8th Annual ACMEGS Meeting | Thursday, February 6, 2014

Westin Peachtree Plaza, Atlanta, GA

ACMEGS

AMERICAN CLINICAL MEG SOCIETY

Welcome to Atlanta!

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

This is our 8th 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).

As usual, we kept the Annual Business meeting and the MEG-Economics component to the morning session to encourage interested ACNS members to join us subsequently for the 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 six presentations delivered by experts in the field of clinical MEG, and we are very glad to welcome among them Dr. Fernando Maestu from Spain.

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 Atlanta 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

Organizing Committee:
Anto Bagić, University of Pittsburg, Pittsburgh PA
Susan Bowyer, Henry Ford Hospital, Detroit MI
Richard Burgess, Cleveland Clinics Foundation, Cleveland OH
Michael Funke, University of Texas, Houston, TX
Paul Ferrari, University of Texas at Austin, Austin, TX
John Ebersole, Atlantic Neuroscience Institute, Summit, NJ
Gretchen Von Allmen, University of Texas, Houston, TX

2014 ACMEGS Conference
Thursday, February 6, 2014
Westin Peachtree Plaza • Atlanta, Georgia

8:00 AM — Arrival / Breakfast Reception

8:45 AM — ACMEGS Presidential Address 2014
Welcome and Introduction (Anto Bagic, Pittsburgh, PA)

9:00 AM — Annual Business Meeting (for ACMEGS members only)
- Minutes of February 7, 2013, Business Meeting (Anto Bagic, Pittsburgh, PA)
- Presidents Report (Anto Bagic, Pittsburgh, PA)
- Financial Report (Susan Bowyer, Detroit MI)
- Public Relations Committee (Susan Bowyer, Detroit MI)
- New Business
  - Elections
  - Changes to Bylaws (Michael Funke, Houston TX)
- Affordable Care Act (ACA) and MEG (Michael Longacre, Crofton MD)

10:00 AM — Current Issues and Enduring Questions in Clinical MEG
- Everything You Always Wanted to Know About Source Models (But Were Afraid to Ask) (John Moran, Detroit MI)
- Whole-Brain Functional Connectivity in Focal Epilepsy (Deepak Madhavan, Omaha, NE)
- Clinical Application of MEG Source Connectivity Analysis (Wenbo Zhang, Minneapolis MN)
- MEG Results in the Operating Theater: How We Do it (Anto Bagic, Pittsburgh PA)

12:00 PM — Annual ACMEGS Photo Shoot / Lunch

1:00 PM — Platform Presentations

2:00 PM — Towards a New Biomarker in Dementia
- Why and What Biomarkers are Ideally Needed (Jim Becker, Pittsburgh PA)
- First Results of the Multi-Center MAGIC-AD Study (Fernando Maestu, Madrid ES)

3:30 PM — Coffee Break

4:00 PM — Update on Educational Initiatives
- Update on MEG Fellowship Curriculum (Rick Burgess, Cleveland OH)
- Update on MEG/EEG Technologist Activities (Janice Walbert, ABRET & Judy Ahn-Ewing, ASET)
- Update on Clinical Startup Recommendations (Paul Ferrari, Austin TX & Ron Gordon, Vancouver BC)

4:30 PM — Meeting Adjourn

6:00 PM — ACMEGS Dinner at ECCO
40 7th Street NE Atlanta, GA 30308 - (404) 347-9555
Ecco features seasonally inspired European cuisine and is conveniently located in the heart of Atlanta's thriving midtown neighborhood, Ecco—which was named a "Best New Restaurant in America" by *Esquire* and "Best New Restaurant" by *Atlanta Magazine* when it opened in 2006.

ACMEGS
AMERICAN CLINICAL MEG SOCIETY

Presidential Address

Anto Bagic, Pittsburgh, PA

Presidential Address 2014

Anto Bagić, MD, PhD
(Pittsburgh, PA)

February 6, 2014; Atlanta, GA

ACMEGS Year In Retrospect (1/5)

- Society in good standing with Commonwealth of MA.
- Center Members (15): (30 delegated members).
- Individual Members: 15 (7 full + 8 associate)
- Changed a management agency from S&S to **EDI**.

Society In Good Standing With The Commonwealth Of MA

- **Administrative Issues**
  - All resolved
  - Collective efforts:
    - ACMEGS Board
    - **EDI (Milwaukee, WI).**
    - Attorney in Boston
    - Accountant in Pittsburg

2013 ACMEGS Honor Roll

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	Froedtert Hospital	Milwaukee	WI
7	Henry Ford Hospital	Detroit	MI
8	Meadowlands Hospital	Secaucus	NJ
9	MIND-University of New Mexico	Albuquerque	NM*
10	Minnesota Epilepsy Group	St. Paul	MN
11	Nebraska Medical Center	Omaha	NE
12	University of California San Francisco	San Francisco	CA*
13	University of Pittsburgh Medical Center	Pittsburgh	PA
14	University of Tennessee	Memphis	TN
15	UT Memorial Herman	Houston	TX

(30 delegated members)

Individual ACMEGS Members

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

Our First Half Year With EDI
(Since 07/01/2013)

Ms. Kay Whalen, MBA, CAE
President of EDI

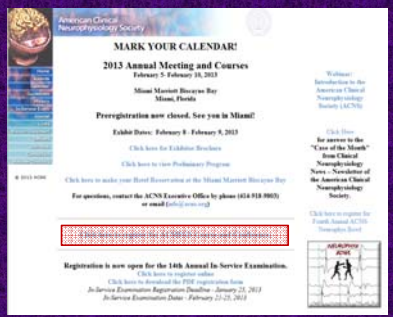
ACMEGS Executive Director:
Ms. Megan Kelly

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

ACMEGS Year In Retrospect (2/5)

- Continued productive relationship with the ACNS.
- The 2nd Board Retreat (Pittsburgh, PA; May 12-14, 2013).
- Informal interactions with Elekta representatives (AES, December 6-10, 2013).

Continued Productive Relationship with the ACNS



Planning for the 2nd Board Retreat (Pittsburgh, PA; May 12-14, 2013).

The 2nd ACMEGS Board Retreat (Pittsburgh, PA; May 12-14, 2013).

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.**
  - Newsletter (Check it out and contribute!).
  - Website redesign (upcoming).

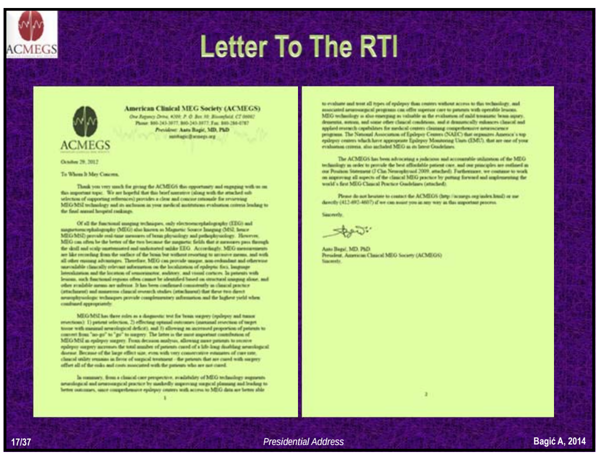
ACMEGS Web-based Resources

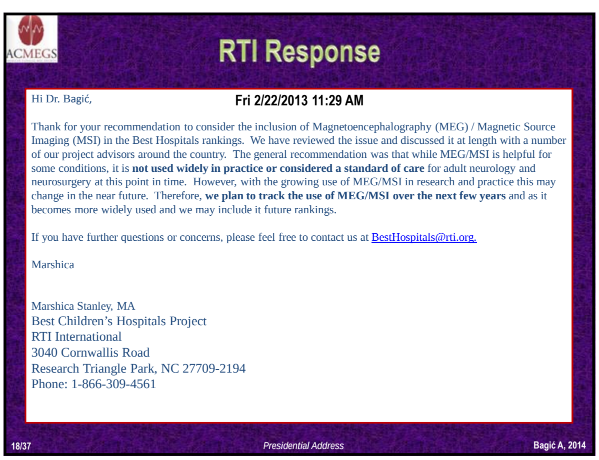
ACMEGS Year In Retrospect (4/5)

- **Sustained efforts on increasing the value of the MEG centers to their institutions:**
  - Supplementing the items on the previous slide.
  - 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.

Engaging with the RTI

Rank	Hospital	Points	Specialties
1	Johns Hopkins Hospital, Baltimore	30	15
2	Massachusetts General Hospital, Boston	29	16
3	Mayo Clinic, Rochester, Minn.	29	15
4	Cleveland Clinic	27	14
5	UCLA Medical Center, Los Angeles	19	13
6	Northwestern Memorial Hospital, Chicago	17	12
7	New York-Presbyterian University Hospital of Columbia and Cornell, N.Y.		
7	UCSF Medical Center, San Francisco		
9	Brigham and Women's Hospital, Boston		
10	UPMC-University of Pittsburgh Medical Center		





Hi Dr. Bagić, **Fri 2/22/2013 11:29 AM**

Thank for your recommendation to consider the inclusion of Magnetoencephalography (MEG) / Magnetic Source Imaging (MSI) in the Best Hospitals rankings. We have reviewed the issue and discussed it at length with a number of our project advisors around the country. The general recommendation was that while MEG/MSI is helpful for some conditions, it is **not used widely in practice or considered a standard of care** for adult neurology and neurosurgery at this point in time. However, with the growing use of MEG/MSI in research and practice this may change in the near future. Therefore, **we plan to track the use of MEG/MSI over the next few years** and as it becomes more widely used and we may include it future rankings.

If you have further questions or concerns, please feel free to contact us at BestHospitals@rti.org.

Marshica

Marshica Stanley, MA
Best Children's Hospitals Project
RTI International
3040 Cornwallis Road
Research Triangle Park, NC 27709-2194
Phone: 1-866-309-4561

ACMEGS Year In Retrospect (5/5)

- **ACMEGS educational efforts and activities:**
  - **Annual Course (3rd yesterday).**
  - Survey on the training opportunities (Dr. R. Burgess).
  - Upcoming discussion later today (Moderator: Dr. R. Burgess).
  - **Are we ready for a fellowship concept?**
  - MEG technologists survey (ASET).
  - Web-based resources.
  - **Individual help**.

The 3rd Annual MEG Course

Clinical MEG Course (5.75 hours CME credit)

2014 ACMEGS MEG Courses
Wednesday, February 5, 2014
Westin Peachtree Plaza
Atlanta, Georgia

Principles and Practice of Clinical Magnetoencephalography

8:00 AM	Registration/Breakfast	
8:30 AM	Welcome/Introduction	John Ebersole, MD
8:35 AM	Neurophysiological Basis and Recording Fundamentals of MEG and EEG	Richard Burgess, MD, PhD
9:25 AM	MEG Lab Organization and Data Acquisition	Anto Bagic, MD, PhD
10:15 AM	Break	
10:30 AM	Dipole Modeling of Epileptiform Activity	John Ebersole, MD
11:20 AM	Source Modeling of Evoked Activity	Michael Funke, MD, PhD
12:10 PM	Questions/Discussions	Faculty
12:30 PM	Closing Remarks/Farewell	John Ebersole, MD

Clinical Usefulness of MEG/EEG Source Modeling: Case Reviews

5:30 PM	Registration/Refreshments	
6:00 PM	Welcome/Introduction	John Ebersole, MD
6:05 PM	Pediatric and Adult Case Presentations	Faculty
8:00 PM	Closing Remarks/Farewell	John Ebersole, MD




The 1st ACNS MEG SIG

The Coming of Age of Magnetoenecphalography

Chair: Anto Bagić, MD, PhD
Pittsburgh, PA

ACNS Annual Meeting 2013
February 9, 2013
Miami, FL

Speakers

Michael Wagner, PhD [Compumedics Germany GmbH (f)]
Seeking the Sources: Dealing with Ill-Posed Problems of MEG and EEG Source Localization

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

Michael E. Funke, MD, PhD (HTHSC, Houston, TX)
What Do You Mean What I and How I feel? Current Role of MEG in Brain Mapping

Anto Bagić, MD, PhD (University of Pittsburgh, Pittsburgh, PA)
Quo Vadis Clinical MEG?

