation of its relevance; a neurocognitive pattern often noted in ASD.
- A strengthening of connectivity between the right angular gyrus and inferior temporal regions without frontal mediation, particularly from medial prefrontal regions, may further contribute to a heightened attention to the invariant features of the face or its components without an ability to extract essential social relevance.

- Our method allows us to provide a direct numerical comparison between pathways, both inter- and intra-hemispherically, and to examine group differences between frequency bands known to underlie short- and long-range connectivity.
- This altered pattern of oscillatory activity may contribute to aberrant connectivity that underlies the failure of individuals with ASD to appropriately orient to eye gaze, which has a cascading negative effect on typical social and language development.

Next Steps

Biological Markers — Can we use patterns of neural synchrony to diagnose Autism Spectrum Disorder in pre-linguistic infants?

Cognitive Mechanisms — Why do observed neural synchrony disruptions impair social communication?

Effective Interventions — How can we measure the efficacy of intervention through alterations in neural synchrony?

Acknowledgments

- Henry Ford Health System
  - Susan Bowyer, Ph.D.
  - John Moran, Ph.D.
  - Norman Tepley, Ph.D.
- Research supported by
  - New Faculty Award –EMU (RLO)
  - Faculty Research Fellowship-EMU (RLO)
- Eastern Michigan University
  - Annette Richard
  - Lesley Pawluk
  - Daniel Jacobson
  - Amy Olszewski
  - Kelly Luce
  - Alfred Mansour, Ph.D.
  - Adam Chase
  - Mellisa Boyle
  - Aimee Moore
  - Natalie Morris
  - Jeff Titus, Ph.D.
  - Nick Velissaris

Abnormal MEG Coherence Imaging in Panic Disorder

Nash Boutros, MD

Increased Coherence Imaging (CI) values, as determined by magnetoencephalography (MEG), are indicative of increased neural excitability. The purpose of this investigation was to examine the CI values in patients with panic disorder (PD). We also ascertained if regions with increased coherence had higher representation in the limbic fronto-temporal regions (LFTRs). The highest CI values and their locations were determined in six PD patients and six age-matched healthy control subjects (from archives). MEG scans were acquired with 148 magnetometer channels and 32 channels of simultaneous EEG. Despite the small sample size, CI values were significantly higher in PD patients. Brain regions with increased coherence in the PD patients were significantly more in areas typically associated with the LFTRs when compared to the control subjects. The above data suggest that coherence values may be increased in the LFTRs of patients with PD. Recent advances in epilepsy research suggest that increased coherence may reflect increased excitability in these brain regions. Based on the data provided here as well as available literature, we propose that additional research examining coherence values in LFTRs of PD patients could inform the choice of medications in this patient population with increased coherence (i.e., increased excitability) being a biomarker for favorable response to anti-seizure drugs. PD is discussed as a prototype for epilepsy spectrum disorders (ESD).

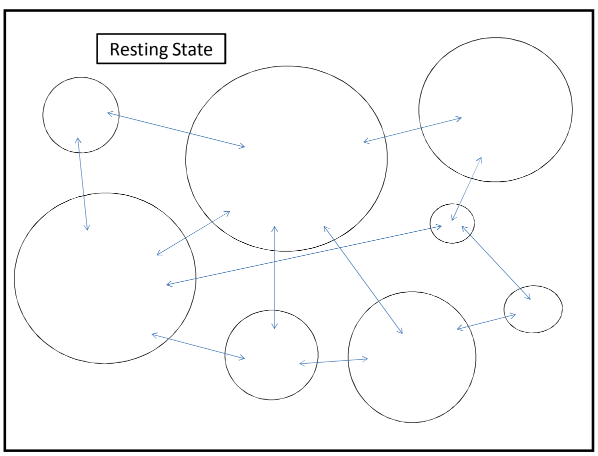
Abnormal MEG Coherence Imaging in Panic Disorder