ISACM August 27, 2013


Satellite Symposium

SS Satellite Symposium : Elekta 15:30 - 17:30 / Tue 27th

Chair : Kyousuke Kamada (Department of Neurosurgery, Asahikawa Medical University, Asahikawa, Japan)

SS-1 "Clinical utility of magnetoencephalography in surgical management of epilepsy"
Richard Burgess
(Director, Magnetoencephalography Laboratory, and Cleveland Clinic Epilepsy Center, Cleveland, Ohio, USA)

SS-2 "Potential clinical utility of magnetoencephalography in traumatic brain injury and Alzheimer's disease"
Fernando Maestú
(Laboratory of Cognitive and Computational Neuroscience, Center for Biomedical Technology)

SS-3 "Machine learning and brain-computer interfacing with MEG: Clinical aspects"
Lauri Parkkonen
(Department of Biomedical Engineering and Computational Science (BECS), Aalto University, Finland)

関連講習会 (Japanese session)

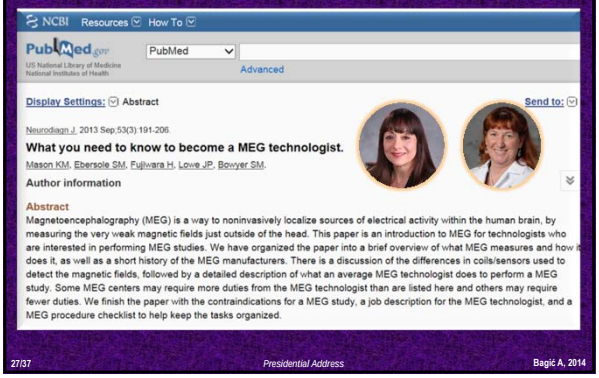
「Matlab講習会」››› 特設ページへ 13:00 - 15:00 / Tue 27th

Elekta Webinar

- November 21, 2013 (10:00 – 11:00 EST)
- Richard Burgess, MD, PhD

- **"What the referring physician needs to know about magnetoencephalography (MEG)"**

ABRET/ASET + ACMEGS


NCBI Resources How To

PubMed.gov US National Library of Medicine National Institutes of Health

PubMed Advanced

Display Settings: Abstract Send to:

Neurodiagn J. 2013 Sep;53(3):191-206.

What you need to know to become a MEG technologist.

Mason KM, Ebersole SM, Fujiwara H, Lowe JP, Bowyer SM.

Author information

Abstract

Magnetoencephalography (MEG) is a way to noninvasively localize sources of electrical activity within the human brain, by measuring the very weak magnetic fields just outside of the head. This paper is an introduction to MEG for technologists who are interested in performing MEG studies. We have organized the paper into a brief overview of what MEG measures and how it does it, as well as a short history of the MEG manufacturers. There is a discussion of the differences in coils/sensors used to detect the magnetic fields, followed by a detailed description of what an average MEG technologist does to perform a MEG study. Some MEG centers may require more duties from the MEG technologist than are listed here and others may require fewer duties. We finish the paper with the contraindications for a MEG study, a job description for the MEG technologist, and a MEG procedure checklist to help keep the tasks organized.

Special Interest Group

December 9, 2013

MEG Navigates to Neurosurgeons: A Trajectory of Success

Coordinator: **Anto Bagić, MD, PhD**
University of Pittsburgh, PA

American Epilepsy Society | **Annual Meeting**
Washington, DC

AMERICAN EPILEPSY SOCIETY 2013

AES/MEG SIG:
MEG NAVIGATES TO NEUROSURGEONS: A TRAJECTORY OF SUCCESS
Program

- 15:45 – 15:50 *Introduction*
- **15:50 – 16:10** **Andreas Alexopoulos, MD, MPH**
- 16:10 - 16:20 *Panel & Audience Discussion*
- **16:20 – 16:40** **Stefan Rampp, MD**
- 16:40 - 16:50 *Panel & Audience Discussion*
- **16:50 – 17:10** **Jorge Gonzalez-Martinez MD, PhD**
- 17:10 - 17:15 *Panel & Audience Discussion*



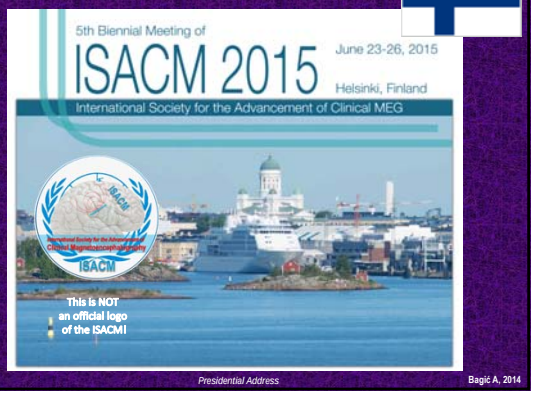
American Epilepsy Society | **Annual Meeting**

AMERICAN EPILEPSY SOCIETY 2013

What Is Ahead?

- **Sustain the current efforts on all fronts.**
- Escalate efforts on increasing (center) membership.
- Cultivate the relationship with the ACNS.
- Structure relationship with Elekta.
- Foster the relationship with the AES, AAN, ASET, ABRET, ISACM.
- **Increase our presence at appropriate neurosurgical conferences.**
- **Facilitate collaborative efforts on clinical research leading to new potential indications for MEG.**

http://www.biomag2014.org/



Tune Your Travel Plans

- **ACNS 2014** Annual Meeting (February 7 - 9, 2014; Atlanta, GA).
- **Biomag 2014** (August 24 - 29, 2014; **Halifax, Canada**).
- **ISACM 2014** (August 24 - 29, 2014; **Halifax, Canada**).
- **AES 2014** Annual Meeting (December 5 – 9, 2014; **Seattle, WA**).
- **ACMEGS 2015** Annual Meeting (February 5, 2014; **Houston, TX**).
- **ACNS 2015** Annual Meeting (February 3-8, 2015; **Houston, TX**).
- **ISACM 2015** (August 24 - 29, 2014; **Halifax, Canada**).

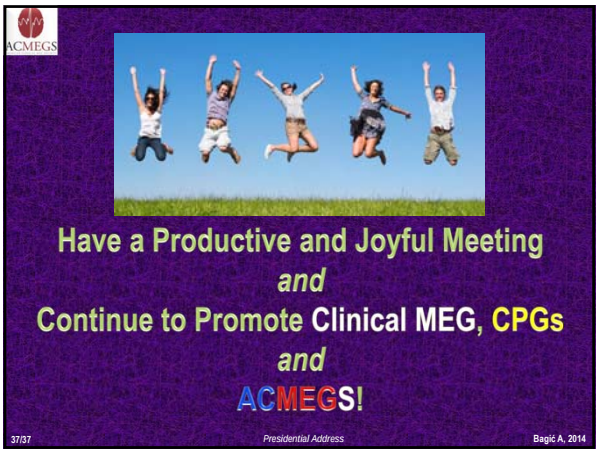
Acknowledgments

- **ACMEGS Members (Centers and individuals)**
- **ACMEGS Board Members**
- **Michael Longacre & Gregory R. Barkley**
- **Elekta Neuromag Oy**
  - Unrestricted educational grant
- **ACNS**
  - Synchronized meetings, CME approval, Sharing posters
- **ASET/ABRET**
  - Educational programs for technologists
- **S&S Management Inc.** (Jackie Coleman, Marie Westlake)
- **EDI** (since July 1, 2013: Megan Kelley)

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.*


ACMEGS
Have a Productive and Joyful Meeting
and
Continue to Promote Clinical MEG, CPGs
and
ACMEGS!
37/37
Presidential Address
Bagić A, 2014

Annual Business Meeting

(ACMEGS Members Only)

- Minutes of February 7, 2013, Business Meeting (Anto Bagic, Pittsburgh, PA)
- Presidents Report (Anto Bagic, Pittsburgh, PA)
- Financial Report (Susan Bowyer, Detroit MI)
- Public Relations Committee (Susan Bowyer, Detroit MI)
- New Business
    - Elections
    - Changes to Bylaws (Michael Funke, Houston TX)
- Affordable Care Act (ACA) and MEG (Michael Longacre, Crofton MD)
- Adjourn

ACMEGS
AMERICAN CLINICAL MEG SOCIETY

President's Report

Anto Bagic, Pittsburgh, PA

Financial Report

Susan Bowyer, Detroit, MI

ACMEGS
AMERICAN CLINICAL MEG SOCIETY

Public Relations Committee Report

Susan Bowyer, Detroit, MI

New Business: Elections

New Business: Changes to Bylaws

Affordable Care Act (ACA) and MEG

Michael Longacre, Crofton, MD

Once again CMS has reduced the reimbursement for MEG. This presentation will focus on the potential reasons for this reduction and proposed remedies by individual MEG Centers, the AAN and the ACMEGS.

GIGO!

Michael Longacre
National Accounts Director
Special Projects – Payer Markets
Assurex Health Inc.

Core Principle

Garbage in, garbage out (**GIGO**) refers to the fact that computers will unquestioningly process unintended, even nonsensical, input data ("garbage in") and produce undesired, often nonsensical, output ("garbage out").

Disclaimer

- The focus of todays presentation/discussion is to encourage the accurate capturing of **costs** associated with the use MEG.
- The purpose of this presentation/discussion is to encourage the accurate reporting of **costs** to CMS which is their basis for a calculated reimbursement for MEG.

Reminder: At no time should actual **charges** for MEG be mentioned or discussed.

Costs for Hospital Outpatient Services

CMS reported costs for hospital outpatient services, by HCPCS code for CY 2014

HCPCS Code	APC	Payment Rate	Total Frequency	Minimum Cost	Maximum Cost	Median Cost	Geometric Mean Cost
95965	0065	$1,740.86	88	$500.51	$11,602.83	$1,675.51	$1,802.13
95966	0065	$1,740.86	49	$198.01	$7,249.64	$1,650.53	$1,738.13

(CPT codes and descriptions only are copyright 2011 American Medical Association. All Rights Reserved. Applicable FARS/DFARS Apply.)
http://www.cms.gov/apps/ama/license.asp?file=/Medicare/Medicare-Fee-for-Service-Payment/HospitalOutpatientPPS/Downloads/CMS-1601-FC-Cost-Stats.zip

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http://www.cms.gov/apps/ama/license.asp?file=/Medicare/Medicare-Fee-for-Service-Payment/HospitalOutpatientPPS/Downloads/CMS-1601-FC-Cost-Stats.zip

MEG CPT Codes

95965 Magnetoencephalography (MEG), recording and analysis; for spontaneous brain magnetic activity (eg, epileptic cerebral cortex localization)

95966 Magnetoencephalography (MEG), recording and analysis; for evoked magnetic fields, single modality (eg, sensory, motor, language, or visual cortex localization)

95967 Magnetoencephalography (MEG), recording and analysis; for evoked magnetic fields, each additional modality (eg, sensory, motor, language, or visual cortex localization) (list separately in addition to code for primary procedure)

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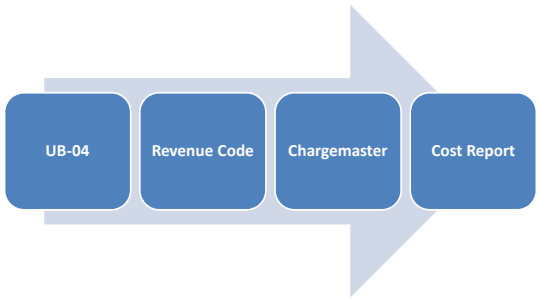
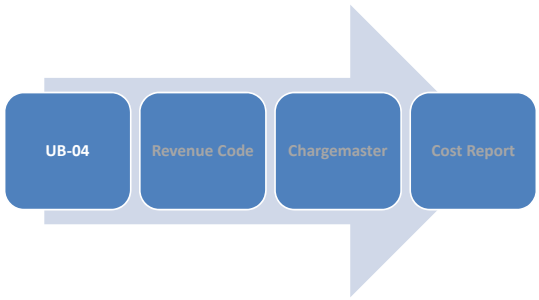
http://www.acmegs.org/health-insurance-codes-and-meg-payors

Key Terms



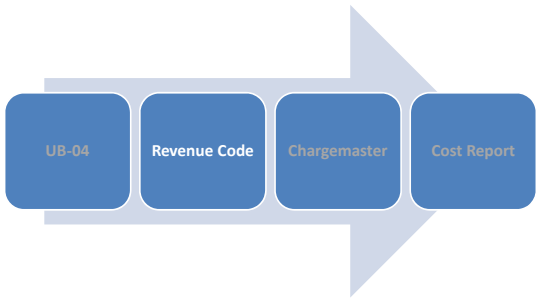
Key Terms



UB-04

The UB-04 is a uniform institutional billing claim form used by hospitals, clinics, ambulatory surgery centers, rehabilitation centers, etc.

Key Terms



Definitions

- **UB- 04** - The UB-04 is a uniform institutional billing claim form used by hospitals, clinics, ambulatory surgery centers, rehabilitation centers, etc.
- **Revenue Code** - Revenue codes are 3-digit numbers that are used on hospital bills to tell payors either where the patient was (department) when they received treatment, or what type of item a patient might have received as a patient
- **Chargemaster** - A charge master is a listing of every single procedure that a hospital can provide to its patients. Hospitals have charge masters because it helps to make the process of charge capture and billing move smoother. Charge masters have more than procedures on them. Pharmaceuticals, supply charges, and even some room charges are on charge masters
- **Cost Report** - The cost report is an annual report submitted by all facilities participating in the Medicare program. The cost information and statistical data reported must be current, accurate and in sufficient detail to support an accurate determination of payments made for the services rendered.

Revenue Codes for MEG

Revenue Codes (Department)

0860 - Magnetoencephalography (MEG) – General Classification

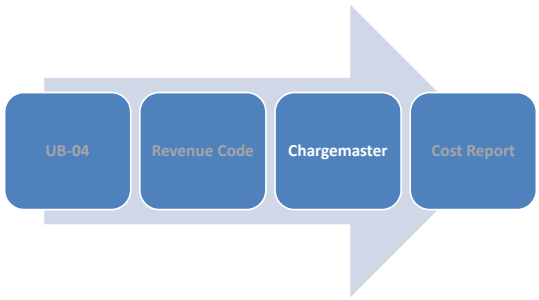
0861 – MEG

http://www.acmegs.org/health-insurance-codes-and-meg-payors

MEG Revenue Codes

This was accomplished with the addition of MEG specific revenue codes for 2012.

Key Terms



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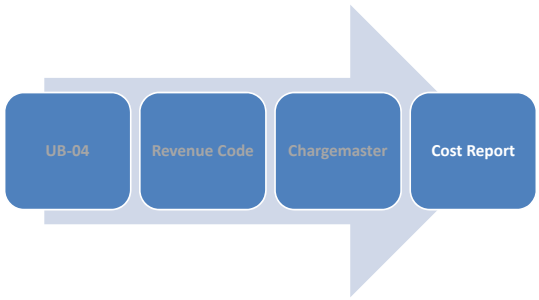
Charge

- The charge dollar amount represents the amount charged for the item and the amount that will appear on the patient's detailed bill.
- The charge does not indicate the reimbursement amount.
- Some facilities prefer to use the term "price" instead of "charge."
- There is no magical formula to assist facilities with setting the correct charge for a procedure.
- The charge is usually based on how much a procedure costs to perform and marked up a set percentage to cover expenses.

Key Terms



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- **Cost Report** - The cost report is an annual report submitted by all facilities participating in the Medicare program. The cost information and statistical data reported must be current, accurate and in sufficient detail to support an accurate determination of payments made for the services rendered.

Cost Report

The Medicare Cost Report is the core basis of Medicare payment system. For almost five decades the government has used the Cost Report to calculate payments to hospitals. So over the decades any good CFO would make sure that his charges maximized his governmental payments.

Medicare and Medicaid usually make up 60% or the his total payments. Some 53 years ago charges became a substitute for statistics and cost accounting to estimate how much the government was going to pay you. Hospitals get paid based on DRGs, but still must do a Cost Report to justify the DRG amounts.

CMS Responds to ACMEGS Request for Separate Cost Line

CMS-1525-P
Federal Register / July 18, 2011/ Proposed Rules / page 64

…….. we believe that the CCRs that we apply to the EEG revenue codes are more likely to result in a more accurate estimated cost for MEG than would the application of the hospital-specific overall ancillary CCR. For hospitals that report charges under revenue code 860 or 861 but do not report costs on their cost report under cost center 3280 or 5400, we are proposing to apply the hospital-specific overall CCR to the charges reported under revenue code 860 or 861 for purposes of estimating the cost of these services.

CMS Responds to ACMEGS Request for Separate Cost Line

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Definitions

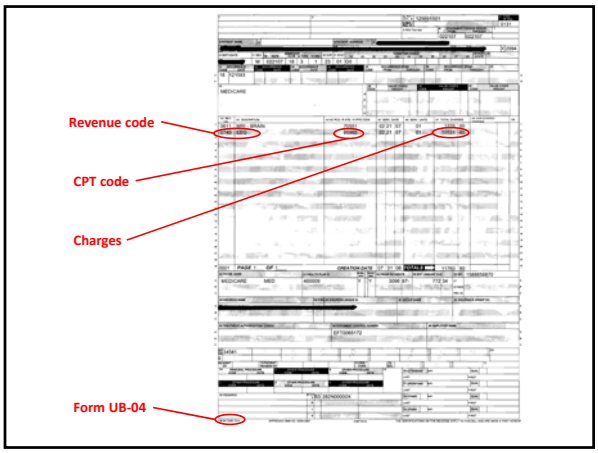
- **UB- 04** - The UB-04 is a uniform institutional billing claim form used by hospitals, clinics, ambulatory surgery centers, rehabilitation centers, etc.
- **Revenue Code** - Revenue codes are 3-digit numbers that are used on hospital bills to tell payors either where the patient was (department) when they received treatment, or what type of item a patient might have received as a patient
- **Chargemaster** - A charge master is a listing of every single procedure that a hospital can provide to its patients. Hospitals have charge masters because it helps to make the process of charge capture and billing move smoother. Charge masters have more than procedures on them. Pharmaceuticals, supply charges, and even some room charges are on charge masters
- **Cost Report** - The cost report is an annual report submitted by all facilities participating in the Medicare program. The cost information and statistical data reported must be current, accurate and in sufficient detail to support an accurate determination of payments made for the services rendered.

MEG Center To Do List

Reach out to your administration:

- Obtain a copy of UB-04 for a Epilepsy Medicare patient.
  - CPT Code
  - Revenue Code
  - Reasonable Charges

UB-04 Form



Costs for Hospital Outpatient Services

CMS reported costs for hospital outpatient services, by HCPCS code for CY 2014

HCPCS Code	APC	Payment Rate	Total Frequency	Minimum Cost	Maximum Cost	Median Cost	Geometric Mean Cost
95965	0065	$1,740.86	88	$500.51	$11,602.83	$1,675.51	$1,802.13
95966	0065	$1,740.86	49	$198.01	$7,249.64	$1,650.53	$1,738.13

(CPT codes and descriptions only are copyright 2011 American Medical Association. All Rights Reserved. Applicable FARS/DFARS Apply.)
http://www.cms.gov/apps/ama/license.asp?file=/Medicare/Medicare-Fee-for-Service-Payment/HospitalOutpatientPPS/Downloads/CMS-1601-FC-Cost-Stats.zip

AAN Comments to CMS

For 2014, CMS finalized its policy to group all of the magnetoencephalography (MEG) codes (95965-95967) in APC 0065. The AAN disagrees with CMS' decision to move MEG code 95965 from APC 0066 to APC 0065. We also do not believe that APC 0065 captures the cost of add-on code 95967. In fact, it appears that all of the costs for CPT code 95967 are lost since it is now packaged with CPT code 95966.

The cuts in reimbursements are dramatic and unsustainable for neurologists who perform MEG. The reimbursement does not cover the cost of doing business and are likely to impact patients' access to this service. **CMS should work with the AAN to develop a solution to this problem and assign more appropriate APCs – whether it is identifying existing APCs or developing new APCs.**

ACMEGS To Do List

- Access MedPAR data base to determine accuracy of MEG claims.
- Continue our partnership with the AAN in dialogues with CMS to find potential solutions.
- Provide a resource to the MEG centers to assist in determining appropriate reporting of MEG on the UB-04, Chargemaster and Cost Report.

Thank you for your time and attention!

ICD-10

ICD-10 Implementation October 1, 2014

Current Issues and Enduring Questions in Clinical MEG

Everything You Always Wanted to Know About Source Models (But Were Afraid to Ask)

John Moran, Detroit MI

In discussions of MEG based analysis of brain activity, the topic of source models have traditionally been restricted to discussions of the merits of various forward modeling and source imaging techniques. These topics are important but highly technical and can require a high degree of nuance in their application to bioelectric imaging. Rather, important factors that underlies measurement of brain signals and subsequent construction of imaging methods are detailed. Then, the emphasis of the presentation shifts to source models which consist of interacting brain regions. In these models, most bioelectric source activity is determined by received signals from other regions. Thus, a network source model is mathematically constrained to account for these network interactions as well as explain the measured MEG data. In particular, a network source model is constructed where MEG coherence imaging is used to identify active network sites while fiber tract based connectivity determines the physical site-to-site connections. Clinical utility of this approach is demonstrated by identifying the site of an epileptic focus based completely on subsequent parameter analysis of the constructed epileptic source model network.

BIOELECTRIC SOURCE SPACE

Every thing you wanted to know about it?