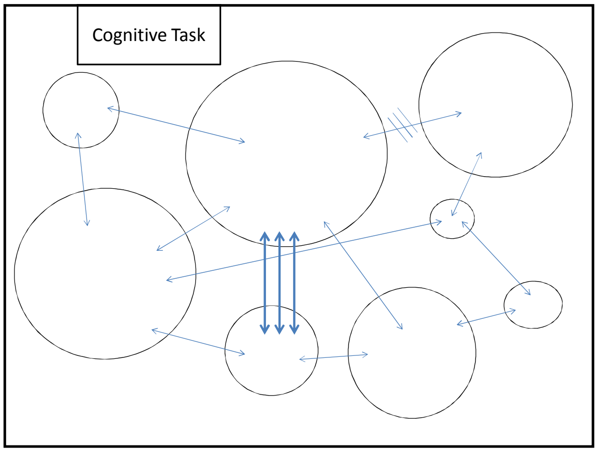
EPILEPSY SPECTRUM DISORDERS

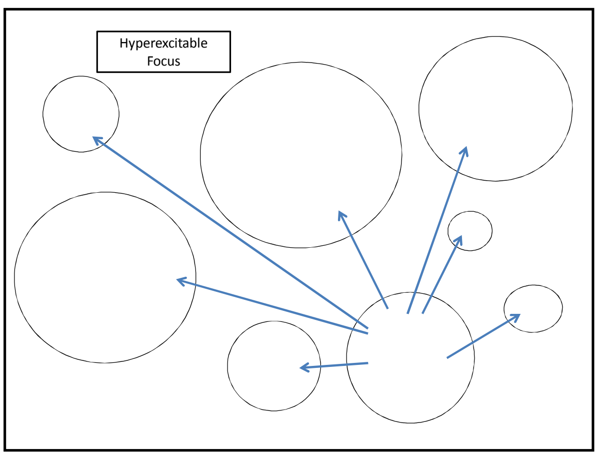
NASH BOUTROS
UNIVERSITY OF MISSOURI
KANSAS CITY (UMKC)
DEPARTMENT OF PSYCHIATRY

Conflict of Interest

- NONE




Cognitive Task


Hyperexcitable Focus


Prefrontal cortex
Orbital cortex
Mediodorsal thalamic nucleus
Olfactory bulb
Ventral striatum
Cingulate gyrus
Hypothalamus
Medial nuclei
Lateral nuclei
Central nucleus
Pain, taste
External and internal sensory signals
Brainstem
PAG
Autonomic motor nuclei
Uni- and multimodal sensory areas and insular cortex
Brainstem
Autonomic sensory nuclei

A seriously under-examined phenomenon

,

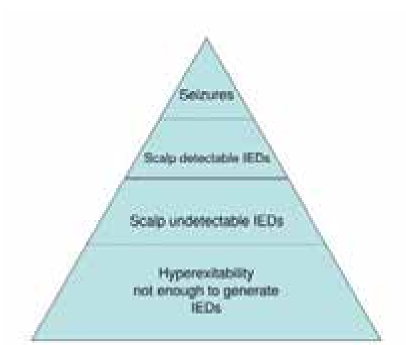
While there has always been an undercurrent of opinion simmering just below the level of verification that paroxysmal EEG discharges without overt seizures may have behavioral consequences such as emotional lability, irritability, or temper dyscontrol,

The major dictum REMAINS NOT to Treat EEGs.

Some History

- Subictal Neurosis — Jonas, 1965
- Episodic Dyscontrol — Monroe, 1970
- Variants of CPS — Tucker et al, 1986
- Subclinial Seizures — Sperling & O'Connor, 1990
- Post-traumatic temporal lobe dysfunction — Zappala & Cameron, 1990
- Atypical Psychosis — Neppe 1991
- Epilepsy Spectrum Disorders — Springer et al, 1991
- ESD — Roberts et al, 1992
- Multiple partial seizure-like symptoms without stereotyped spells — Verduyn et al, 1992

TIP OF THE ICEBERG



MEG and EEG may measure different spike populations

- MEG and EEG read separately in 19 patients with mTLE
- 14 had no spikes
- 50-80% more MEG spikes than EEG spikes

Zijlmans et al, 2002

Panic Disorder

- Panic symptoms carry a significant resemblance to symptoms induced by temporo-limbic epileptic activity particularly those originating from the Sylvian fissure. Fear, derealization, tachycardia, diaphoresis, and abdominal discomfort are characteristic symptoms of simple partial seizures with psychiatric and autonomic symptomatology.

Pathophysiology Hypotheses

Many theories:

- Noradrenergic, Serotonergic, GABAergic.
- Genetic-environment.
- Hyperventilation theory.
- Many psychological theories (dynamic-Cog/Beh).
- NEUROANATOMICAL: Gorman et al, 1989/2000. Panic originates from an abnormally sensitive fear network that involves the prefrontal cortex, insula, thalamus, amygdala and its projections to brain stem/hypothalamus and hippocampus.

Could the physiological changes induce panic in otherwise healthy limbic frontal structures?

- Answer is most probably NO.
- Gutman et al (2005). Only panic patients developed panic when the physiological changes were induced.

Response to AEDs

- Ten reports were identified for use of AEDs in PD patients with abnormal EEGs.
- Seven of the ten reports were single-case reports and three included case series.
- No controlled studies were found.
- A total of 20 patients were included in the ten papers. Of the 20 patients 17 responded well to an AED.

Response to AEDs

- Most reports did not specify the side, location or severity/frequency of the abnormalities.
- The side of the abnormality was reported in only three cases and in all three the abnormality was left sided. Furthermore, only one report specified the location of the abnormality on the temporal lobe (anterior, middle or posterior) which was anterior temporal.
- While a number of AED agents were used, carbamazepine was the most frequently utilized (7 of the 10 reports).

AEDs in unselected PD patients

*18 reports were identified. Four reports were single-case and eleven were case series ranging from 4 to 47 patients.

*Three reports described controlled studies. The three controlled studies were double-blind and randomized, one was placebo controlled, one was placebo controlled in a cross-over design and the third was placebo-controlled in a parallel-groups design.

*A total of 253 patients were included, and treated with an AED. Based on material provided in the reports, 137 of the 253 responded well (at least 50% decrease of panic attack frequency and severity).

AEDs in unselected PD patients

*These studies had a predominance of reports utilizing valproic acid (VPA) (8 studies), while carbamazepine was used in three, tiagabine and gabapentin in two studies each, and one study each for leveteracetam, and lamotrigin.

From reports of PD patients with abnormal EEG's, 17 patients were reported as responders from a total of 20. In unselected patients 137 were reported as responders from a total of 253.

The null hypothesis of no difference between these two groups is rejected at a two tailed α=0.05 with a p-value=0.011.

Do all AEDS work

- Of significant interest are three reports of an AED inducing Panic attacks.
- Clivas et al, (2008) reported a 17 y/o female with diagnosis of borderline personality who developed panic symptoms when placed on relatively low dose of topiramate (25mg).
- Two prior reports described similar cases but with much higher doses of 150mg (Damsa et al, 2006) and 100 mg (Goldberg, 2001).

- Clivaz E, et al. Topiramate and panic attacks in patients with borderline personality disorder. Pharmacopsychiatry. 2008; 41(2):79.
- Damsa C, et al. Panic attacks associated with topiramate. J Clin Psychiatry. 2006; 67:326-327.
- Goldberg JF. Panic attacks associated with use of topiramate. J Clin Psychopharmacol. 2001; 21:461-462.

Coherence Imaging

Hyperexcitable brain tissue is the characteristic of epileptogenic brain foci and recent advances from epilepsy research point to abnormally increased localized coherence within epileptic zones [Elisevich & Bowyer et al, 2011].

Coherence is a measure of synchrony between signals from different regions of the brain.

The Epilepsy Group at HF recently developed and tested a new methodology "Coherence Imaging" to assess neural coherence within neural tissue (as contrasted to coherence assessed at surface electrode recording locations)[Moran & Bowyer et al, 2008; Elisevich & Bowyer et al, 2011].

Schevon et al [2007] found the high coherence regions adjacent to the epileptic foci and that surgical resection of these areas decreased post-operative seizures.

Elisevich K, et al. An assessment of MEG coherence imaging in the study of temporal lobe epilepsy. Epilepsia. 2011;52: 1110-1114

Moran JE. MEG Tools available at www.megimaging.com. Detroit: MEG signal processing, data visualization, MRI integration, Brain imaging and visualization. 2008

Schevon C A, et al. NeuroImage. 2007:35: 140-148

Pilot Data

The purpose of this investigation was to examine the Coherence Imaging values in patients suffering from PD and if regions with increased coherence had higher representation in the limbic fronto-temporal regions (LFTRs).

The highest Coherence Imaging values and their locations, among 54 Brodmann areas, were determined in six PD patients and six age-matched healthy control subjects. MEG scans were acquired with 148 magnetometer channels and 32 channels of simultaneous EEG.

Despite the small sample size, Coherence Imaging values were significantly higher in PD patients.

Brain regions with increased coherence in the PD patients were significantly more in areas typically associated with the LFTRs when compared to the control subjects.

Based on these data as well as available literature, we propose that additional research examining coherence values in LFTRs of PD patients could inform the choice of medications with increased coherence (i.e., increased excitability) being a biomarker for favorable response to medications that limit excitatory transmission, namely benzodiazepines or anti-seizure drugs.

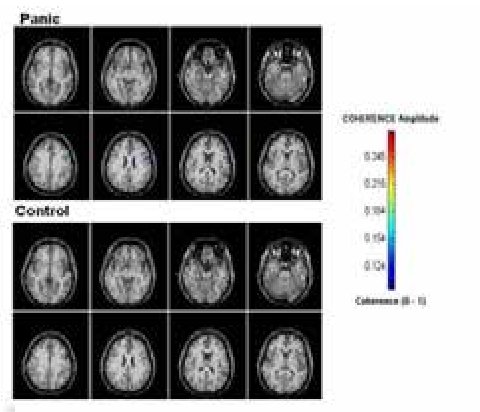
Boutros et al,. In Press, NeuroReport.

Coherence and Treatment Response

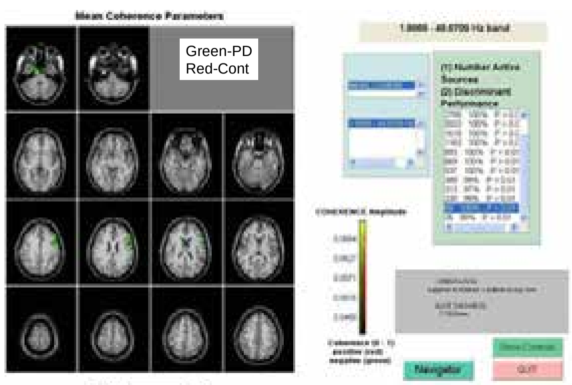
Of the six patients examined two reported being SSRI/SNRI responders and exhibited the least coherence values, two reported needing either an AED or clonazepam to bring the panic attacks under control and had the highest coherence values.

The last two had intermediary coherence values and needed a combination of both types of medications to achieve clinical control.

This VERY limited data set supports the predicted direction of patients exhibiting high coherence-imaging values in LFTRs being SSRI/SNRI non-responders.



Group difference



Flowchart to be tested

Proposed Diagnostic Criteria for ESD

A: Probable ESD: Presence of any form of episodic symptoms. This can include but not limited to: panic and other dissociative attacks, episodic violence;

- And/or 2) Scores 44 or higher on the SCIPS. This criterion allows the consideration of ESD even if episodic symptoms are not prominent like in conditions of Autism Spectrum and some cases of mood disorders.

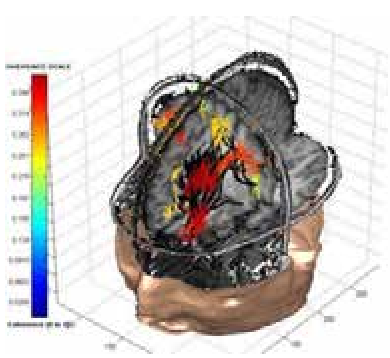
B: Possible ESD: 1) Evidence from EEG or MEG for paroxysmal discharges.