Better yet, some important things you should know

John E Moran PhD
Biomedical Physicist
Epilepsy Center
Cleveland Clinic

Signals

EXTERNAL NUSANCE SOURCES:
- Distant sources:
  - Elevators, Trucks, cars, trains, power lines)
- Near By Sources:
  - Electrical Artifact: (stimulators, monitors)
  - Magnetic Atrifact: (Teeth, etc)
  - Bio-electric Artifact: (Heart, Eye movements)

BRAIN NETWORK SOURCES
- NUSANCE SOURCES
  - Spontaneous Activity
  - Event Related Activity
- INTERESTING SOURCES
  - Spontaneous Activity
  - Event Related Activity

Primary Guiding Factor of Soure Space Analysis

ACHIEVE THE MEASUREMENT GOAL:

Reliably measure signals of interest as required as a basis for quantifying a specific feature of Brain Network Activity

Factors in achieving the goal

- Sensor Type and Design: (Signal Sensitivity and Specificity)
  - EEG, ECoG, MicroElectrodes,
  - MEG (Magnetometer, Axial and Planar Gradiometer)
- Auxiliary Signal Processing:
  - Signal extraction and frequency band isolation
  - Signal-to-Noise enhancement
- Forward Modeling Methods:
  - Spherical, BEM, FEM, homogeneous conductor?
  - Cortical Surface Constraint versus Full Gray Matter Volume
- Goal oriented Imaging Technique Selection: ECD, Beamforer, Current Density
- Brain Network Analysis of imaged activity

MEG Sensor Configurations

The Elekta Neuromag sensor elements consists of two orthogonal planar gradiometers and one magnetometer coupled to a SQUID

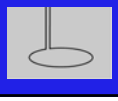
The 4D Neuroimaging sensor elements consists of either axial gradiometers or magnetometers coupled to a SQUID

CTF utilizes axial gradiometers coupled to a SQUID

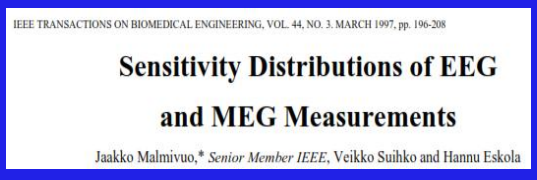
Planar Gradiometer

Axial Gradiometer

Magnetometer



Sensitivity of Sensors to Brain Source Location

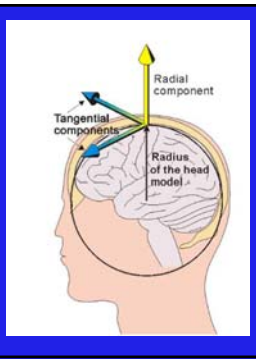


IEEE TRANSACTIONS ON BIOMEDICAL ENGINEERING, VOL. 44, NO. 3. MARCH 1997, pp. 196-208

Sensitivity Distributions of EEG and MEG Measurements

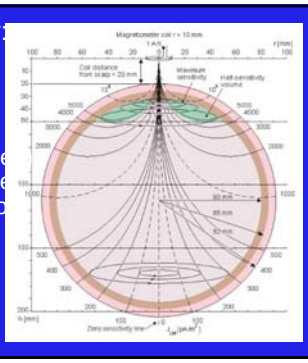
Jaakko Malmivuo,* *Senior Member IEEE*, Veikko Suihko and Hannu Eskola

Spherical Head Model: Current Components



Head Volume Sensitivity:
Magnetometer
Axial Gradiometer

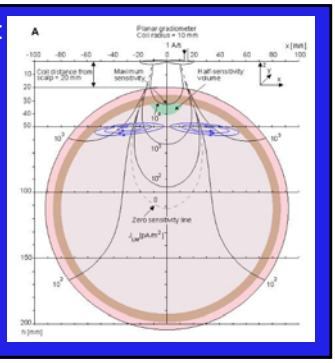
Half-Sensitivity Volume (gree
Magnetometer > Gradiomete
Zero Sensitivity under senso



Head Volume Sensitivity:
Planar Gradiometer
(along axis)

Half-Sensitivity Volume (green) Along the gradiometer axis

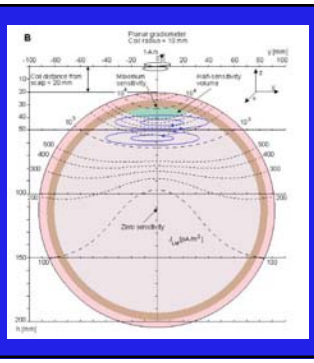
Zero Sensitivity loop between sensors



Head Volume Sensitivity: Planar Gradiometer (across axis)

Half-Sensitivity Volume (green) Along the gradiometer axis

Zero Sensitivity between sensors



Sensor Sensitivity & Specificity

Magnetometer & Axial Gradiometer

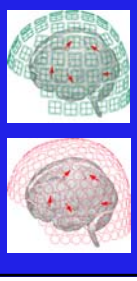
- Significant Sensitivity to Deep Cortical Sources
- Large Half Sensitivity Volume is donut shaped with hole is center.
- No sensitivity to sources directly under sensor
- Gradiometer Sensitivity depends on coil separation.

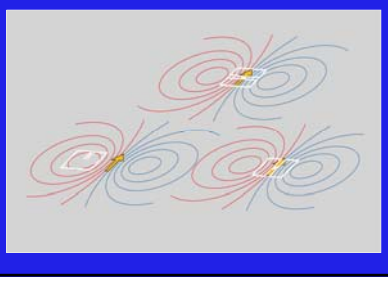
Planar Gradiometer

- Significantly greater specificity to shallow sources under sensor
- Low sensitivity for deep sources
- Small Half Sensitivity Volume.

Planar Gradiometers and Axial Sensors Array Utilize Information in Sensor Coupling to Neural Sources

(Neuromag configuration)

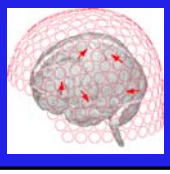


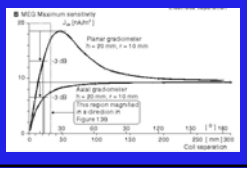


Magnetometer Array = Planar Gradiometer Array (mathematically equivalent)
Planar/Axial Gradiometer Array = Planar Gradiometer Array of Planar/Axial Gradiometers

Mathematical Equivalence utilized for:
- Artifact and noise suppression
- Extraction of signals of interest
- imaging locations of activation
- Calculation of Brain Coherence

Malmivuo, IEEE Tran BioMed. 1997





MEG Imaging:
The Magnetic Field Equation for Active Sources

$$B = GQ$$

- B = MEG DATA (Known but corrupt)
- Q = Brain Electric Source Activity (unknown)
- G = Gain matrix = $\Delta B/\Delta Q$ (unknown)

(Many Sources generate the same MEG Data)

MEG Imaging:
The Active Source Equation for MEG data

$$Q = WB$$

- B = MEG DATA (Known but corrupt)
- Q = Brain Electric Source Activity (unknown)
- W = Imaging Weight matrix = $\Delta Q/\Delta B$ (unknown)

(Q depends on assumption to calculate W)

Strategies for calculating W

(1) Optimize W to satisfy Source Selectivity (beamformer)
(2) Optimize W to satisfy statistical goal or property
(3) Optimize W to satisfy combination of 1 and 2
(4) Introduce added constraints:
anatomical constraints
number of source constraint (ECD)
mathematical uniqueness, stability
(5) Recursive estimation, L norm (non-linear, focal Q)
(6) Particle Filtering, Kalman Filter, W(t) , non-linear

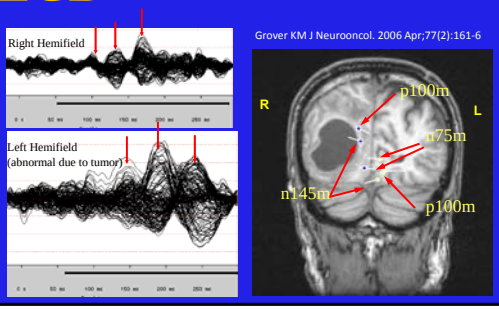
Calculation of W: Calibration

After Estimating Q by other means

$$Q_e = WB_f \Rightarrow W = Q_eB_f^{-1}$$

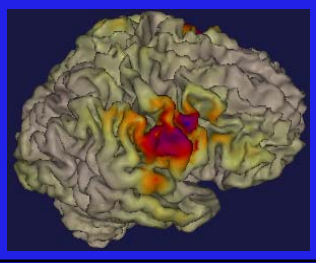
- B_f = MEG Forward Data (Known)
- Q_e = Brain Electric Source Activity (known)
- W = Imaging Weight matrix = $\Delta Q/\Delta B$ (unknown)

ECD



Grover KM J Neurooncol. 2006 Apr;77(2):161-6

MUSIC Find Multiple Sites of Activity



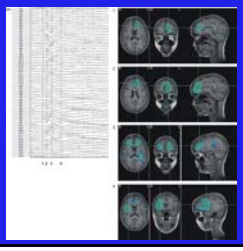
(Brainstorm Software)
(SEF 120 msec)

BEAMFORMERS

Site Specific Imaging Filters of Independent Activity

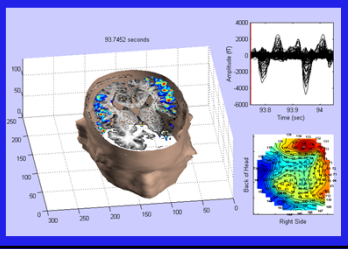
Beamformer analysis of spike propagation of two MEG spike–wave complexes. Both showed predominant epileptic activity frontal mesial bilateral and perisylvian region, however propagation sequence was different (blue to cyan signifies increasing activity).

Activity propagated from left frontal mesial area, to larger frontal areas, including polar and basal, bilateral frontal and subsequently to perisylvian areas. (Stefan et al , 2009)



Extended Source Imaging

Minimum Norm, Focuss, MR-Focuss, others



Source Space:

Network Analysis

Coherence Imaging

utilizes FFT transform

Phase Synchrony Analysis

narrow frequency band analysis of signal phase

Directed Network Analysis

requires knowledge of physical connectivity and signal timing

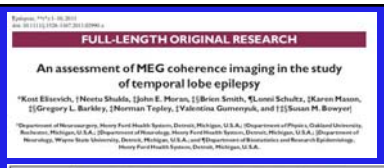
Coherence Imaging

- Spontaneous Activity (Default Mode Network)
  - Persistently Active Network Sites
  - Long Data Study
  - Average Region Functional Network Connectivity
- Evoked Activity
  - Visualize Goal directed Network
  - Estimate Strength interaction
  - Forward and backward Information Flow requires Grainger Causality Analysis of Imaged Activity

Coherence Imaging

- 10 minutes of MEG data sampled at 508 Hz
- Filtered 3-50 Hz and Heart artifact removed
- Divided into 7.5 second intervals for imaging and coherence calculations
  - ICA for extracting neuronal bursts of brain activity
  - MR-FOCUSS/Coherence imaging for determining the global extent of the epileptic network and the local spectrum of overall network coherence and connectivity.
- FFT with 256 point hanning window and 25% overlap
- Coherence results for all 7.5 second interval are averaged.
- Multiple runs processed to check for stability of results

Diagnostic Utility



FULL-LENGTH ORIGINAL RESEARCH

An assessment of MEG coherence imaging in the study of temporal lobe epilepsy

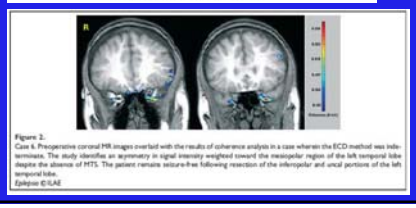
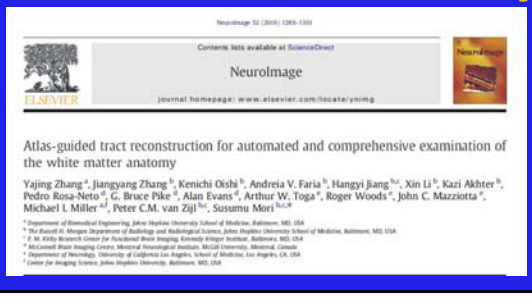


Figure 2.
Case 6. Preoperative coronal MR images overlaid with the results of coherence analysis in a case wherein the ECD method was indeterminate. The study identifies an asymmetry in signal intensity weighted toward the mesiopolar region of the left temporal lobe despite the absence of MTS. The patient remains seizure-free following resection of the interpolar and uncal portions of the left temporal lobe.
Epilepsia © ILAE