- and/or 2) Evidence of localized increased coherence (MEG).
- and/or 3) Evidence from PET scanning for a hypometabolic focus suggestive of an epileptic process.
- and/or 4) Neuropsychology testing suggestive of focal deficit in a brain region relevant to the clinical presentation.
- It is likely and testable that the presence of more than one of these criteria increases the likelihood that in fact the condition under investigation is an ESD.

C) Definite: A significant clinical response to anticonvulsant medications when the above criteria are present.

THANK YOU

Patient Panic patient Brain activity during panic attack



ACMEGS
AMERICAN CLINICAL MEG SOCIETY

THE MEG SLOW DIMENSIONS: SIFTING FACTS FROM ARTIFACT

Ernst Rodin, MD

With the help of friends and colleagues I have been investigating during the past 11 years EEG infraslow activity (ISA, 0.1-0.01 Hz) from scalp and intracranial data. It could not only be established that the ictal onset baseline shifts do not require DC amplifiers for their demonstration but also that this frequency band is a normal component of the EEG spectrum and can provide additional localizing information in epilepsy patients. EEG data have, however, the potential disadvantage of contamination by electrode polarization and in case of scalp recordings also by skin potentials. These sources of possible artifacts do not apply to the MEG which made comparisons with scalp recorded EEGs important. Over the years, 89 MEG recordings from Dr. Michael Funke's laboratory in Salt Lake City (SLC), became available as well as 30 from Dr. Susan Bowyer at the Henry Ford Hospital (HFH) in Detroit. There was a difference between these two data sets inasmuch as the HFH data were obtained on a BTI system with DC amplifiers while the SLC data were recorded with a Neuromag/Elekta system and an input filter of around 0.01 Hz. Furthermore, the BTI system contained only magnetometers, while the Elekta system had planar gradiometers as well as magnetometers. In both data sets the EEG was co-registered; with 31 electrodes at HFH and 60 electrodes at SLC. This presentation will, however, only deal with MEG results. Data analysis was performed with the BESA software.Common to both data sets was the observation that activity below the delta frequency band (0.5-3 Hz) was clearly present in all individuals and in the SLC data set this included 15 normal persons who were engaged in a variety of cognitive tasks. In some instances a pronounced local or more widespread 0.2-0.3 Hz rhythm was noticed and identified as respiration artifact. Yet it could have clinical meaning. Since all the patients in the SLC data had been sedated with chloral hydrate and were asleep, sleep apneas could be observed in some instances. This activity could not be completely removed by limiting the frequency band to 0.01-0.1 Hz, or independent component analysis (ICA), and required Dr. Samu Taulu's method of Maxwell filtering (tsss) which was also used to remove other sources of external artifact. It could, however, only be applied to the Elekta data and frequently eliminated all activity below 0.1-0.5 Hz. The examples in this presentation were filtered by Dr. Taulu at 0.03 Hz. Six cases will be presented demonstrating the similarities and differences of the SLC and HFH data. It will become apparent that MEG slow wave activity should be included in the clinical evaluation of the recordings because it can provide information above and beyond spike localization. Frequencies below 0.01 Hz showed in the HFH data drifts in some channels which may or may not be artifactual. This determination will require special filters which were not available for this investigation. But since EEG near-DC activity can be demonstrated in records lasting an hour there is no reason why this should not also be present in the MEG. Since ISA and near-DC activity (<0.01 Hz) are currently largely neglected in cerebral electromagnetic research the audience is encouraged to investigate this frequency range for what it can tell us about normal and abnormal brain functions.

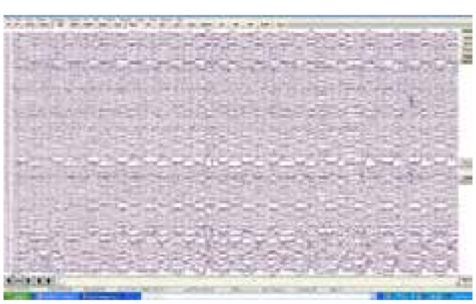
The MEG Slow Dimensions
Sifting fact from artifact

Ernst Rodin MD
Adj. Prof. Dept. of Neurology
University of Utah

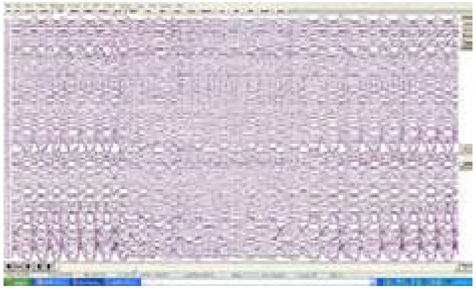
PURPOSE

- To demonstrate:
- that activity below the delta frequency band is available in archived routinely obtained clinical MEG recordings regardless whether the system is by Neuromag/Elekta or BTI.
- that the information contained can have clinical relevance and deserves intensive investigations from the clinical as well as basic science point of view.