Fiber Tract Connectivity



NeuroImage

journal homepage: www.elsevier.com/locate/ynimg

Atlas-guided tract reconstruction for automated and comprehensive examination of the white matter anatomy

Yajing Zhang [a], Jiangyang Zhang [b], Kenichi Oishi [b], Andreia V. Faria [b], Hangyi Jiang [b,c], Xin Li [b], Kazi Akhter [b], Pedro Rosa-Neto [d], G. Bruce Pike [d], Alan Evans [d], Arthur W. Toga [e], Roger Woods [e], John C. Mazziotta [e], Michael I. Miller [a,f], Peter C.M. van Zijl [b,c], Susumu Mori [b,c,*]

Fiber Tract based Network Analysis

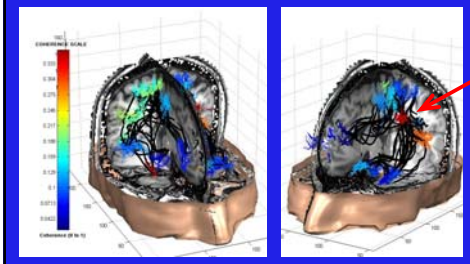
Physical Connectivity Network
- Connectivity by tract for 375 anatomical/functional sites

Functional Connectivity Sites
- Coherence and activity amplitude identify active sites

Directed Connectivity Strength
- Calculated for directly connected active sites
- Activity correlation lags used to define directionality

Unique Network Input
- Signal components not derived from network interaction

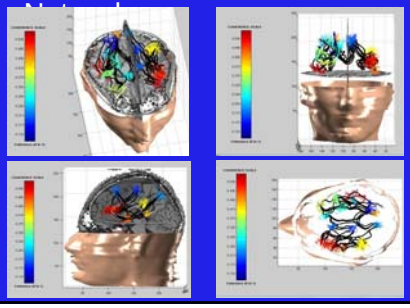
Coherence & maximum connection network



Unilateral Right Tinnitus
high coherence
Left Auditory Cortex

Note: Red &Orange in Left AC is most active.

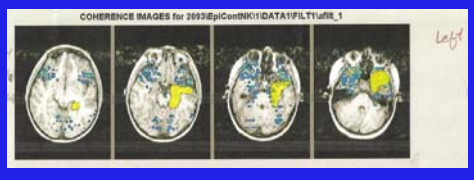
Schizophrenia

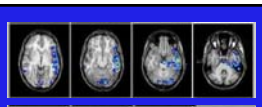


Visualize direct and indirect connectivity between active regions

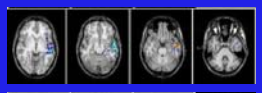
Identify Epileptic Zone

30 patients with good outcome
(mark surgically removed tissue)

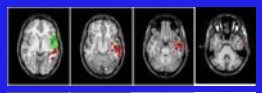




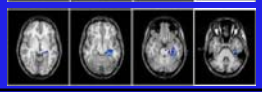
Coherence Imaging



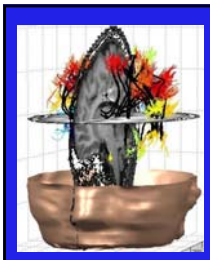
Network Feedback Strength



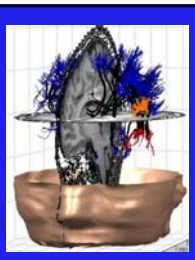
Network Directionality Out = Red In = Green



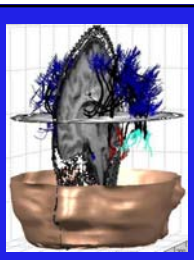
Network Signal Input



Coherence Imaging



Feedback Strength



Network Signal Input

Red = High Amplitude Blue = Low Amplitude

•ACHIEVE THE MEASUREMENT GOAL:

- Reliably measure signals of interest as required as a basis for quantifying a specific feature of Brain Network Activity
- Utilize appropriate signal processing to quantify the active network and network interactions.
- Quantify features of the network that are of interest

Thank you for your interest

moranhfh@gmail.com

ACMEGS
AMERICAN CLINICAL MEG SOCIETY

Current Issues and Enduring Questions in Clinical MEG

Whole-Brain Functional Connectivity in Focal Epilepsy

Deepak Madhavan, Omaha, NE

The analysis of interictal epileptiform discharges (IEDs) using magnetoencephalography (MEG) is utilized for the localization of seizure onset zones in the presurgical planning of epilepsy patients. Additionally, resting-state functional connectivity analyses using the IED area may provide novel insight into the underlying brain networks. In this study, we evaluate whether chronicity of seizures is related to whole-brain functional connectivity metrics using the area of IED generation (derived from MEG) as the seed region. We found a positive correlation between the duration of seizures and beta-band functional connectivity between the epileptogenic zone and other brain areas. This suggests the presence of inhibitory GABAergic modulation of distal brain regions in response to chronic epileptiform activity. We are also trying to extend this concept to explore functional connectivity based relationships between intracranial EEG and MEG, in order to develop presurgical analysis protocols.

MEG Connectivity at UNMC

Deepak Madhavan, M.D.
Tony Wilson, Ph.D.
Hannah Kyllo, B.S.

Rationale

- Localization of Interictal Epileptiform Discharges (IEDs) using MEG is a reliable indicator of the epileptogenic onset zone
- IEDs have also been examined in the setting of resting state functional connectivity
  - Primarily using EEG/fMRI
  - Involvement of default mode network, thalamocortical circuits, etc.
- Goal of our study was to evaluate MEG resting functional connectivity
  - Using localized areas of IED generation as seed points
  - Relating to the time frame of each patients' epilepsy
- We anticipated that whole-brain connectivity increased as a function of the age of the epilepsy
  - Recruitment of distal brain regions into the epileptic pathway due to chronic seizure activity

Data Acquisition

- 12 adults (4 female) with intractable CPS
  - Mean number of AEDs tried and failed: 5.67
  - Mean age: 40.58 years
  - Range of epilepsy duration: 40-408 months
- Sleep deprived MEG
  - Acquisition bandwidth 0.1-330 Hz
  - 1 kHz sampling rate
  - Noise reduction with signal space separation with temporal extension

Source Analysis

- Following tSSS, raw MEG signal was filtered 1-45 Hz
- IEDs localized using ECD from 204 gradiometer sensors
- ECDs overlaid onto MRI images
- Dipole clusters used as seed region for connectivity analyses
  - Phase coherence was computed using a dense grid of regional source spaced equidistantly and the IED area as the seed region.

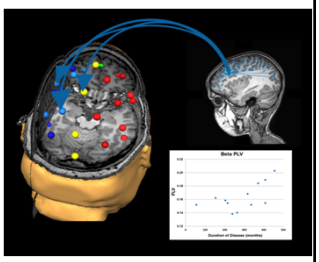
Connectivity Analyses

- We examined the relationship between the duration of epilepsy and phase-locking value between the epileptogenic zone and all other brain regions within the delta (1.0-4 Hz), theta (4-7.0 Hz), alpha (8-14 Hz), and beta (14-30 Hz) frequency bands using Pearson-correlation analyses

Results

- Pearson correlation

Indicated that duration of epilepsy was positively correlated with the amplitude of beta-band functional connectivity between the epileptogenic zone and other brain areas



Discussion

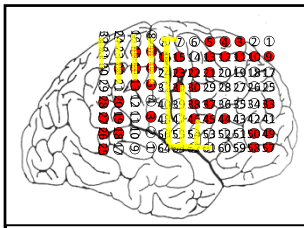
- Why only the beta band?
  - Increased beta activity thought to be related to increased GABAergic modulation
    - Possibly due to increased inhibitory interneuron activity?
  - Presence of increased beta band functional connectivity could indicate cerebral mechanisms responsible for maintaining local and remote inhibitory environments for the endogenous control of seizures

Further Work

- Can connectivity measures be used to demonstrate relationships between seizure onset zones in intracranial EEG vs MEG?
- Can identification of 'hyper-connected' areas within the visually identified seizure onset zone be used for more focal ablative surgical approaches?

Intracranial EEG "Accounting"

- Isolated epileptiform spikes (IEDs) from intracranial EEG
- Measured correlation of time-frequency for each spike and ran coherence tests (with BESA software) using each electrode within the visually identified seizure onset zone as a seed for all other electrodes
- PLV was computed for signals between 4-50 Hz at a resolution of 2.0 Hz and 25 ms then averaged across time for each frequency bin
- Procedure was iterated using each electrode within the SOZ as the seed for PLV computation
- We then identified the frequency bins where the PLV was at least one SD above the grand mean, and computed the percent of frequency bins (per electrode pair) where this threshold was exceeded.



= SEIZURE ONSET (resected)

= Arm/face/tongue motor

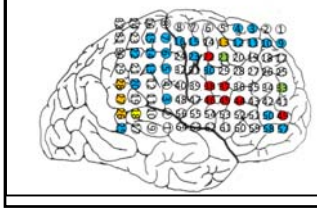


Figure legend:
Red = 90% connectivity with surrounding electrodes
Orange = 80-90% connectivity
Yellow = 70-80% connectivity
Green and blue = less than 70% connected

Lingering Questions

- Can we analyze connectivity measures using either MEG or intracranial EEG to isolate hyperconnected regions of the seizure onset zone?
  - Would focal ablation of these areas be necessary and/or sufficient in treating seizures?
  - Possible strategy for less invasive laser guided therapies

References

Thank You!

Current Issues and Enduring Questions in Clinical MEG

Clinical Application of MEG Source Connectivity Analysis

Wenbo Zhang, Minneapolis, MN

MEG/MSI has been approved for pre-surgical epileptogenic zone localization for more than 10 years. Epileptogenic zone can be defined by MEG when interictal magnetic fields clustered. However the MEG network study of epilepsy remains scarce, especially for neocortical epilepsy. eConnectome (Electrophysiological Connectome) is an open-source MATLAB software package for imaging brain functional connectivity from electrophysiological signals. It provides interactive graphical interfaces for EEG/ECoG/MEG preprocessing, source estimation, connectivity analysis and visualization. Connectivity from EEG/ECoG/MEG can be mapped over sensor and source domains. It is free for download at http://econnectome.umn.edu . Cases will be presented analyzed with the methodology. It provided a robust way to analyze source connectivity of MEG/MSI using directed transfer function (DTF) analysis. More case analysis should be done to better understand the clinical significance of DTF analysis. In conjunction with diffusion tensor imaging tractography, a more complete picture of interictal epilepsy network can be drawn.

Clinical application of MEG Source Connectivity Analysis

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Allina Health and Minnesota Children's Hospitals and Clinics
Adjunct Faculty of University of Minnesota

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Minnesota Epilepsy Group at Allina Health and Minnesota Children's Hospitals & Clinics

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Jason Doescher, M.D., Pediatric Epileptologist, Adjunct Faculty of University of Minnesota

Brian Owens, Neurodiagnostic Technologist, IGS technologist

University of Minnesota, Department of Biomedical Engineering

Bin He, PhD, Distinguished McKnight University Professor, Medtronic-Bakken Endowed Chair, Director, Institute for Engineering in Medicine, Director, Center for Neuroengineering

Yakang Dai, PhD, Post-doctoral associate (currently Professor of Biomedical Engineering, Chinese Academy of Sciences)

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All Patients from Minnesota Epilepsy Group

- Pediatric Epileptologists

Michael D. Frost, Frank Ritter, Jason Doescher, Dimitros Arkilo

- Adult Epileptologists

Patricia Penovich, Deanna Dickens, James White, Julie Hanna, Paul Atkinson, Michaela Chatman

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Clinical MEG/MSI

- CPT 90955 Magnetoencephalography, recording and analysis for spontaneous brain magnetic activity (e.g. epileptic cerebral cortex localization)
- MEG: non-invasive testing between phase 1 and 2 for medically refractory epilepsy
- Guide subdural grid placement or other invasive procedure; or more ambitiously guide resection?!