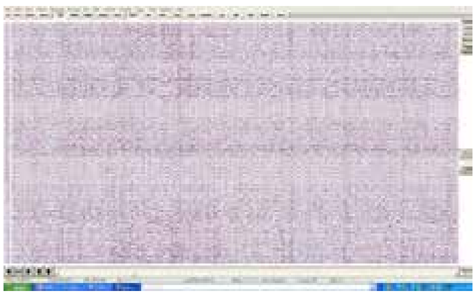
The phenomenon that started it. Elekta system 1 minute LF 0.5 Hz



Same file 80 seconds



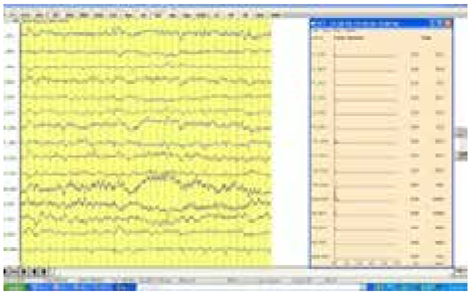
Same data tsss filtered



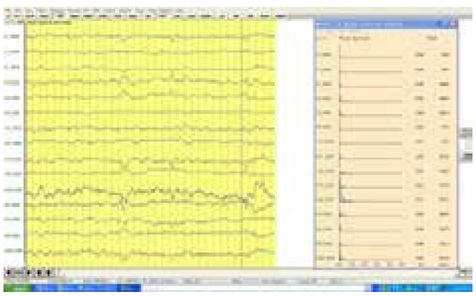
Patient A

- 14 year old female with complex partial seizures since age 9.
- Video EEG: ictal onset probably frontal, postictal left frontal slowing.
- SPECT: increased activity right frontal.
- MRI: nonspecific bifrontal white matter changes.
- MEG: right temporo-parietal slowing; spikes left cingulate gyrus some right sided.

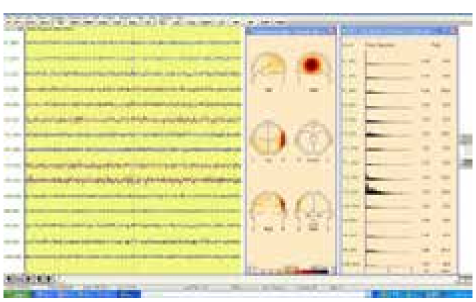
5 minutes empty room open filters gradiometers



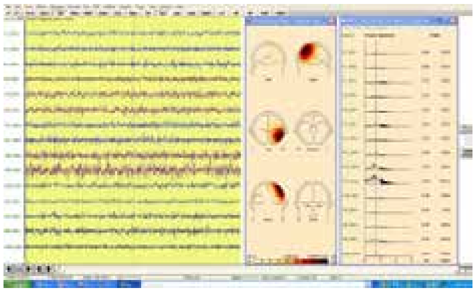
Empty room open filters magnetometers



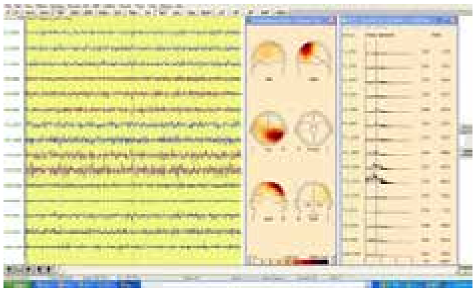
10 minutes; patient asleep 1-3 Hz



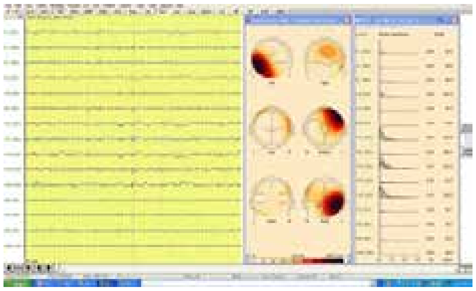
Open filters



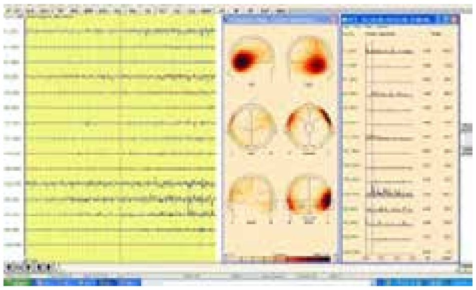
Open filters magnetometers



40 minutes later open filters



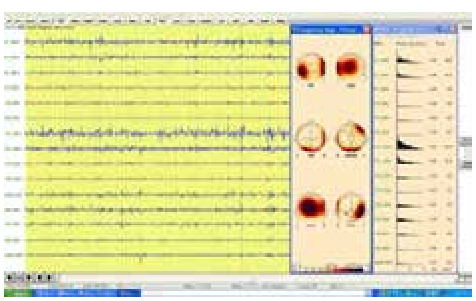
Postictal; filters open



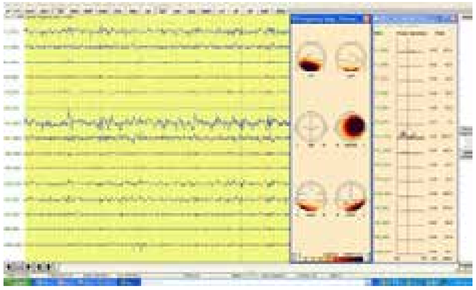
Patient B

- 14 year old male partial and secondary generalized seizures; developmental delay.
- Video EEG: left fronto-temporal abnormalities
- MRI: negative
- MEG: intermittent left and right temporal slowing. Spikes most frequently right basal temporal, less common left fronto-temporal.

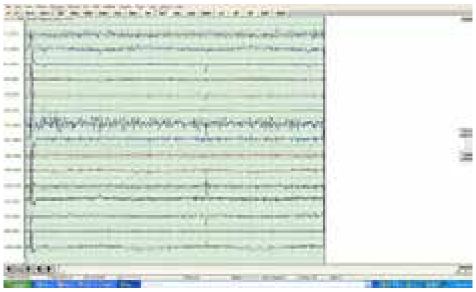
20 minutes 1-3 Hz



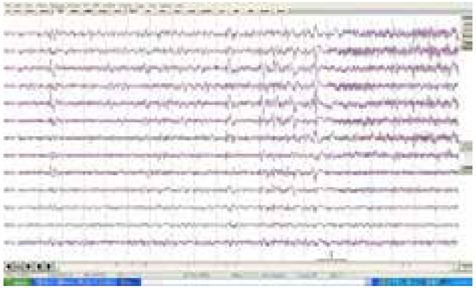
Same data open filters



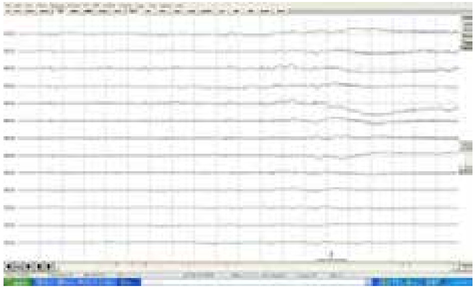
Total file 40 minutes open filters



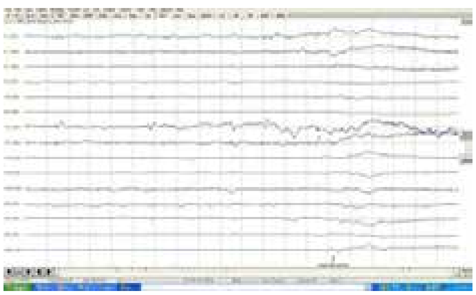
20 seconds ictal onset



Same data LF 0.01 Hz



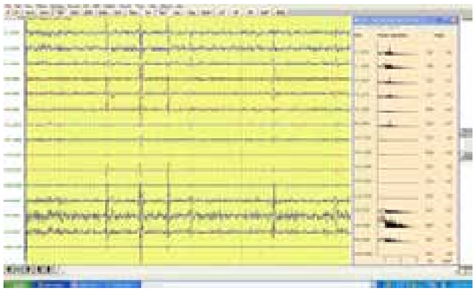
Same data source montage



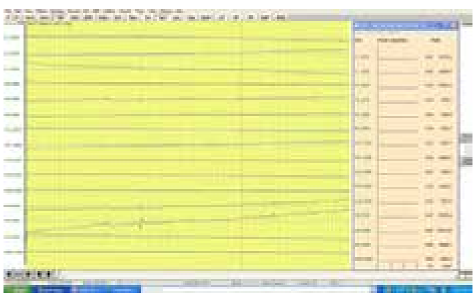
Conclusions for Elekta System

- Delta activity extends at least to 0.5 Hz and may, at times, represent filtered ISA.
- ISA down to 0.01 Hz is readily accessible from archived clinical recordings.
- Ictal onset baseline shifts, similar to what is seen in the EEG, can also be seen.
- Maxwell filtering (tsss) attenuates amplitudes, but does not distort locations. Care must be taken to set the parameters in a manner that leaves ISA intact.

HFH 15 minutes empty room BTI system 0.5-3 Hz



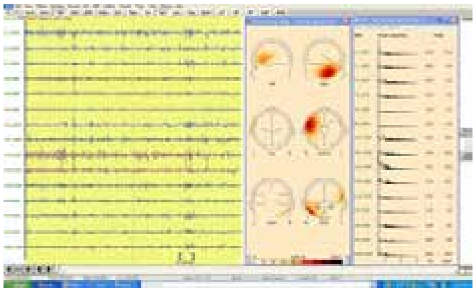
HFH empty room LF open



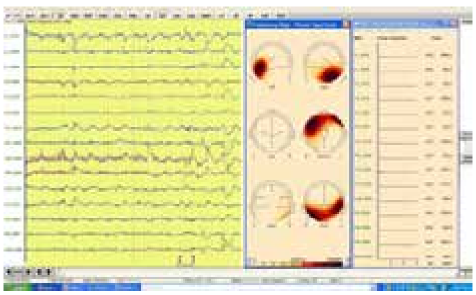
Patient 1

- 28 year old female with complex partial seizures diagnosed at age 6.
- EEG: mainly left anterior temporal spikes, also some left frontal and a few right temporal.
- MRI: left frontal cortical dysplasia
- PET: diffuse mild hypometabolism left fronto-temporo-parietal.
- MEG: multifocal spikes scattered throughout left hemisphere. Partial seizure left temporal.