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Interictal magnetic fields

- Spikes with <70ms fast electromagnetic transients; sharps 70-120 ms followed by a slow wave lasting a few hundred ms
- Spikes propagate, could lead a seizure
- Cluster of interictal spikes indicate the epileptogenic zone
- area of neurons simultaneously firing can be detected: 4-6 cm^2 for MEG; 6-10 cm^2 for EEG.
- At least 5000 neurons firing simultaneously for MEG to be detected.

Okada and Xu. Neuroscience Letters 1996

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Interictal magnetic fields

- "The Neurosurgeon wants to know both the seizure onset zone and the region of immediate cortical spread to determine the epileptogenic zone to be resected." Rose et al. Front Neurol. 2013
- Primary epileptogenic zone or secondary?
- Patterns of interictal magnetic fields propagation?
- Defining the "quick spread zone" or remote propagation?

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Techniques

- Equivalent dipole model of EEG/MEG. (Ebersole 1991, Sutherling 1989)
- Beamformer Analysis (Robinson and Vrba, 1999; Stefan 2009; Rose 2012)
- Minimum Norm Estimates (MNE) (Hämäläinen 1989, Tanaka 2010)
- Granger causality , directed transfer function (DTF) (Granger 1969, Kamiński and Blinowska 1991, Dai 2012)
- Other techniques: Frequency domain analysis, independent component analysis (ICA) (Malinowska 2013)

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Method

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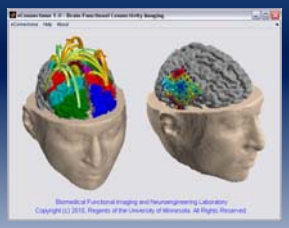
eConnectome

http://econnectome.umn.edu



- MATLAB-based, with GUIs
- for Functional Connectivity
- Support MEG/EEG/ECoG
- Open-source, GPL

Electrophysiological Connectome

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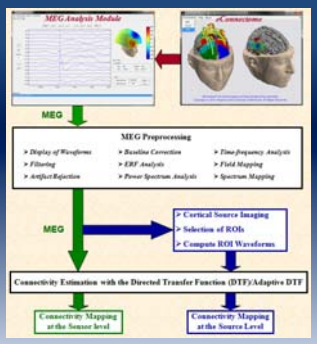
MEG Connectivity Analysis Framework of eConnectome



- Connectivity analysis of the preprocessed MEG data can be performed at the sensor level
- Cortical sources can be reconstructed from MEG data and connectivity analysis can then be performed at the source level
- Connectivity patterns among MEG sensors and cortical regions of interest (ROIs) can be visualized over the MEG sensor surface and realistic cortical surface respectively.
- The MEG source connectivity analysis was used to analyze interictal MEG spikes.

Selected GUIs

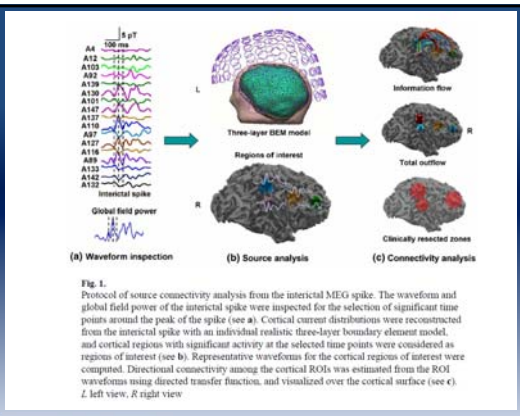


Fig. 1.
Protocol of source connectivity analysis from the interictal MEG spike. The waveform and global field power of the interictal spike were inspected for the selection of significant time points around the peak of the spike (see **a**). Cortical current distributions were reconstructed from the interictal spike with an individual realistic three-layer boundary element model, and cortical regions with significant activity at the selected time points were considered as regions of interest (see **b**). Representative waveforms for the cortical regions of interest were computed. Directional connectivity among the cortical ROIs was estimated from the ROI waveforms using directed transfer function, and visualized over the cortical surface (see **c**). *L* left view, *R* right view

Cases Analyzed

- Five surgical cases from Minnesota Epilepsy Group including 2 temporal lobe epilepsy cases, 3 neocortical cases with multiple foci
- Neocortical cases: multiple epileptogenic foci and more interictal zones
- These are the most challenging cases clinically and for the connectivity study, especially neocortical cases
- The data (Y.D.)is analyzed without prior clinical knowledge

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Cases Analyzed

Patient	Gender	Age of onset of seizures	Seizure type and frequency presurgically	Age at surgery	Pathology	Surgery	Surgery date	Outcome	Engel score
1	Female	2 months	Complex partial seizure, daily	11	Cortical Tubers and gliosis	Right temporal, parietal and frontal topectomy	5/2005	Seizure free remained with AEDs since surgery	IA
2	Female	19 months	Complex partial seizure, daily	3	Cortical tubers and gliosis	Right frontal and parietal topectomy	2/2008	Seizure free with AEDs since surgery	IA
3	Male	5 years	Complex partial seizure, 8-10/week	13	Astrogliosis	Right temporal lobectomy including mesial structure	10/2006	Single simple partial seizure remained with AEDs since surgery	IB
4	Female	10 years	Simple and Complex partial seizure, monthly	32	Moderate to remarked astrogliosis	Left temporal lobectomy and frontal topectomy	11/2005	2 simple partial seizures remained with AEDs since surgery	IB
5	Male	27 years	Complex partial seizure	31	Mild astrogliosis	Left temporal lobectomy including mesial temporal structure	10/2007	Seizure free with AEDs since surgery	IA

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Temporal Lobe Epilepsy

- Majority of TLE is mTLE, few lTLE
- Mesial to lateral temporal lobe
- Temporal to frontal
- To Contralateral temporal lobe
- Ipsilateral frontal central cortex

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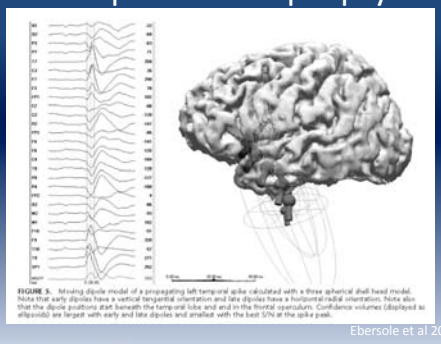
Temporal lobe epilepsy



Ebersole et al 2007

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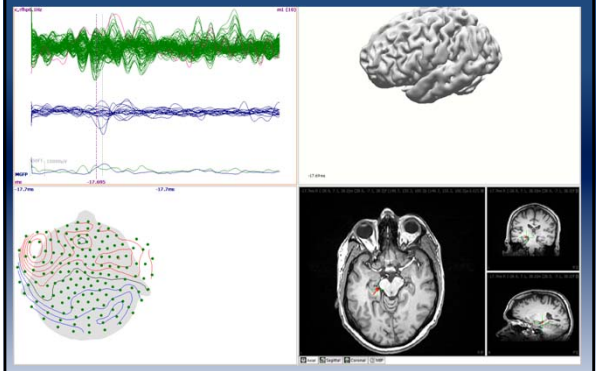

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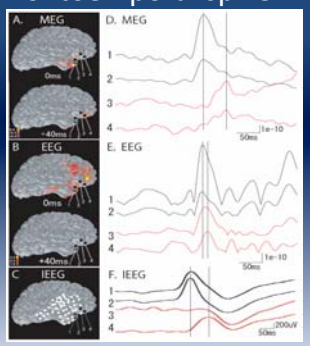
Frontoemporal spike- MNE approach



- A typical spike in Patient 1.
- The MNE-derived source distribution map of a spike obtained through (A) MEG; (B) EEG.Cortical activation is shown with red and yellow colors. Black circles show the sites shown in(C). The map of the MEG spike demonstrates activation in the right anterior temporal lobe at the temporal peak (0ms), and in the right inferior frontal area (+40ms). The EEG spike map demonstrates activation in both temporal and frontal lobes at mostly the same time (0ms), and disappears quickly (+40ms). (C) The location of intracranial electrodes determined on the cortical surface by using post-implantation CT images. Black circles numbered 1-4 show the frequently spiking sites of electrodes in the anterior temporal and inferior frontal lobes. Source waveforms of (D) MEG and (E) EEG extracted from the sites 1-4, shown on the left column. The temporal peak (black lines) precedes the frontal peak (red lines) approximately by 50ms in the MEG spike, and by 10ms in the EEG spike. (F) Waveforms of a typical IEEG spike obtained from the sites 1-4, shown on the left column. The temporal peak (black lines) precedes the frontal peak (red lines) approximately by 50ms. The time difference is similar in IEEG and MEG, whereas EEG shows a smaller difference.
- Tanaka et al. Neuroimage. 2010

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Frontoemporal spike- MNE approach

- The propagated frontal region: secondary, no-resection
- 4-86 ms means propagated to the secondary region
- More correlated with the path "*mediated by uncinate fasciculus, which connects anterior temporal lobe and inferior frontal lobe (Makris and Pandya, 2009)*".
- "*spatiotemporal analysis of MEG spikes may provide more accurate information of spike propagation in our patients than EEG. It may be clinically useful in the presurgical evaluation of epilepsy.*"

Tanaka et al. Neuroimage. 2010

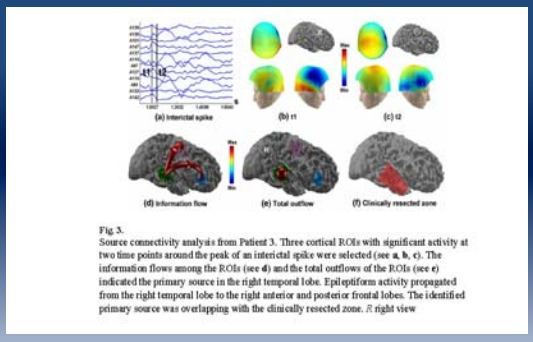


Fig. 3.
Source connectivity analysis from Patient 3. Three cortical ROIs with significant activity at two time points around the peak of an interictal spike were selected (see **a**, **b**, **c**). The information flows among the ROIs (see **d**) and the total outflows of the ROIs (see **e**) indicated the primary source in the right temporal lobe. Epileptiform activity propagated from the right temporal lobe to the right anterior and posterior frontal lobes. The identified primary source was overlapping with the clinically resected zone. *R* right view

Temporal Lobe Epilepsy

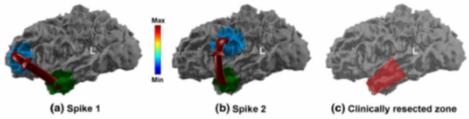


Fig. 5.
Source connectivity analysis from Patient 5. Two ROIs were determined from spike 1 (see **a**) and spike 2 (see **b**). The information flows indicated the primary source in the left temporal lobe. The identified primary source agreed with the clinically resected zone (see **c**). *L* left view

There are 2 temporal lobe epilepsy cases in our group. Both primary foci were identified.