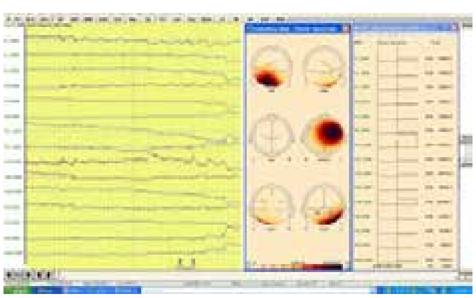
10 minutes 0.5-1 Hz



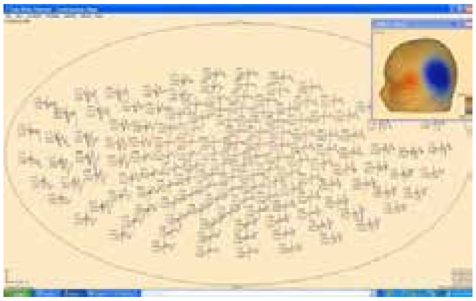
0.01-3 Hz



LF open HF 3 Hz



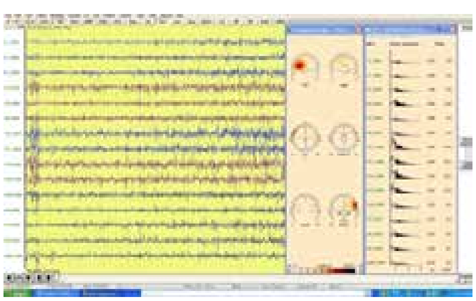
Ictal onset



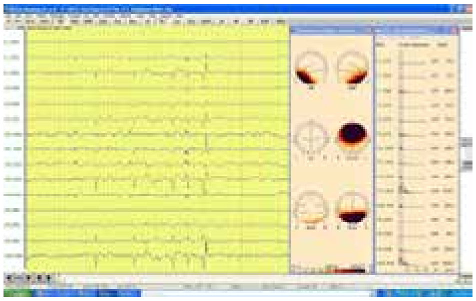
Patient 2

- Age 23
- Febrile seizures at 7 months. Complex partial seizures diagnosed at age 3.
- Clinically: speech arrest, unresponsive, lip smacking, right arm dystonic.
- EEG: left temporal sharp waves
- MRI: left MTS
- MEG: spikes and slow waves left anterior temporal

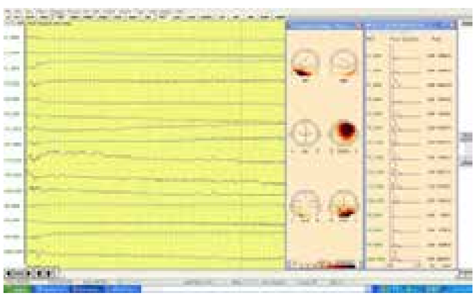
0.5-3 Hz



0.01-3 Hz



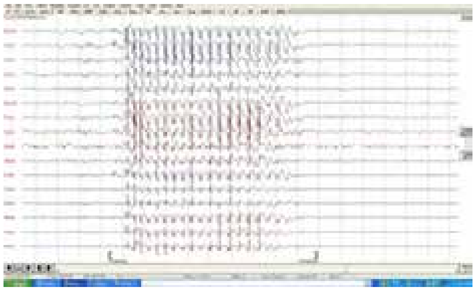
LF open HF 3 Hz



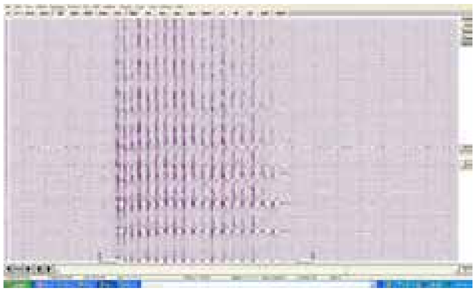
Patient 3

- 11 year old female with absence seizures and myoclonic jerks since age 4.
- Mother had one TC seizure at age 6 months, may have also had absences. Placed on Phenobarbital for 11 years with no further recurrences.
- EEG: typical generalized 3 Hz SW.
- MEG: generalized 3 Hz SW.

20 seconds EEG 1-40 Hz
ipsilateral ear reference

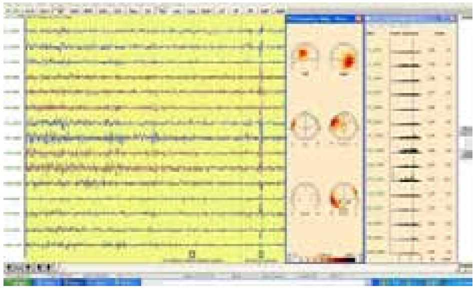


Same epoch MEG 1-40 Hz

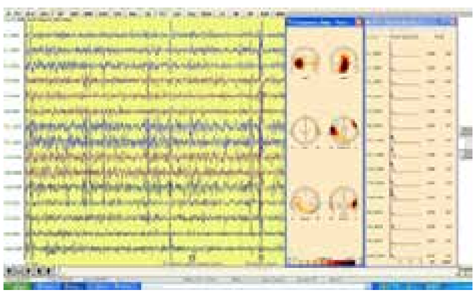


MEG onset 1-40 Hz

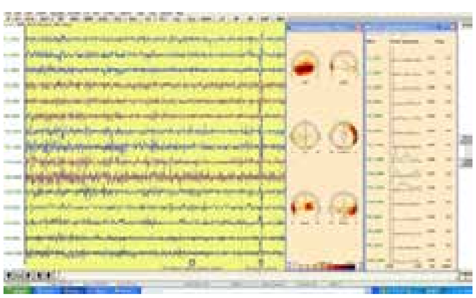
15 minutes 0.5-3 Hz



0.01-3 Hz



LF open HF 3 Hz



CONCLUSIONS

- The MEG, like the EEG, contains non-neuronal activity below the delta band. This has potential clinical relevance and should be intensively studied.
- Wave durations depend on amplifier characteristics, filter settings and file length.
- The Elekta system has a near-DC option and it is, therefore, recommended that it be used for routine data acquisition. The data can be subsequently filtered for clinical evaluations and research purposes. With long duration files and tsss filters it might allow the detection of a genuine near-DC component.