Secondary or propagated regions include inferior frontal lobe and central region

Temporal Lobe Epilepsy

- TLE patients included in our group: In 20 ms, the source propagated to 1. inferior frontal region. 2. central Rolandic region
- Functional pathway between temporal lobe and Rolandic region? Bhardwaj et al. Childs Nerv Syst (2010) 26:185–190
- No DTI for these 2 TLE patients

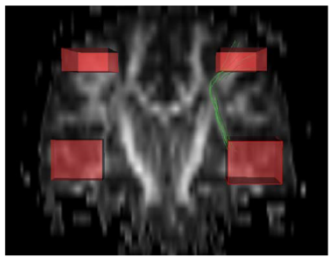


Fig. 3 The aberrant white matter tract travels through the external capsule. DTI tractography is performed on a patient with intractable epilepsy, and the white matter tract found on the lesional hemisphere is shown to pass through the medial aspect of the external capsule on a coronal fractional anisotropy map. The same neuroanatomical course, in the external capsule, is found in all cases of patients with intractable epilepsy. In contrast, identically sized and placed volumes of interest on the contralateral hemisphere do not reveal any such white matter tract

Bhardwaj et al. Childs Nerv Syst (2010) 26:185–190

Neocortical epilepsy

- Surgery Trend: temporal to extratemporal; adult to pediatric
- Extratemporal neocortical epilepsy is challenging
- Pattern of neocortical spike travel is different from that of the temporal spikes
- The neocortical seizure propagation varies depending on the lobe(s) of seizure onset (Dlugos and Sperling 2000)

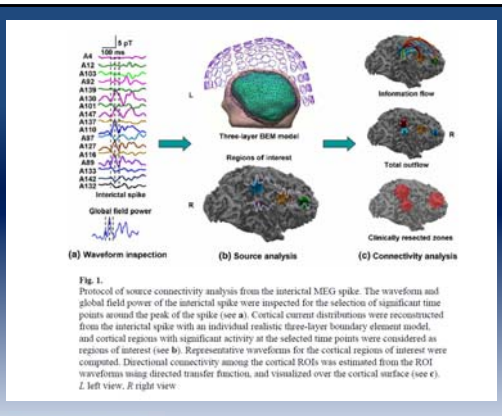
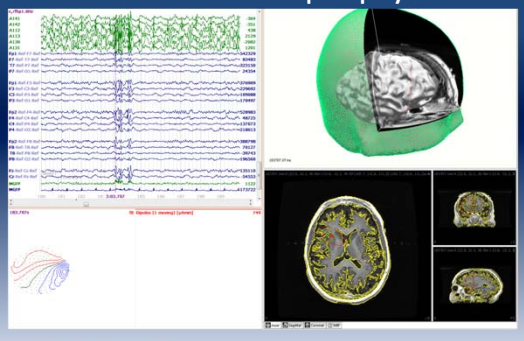


Fig. 1.
Protocol of source connectivity analysis from the interictal MEG spike. The waveform and global field power of the interictal spike were inspected for the selection of significant time points around the peak of the spike (see **a**). Cortical current distributions were reconstructed from the interictal spike with an individual realistic three-layer boundary element model, and cortical regions with significant activity at the selected time points were considered as regions of interest (see **b**). Representative waveforms for the cortical regions of interest were computed. Directional connectivity among the cortical ROIs was estimated from the ROI waveforms using directed transfer function, and visualized over the cortical surface (see **c**). *L* left view, *R* right view

Neocortical epilepsy

- In patient one, one spike identified 3 primary regions
- Maybe multiple regions active simultaneously
- Epileptogenic zones spread quickly?
- The independent epileptogenic zones interconnected through axonal tracts or cortical-cortical excitatory connectivity

Neocortical epilepsy



Neocortical epilepsy

- Diffusion Tensor Imaging (DTI)- tractography demonstrated fibers more connected on epileptogenic region than the same region contra-laterally using similar volumes of interest in this patient
- The abnormal tracts may have facilitated the patient's seizure
- Is this a phenomenon just in TSC patients or in other patients as well?
- Cortical-cortical propagation may co-exist too

Extratemporal neocortical

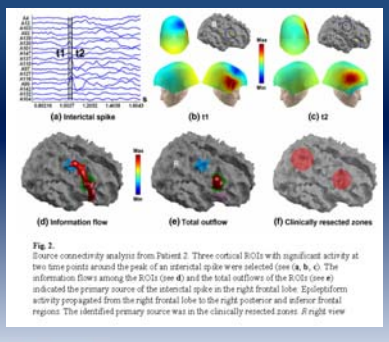


Fig. 2.
Source connectivity analysis from Patient 2. Three cortical ROIs with significant activity at two time points around the peak of an interictal spike were selected (see **a**, **b**, **c**). The information flows among the ROIs (see **d**) and the total outflows of the ROIs (see **e**) indicated the primary source of the interictal spike in the right frontal lobe. Epileptiform activity propagated from the right frontal lobe to the right posterior and inferior frontal regions. The identified primary source was in the clinically resected zones. *R* right view

Extra-temporal neocortical

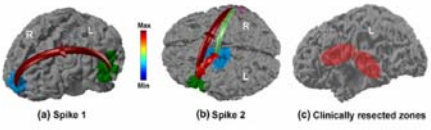


Fig. 4.
Source connectivity analysis from Patient 4. **a** Two ROIs were determined from spike 1 and the information flow indicated the primary source in the left temporal lobe. **b** Three ROIs were determined from spike 2 and the information flows indicated the primary source in the left frontal lobe. The identified primary sources agreed with the clinically resected zones (see **c**). *L* left view

- 23 ms between t1 and t2

Summary

- Source connectivity for interictal MEG spikes is feasible with directed transfer function analysis
- The source analysis identified the primary epileptogenic zones and the feature of their propagation
- eConnectome software is available for download at http://econnectome.umn.edu/
- More connectivity MEG/MSI study needed, especially neocortical epilepsy in conjunction with DTI tractography

Minnesota Epilepsy Group, P.A.

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Thanks for your attention!

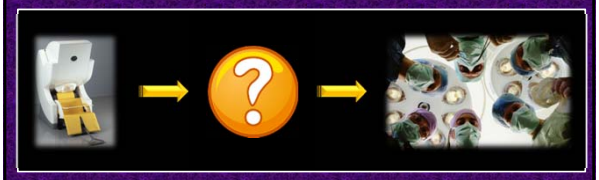
Minnesota Epilepsy Group, P.A.

Allina Health

Current Issues and Enduring Questions in Clinical MEG

MEG Results in the Operating Theater: How We Do It

Anto Bagic, Pittsburgh, PA



MEG Results In The Operating Theater: How We Do It

Atlanta, GA

Anto Bagić, MD, PhD
Chief, Epilepsy Division
Chief Scientific Advisor, MEG Research
Director, UPMC MEG Epilepsy Program
Associate Professor, Neurology & Neurosurgery
Director, University of Pittsburgh Comprehensive Epilepsy Center (UPCEC)

February 6, 2014

UPMC LIFECHANGINGMEDICINE

University Of Pittsburgh Comprehensive Epilepsy Center (UPCEC)
Epilepsy Division, DEPARTMENT OF NEUROLOGY

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- Unavoidable use of brand names for various products does not represent an endorsement or any preferential treatment.
- Sharing personal experience necessitates sharing a brand-specific procedures.
- No other specific personal disclosure pertaining to this presentation.

Outline

- Introduction: only 20 short years ago…
- Dizzy digital world (not all DICOMs are created equal?)
- Current reality: "all ingredients" ≠ a tasty meal
- Our institutional slippery road towards MEG merriment
- Implications on reporting – internal vs. external
- Flow of (external) MRIs and MSIs at UPMC
- Surgical planning: example in *Brainlab*
- Summary & Conclusions

In Only 20 Short Years...

Lancet **1997**;350(9078);657-60.

THE LANCET

Issues of imaging

Towards filmless and distance radiology

David M Hynes, Giles Stevenson, Claude Nahmias

To many people radiology is synonymous with films. For 20 years or so, however, it has been possible to capture digitally data traditionally displayed on film, and that was true from the beginning of computed tomography and magnetic resonance imaging too. There is more to picture archiving and communication systems (PACS) than the economies of filmlessness and the ability to modify images. To realise the full potential of PACS requires huge and expensively equipped networks linking the radiology department, hospital wards, outpatient clinics, laboratories, family doctors' clinics, and so on, permitting simultaneous consultations on different sites and almost instant reporting from specialist radiologists at a distance. The data sets that need to be transferred are huge but some of the technical obstacles are now being overcome and the past few years have seen some hospitals move to a filmless state. The more common pattern, though, will be a piecemeal approach. PACS and teleradiology certainly provide a quicker imaging service. How soon a total PACS will save money for a hospital operating budget is less clear.

Over the past 20 years radiology departments have witnessed a progressive shift from trapping an image on a photographic plate to capturing it with radiation detectors whose output signals can be digitised. Methods such as computed tomography (CT), ultrasound, nuclear medicine, and magnetic resonance imaging (MRI) are inherently digital because the images have been reconstructed by computers. In the more recently developed computed radiography[1,2] and digital videofluoroscopy[3] the signal that would previously have been used to expose a radiographic film is digitised. The advantages of digital acquisition are that all the data are preserved and the images can be modified—made lighter or darker, given more grey scale or less

and they frequently broke down, were only available in the X-ray department, and could not store enough data to replace the film file. Now many small systems dedicated to a single modality, such as ultrasound or CT,[4] have been developed successfully. Work on PACS systems that cover all digital imaging modalities in a radiology department is in progress,[5] and new terms are emerging to describe these—eg, electronic image management and image management and communication. An extension of digital radiology, and therefore of PACS, is teleradiology, which refers to the electronic transmission of images from one location to another using ordinary telecommunication links.[6]

"The components of any PACS system include the image

European Journal of Radiology, 17 (1993) 22–27

EURRAD 00410

Data fusion in medical imaging: merging multimodal and multipatient images, identification of structures and 3D display aspects

C. Barillot*, D. Lemoine, L. Le Briquer, F. Lachmann, B. Gibaud

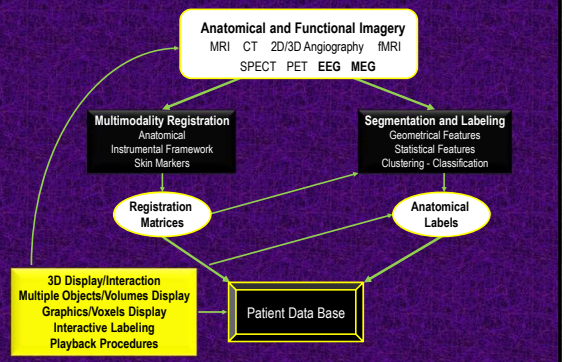
INSERM U335, Laboratoire SIM, Faculté de Médecine, Université de Rennes I, 35043 Rennes Cedex, France

Abstract

Data fusion in medical imaging can be seen into two ways (i) multisensors fusion of anatomical and functional information and (ii) interpatient data fusion by means of warping models. These two aspects set the methodological framework necessary to perform anatomical modelling especially when concerning the modelling of brain structures. The major relevance of the work presented here concerns the interpretation of multimodal 3D neuro-anatomical data bases. Three types of data fusion problems are considered in this paper. The first one concerns the problem of data combination which includes multimodal registration (multisensor fusion applied to CT, MRI, DSA, PET, SPECT, or MEG). In particular, the problem of warping patient data to an anatomical atlas is reviewed and a solution is proposed. The second problem of data fusion addressed in this paper is the identification of anatomical structures by means of image analysis methods. Two techniques have been developed. The first one deals with the analysis of image geometrical features to end up with the determination of a fuzzy mask to label the structure of interest. The second technique consits of labelling major cerebral structures by means of statistical image features associated with relaxation techniques. Finally, the paper presents a review of up to date 3D display techniques with a special emphasis on volume rendering and 3D display of combined data.

Key words: Images, processing; Images, analysis; Images, quality; PACS; Computers in radiology; Departmental management

Intra-individual Image Fusion



Barillot C, IEEE Trans Med Imaging. 2014 Jan 22-7.