Acknowledgements

For the clinical data I am grateful to Dr. Michael Funke and his technologist, Miles Riley, at the University of Utah in Salt Lake City as well as Dr. Susan Bowyer and her technologist, Karen Mason, of Henry Ford Hospital in Detroit.

For technical assistance Drs. Michael Scherg and Harald Bornfleth of BESA software and Dr. Samu Taulu of the University of Washington for tsss filtering.

Without their help this study could not have been performed.

American Clinical Magnetoencephalography Society (ACMEGS)
Annual Business Meeting
Agenda

February 5, 2015
4:45 p.m.
JW Marriott Houston
Houston, Texas

I. Call to Order (Dr. Bagic)

II. Minutes of Previous Business Meetings (Dr. Bagic)

III. President's Report (Dr. Bagic)

IV. Financial Report (Dr. Bowyer)

V. Public Relations Report (Dr. Bowyer)

VI. ACMEGS Website (Dr. Ferrari)

VII. New Business

  a. Coverage Policies and Reimbursement (Dr. Bagic)

  b. Bylaws Revision (Dr. Funke)

  c. Elections (Dr. Bagic)

VIII. Transfer of Presidency

IX. Adjourn

President's Report

Anto Bagic, MD Pittsburgh, PA

ACMEGS
AMERICAN CLINICAL MEG SOCIETY

Financial Report
Susan Bowyer, Detroit, MI

ACMEGS
AMERICAN CLINICAL MEG SOCIETY

Public Relations Report

Susan Bowyer, Detroit, MI

ACEMGS Website Report

Paul Ferrari, Austin, TX

ACMEGS
AMERICAN CLINICAL MEG SOCIETY

New Business

Notes

Directions to ACMEGS Dinner at Mockingbird Bistro

mapquest

Trip to:

1985 Welch St

Houston, TX 77019-6121

4.56 miles / 10 minutes

JW Marriott Houston

View Our Deals & Packages

5150 Westheimer Road, Houston, TX 77056

(713) 961-1500

Download Free App

1. Start out going **west** on **Waterwall Dr / FM-1093**. Map	**0.09 Mi** *0.09 Mi Total*
2. Turn **right** onto **Sage Rd**. Map *Walgreens is on the corner* *If you reach Quarters Ct you've gone about 0.1 miles too far*	**0.7 Mi** *0.8 Mi Total*
3. Turn **right** onto **San Felipe St**. Map *San Felipe St is 0.1 miles past Champlain Bend St* *If you reach Huckleberry Cir you've gone a little too far*	**3.5 Mi** *4.3 Mi Total*
4. **San Felipe St** becomes **Vermont St**. Map	**0.2 Mi** *4.5 Mi Total*
5. Turn **right** onto **Hazard St**. Map *Hazard St is just past McDuffie St* *If you reach Driscoll St you've gone a little too far*	**0.07 Mi** *4.5 Mi Total*
6. Take the 1st **right** onto **Welch St**. Map *If you reach Indiana St you've gone a little too far*	**0.01 Mi** *4.6 Mi Total*
7. **1985 WELCH ST** is on the **left**. Map *If you reach McDuffie St you've gone a little too far*	

1985 Welch St, Houston, TX 77019-6121

Total Travel Estimate: 4.56 miles - about 10 minutes

AMERICAN CLINICAL MAGNETOENCEPHALOGRAPHY SOCIETY
2015 Annual Conference ♦ February 5, 2015

Evaluation Form Summary

Please identify yourself: ☐ Neurologist ☐ Neurosurgeon

☐ Radiologist ☐ MEG/EEG Technologist

☐ Other ______________________________________

Please rate each speaker's effectiveness in conveying the material of his/her presentation using 5 as most effective and 1 as least effective:

Faculty	Most Effective				Least Effective	Comments
Dr. Douglas Rose	5	4	3	2	1	
Dr. William Gaetz	5	4	3	2	1	
Dr. David Clark	5	4	3	2	1	
Dr. Elizabeth Pang	5	4	3	2	1	
Dr. Richard Bucholz	5	4	3	2	1	
Dr. Christos Papadelis	5	4	3	2	1	
Ms. Renee Lajiness-O'Neill	5	4	3	2	1	
Dr. Nash Boutros	5	4	3	2	1	
Dr. Ernst Rodin	5	4	3	2	1	

Please rate using 5 as most effective and 1 as least effective:

Rate your overall satisfaction with the opportunity to network with colleagues.	5	4	3	2	1
Rate your overall satisfaction with the quality of this conference/workshop.	5	4	3	2	1
Please rate your satisfaction with the organization of the conference/workshop.	5	4	3	2	1
How would you rate the cost of registration versus what you personally got out of the conference?	5	4	3	2	1

What topics should be addressed at future meetings?

What features should be added to future meetings?

What features should be deleted from future meetings?
.

Did you perceive commercial bias in any of the presentations? ☐ Yes ☐ No

Explain:

SAVE THE DATE

2016 ACMEGS Course and Meeting
February 10 and 11, 2016
Hilton Orlando Lake Buena Vista
Orlando, Florida

ACMEGS
AMERICAN CLINICAL MEG SOCIETY

www.acmegs.org