MEG or MSI & PACS in MEDLINE

January 3, 2014

MEG & PACS	=>	5 hits/3 relevant (1993)
MSI & PACS	=>	0 hits
MEG & DICOM	=>	2 (2006 & 2009)
MSI & DICOM	=>	0

- Indexing problem?
- Terminological turmoil?
- Benign neglect?
- Irrelevance?

http://www.ncbi.nlm.nih.gov/pubmed, January 3, 2014.

Terminological Turmoil?

- "A direct **neurophysiologic technique**" vs. **imaging method**?
- "**Neuroimaging modalities**, such as functional magnetic resonance imaging (fMRI), magnetoencephalography (MEG), electroencephalography (EEG), and near infrared spectroscopy (NIRS), share similar application purposes, imaging protocol, analyzing methods, and data structure...." [Nakai et al. Magn Reson Med Sci. 2008;7(3):141-55.]
- Are **MEG** and **MSI** really synonyms?
- Are there implications for the field?

Progress In Image Fusion*

Table 1 Progress of image fusion based on different types of procedures showing the major advantages and limitations

Period	Class of registration procedure	Modalities	Major focus	Limitation
1 (early 1980s)	Prospective	PET, CT	Brain, tumour	Procedure involved, preparation
2 (mid-1980s)	Prospective	PET, stereotactic atlas	Brain, standardized anatomical localization	Not applicable to pathology
3 (late 1980s, early 1990s)	Retrospective (automated, interactive)	PET, SPECT, CT, MRI	Brain, body section (thorax, neck)	Difficult for whole-body in clinical environment
4 (mid-1990s, late 1990s)	Retrospective	PET (emission and transmission), CT, MRI	Whole-body	Limited resolution of PET transmission scan
5 (early 2000s)	Prospective	PET/CT, SPECT/CT	Whole-body	Not strictly simultaneous, prone to motion-induced artefacts, limited use for brain imaging
6 (late 2000s)	Prospective	PET/CT, PET/MR	Brain, whole-body	Not always strictly simultaneous (depending on technical solution), limited by patient motion, attenuation correction

"In conclusion, the modern combined PET/MR systems presently available have the potential to achieve almost optimal co-registration. Knowing the intrinsic limitations of such systems will facilitate future improvements in hardware and image processing".

Pietrzyk & Herzog, Magn Reson Mater Phy, 2013, 26:137–147.

Insights Imaging
DOI 10.1007/s13244-013-0296-y

ORIGINAL ARTICLE

Impact of cross-enterprise data sharing on portable media with decentralised upload of DICOM data into PACS

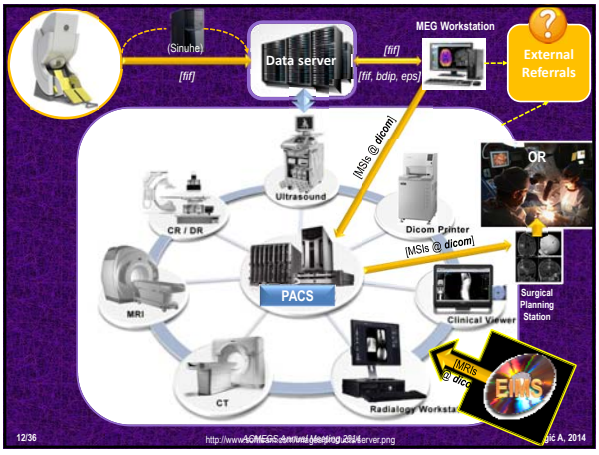
K. Y. E. Aryanto · R. van de Wetering · A. Broekema · Peter M. A. van Ooijen · M. Oudkerk

Key points

- Rapid assimilation of external imaging into a PACS system is essential.
- But data distribution using portable media also carries some disadvantages.
- A DICOM data uploader incorporates studies from portable media to hospital workflow.
- Automated media handling or XDS should solve the steadily growing storage problem.
- Software improvements will facilitate the steady increase in the amount of CDs processed.

...the amount of CDs processed.

...(DICOM) files can be read from compact disc (CD) on any workstation in the hospital, processed quickly to the



http://www.ACMEGS.com/images/server.png

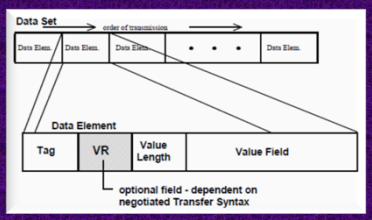
EIMS
(External Images Management System)

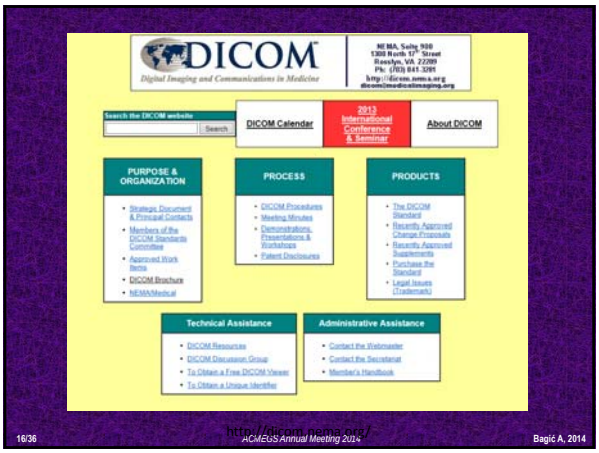
- Currently implemented at UPMC (Pittsburgh, PA)
- Locally developed software
- Any networked computer can be set to run it
- Authorized (via Active Directory authentication) users only
- Two Parts:
  - 1. Requesting Phase (May be done by many users)
  - 2. Filing Phase ("File Room" – done by the Radiology Support staff or other designated users) – interrogates and identifies files on a CD, proceeds with importing it after authentication
  - Takes any "native DICOM" images and imports in ClinicView

DICOM Wrapper

- Of the shelf or locally-developed solutions
- "Wraps" (DICOMizes) other types of files (images)
- "Wrap the image in a DICOM envelope and add important data that is required by the DICOM standard in order to enable all DICOM enabled applications to read and display the image correctly".*
- Examples: JPEGs (i.e. skin lesions, etc.), PDF (i.e. our PSM modalities, etc.), etc.

Illustration of DICOM element encoding in a DICOM data stream*




DICOM
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http://dicom.nema.org
dicom@medicalimaging.org
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DICOM Procedures
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Patent Disclosures
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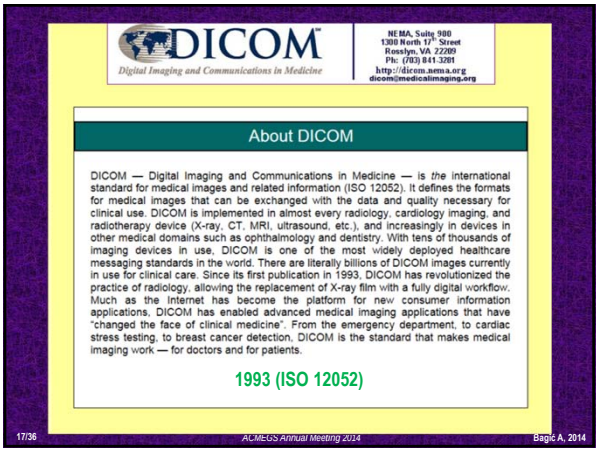
The DICOM Standard
Recently Approved Change Proposals
Recently Approved Supplements
Purchase the Standard
Legal Issues (Trademark)
Technical Assistance
DICOM Resources
DICOM Discussion Group
To Obtain a Free DICOM Viewer
To Obtain a Unique Identifier
Administrative Assistance
Contact the Webmaster
Contact the Secretariat
Member's Handbook
http://dicom.nema.org/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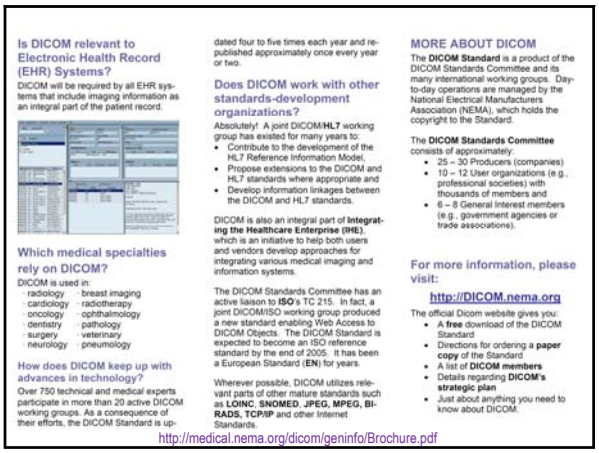
About DICOM
DICOM — Digital Imaging and Communications in Medicine — is the international standard for medical images and related information (ISO 12052). It defines the formats for medical images that can be exchanged with the data and quality necessary for clinical use. DICOM is implemented in almost every radiology, cardiology imaging, and radiotherapy device (X-ray, CT, MRI, ultrasound, etc.), and increasingly in devices in other medical domains such as ophthalmology and dentistry. With tens of thousands of imaging devices in use, DICOM is one of the most widely deployed healthcare messaging standards in the world. There are literally billions of DICOM images currently in use for clinical care. Since its first publication in 1993, DICOM has revolutionized the practice of radiology, allowing the replacement of X-ray film with a fully digital workflow. Much as the Internet has become the platform for new consumer information applications, DICOM has enabled advanced medical imaging applications that have "changed the face of clinical medicine". From the emergency department, to cardiac stress testing, to breast cancer detection, DICOM is the standard that makes medical imaging work — for doctors and for patients.
1993 (ISO 12052)


DICOM
Digital Imaging and Communications in Medicine
What is DICOM?
DICOM is a global Information-Technology standard that is used in virtually all hospitals worldwide. Its current structure, which was developed in 1993, is designed to ensure the interoperability of systems used to:
Produce, Store,
Display, Process,
Send, Retrieve,
Query or Print
medical images and derived structured documents as well as to manage related workflow.
Who needs DICOM?
Hospitals, clinics, imaging centers and specialists. By purchasing only equipment and information systems that conform to the DICOM Standard, you can ensure that these tools will work together to produce, manage and distribute your images regardless of your previous, current or future vendors.
Manufacturers of imaging equipment and imaging information systems. DICOM conformance ensures that every medical imaging facility is a potential customer, because your equipment can work with any workflow or electronic health record systems.
Manufacturers of peripheral equipment (e.g., film scanners, printers, computer monitors and workstations, image archives). DICOM conformance ensures that your products can work with all current or future imaging modalities and related peripheral equipment – regardless of vendor.
DICOM's purpose is to meet each of these diverse requirements.
Who Benefits from DICOM?
Physicians have better access to images and reports when DICOM standards are in place. This allows them to make a faster diagnosis, potentially from anywhere in the world.
Patients can potentially obtain faster and more effective care when the DICOM Standard is used to send their information through the healthcare enterprise.
Payers benefit from this faster and more effective process through potentially lowered cost of care.
WORKFLOW
http://medical.nema.org/dicom/geninfo/Brochure.pdf



Is DICOM relevant to Electronic Health Record (EHR) Systems?

DICOM will be required by all EHR systems that include imaging information as an integral part of the patient record.

Which medical specialties rely on DICOM?

DICOM is used in:

- radiology
- breast imaging
- cardiology
- radiotherapy
- oncology
- ophthalmology
- dentistry
- pathology
- surgery
- veterinary
- neurology
- pneumology

How does DICOM keep up with advances in technology?

Over 750 technical and medical experts participate in more than 20 active DICOM working groups. As a consequence of their efforts, the DICOM Standard is updated four to five times each year and re-published approximately once every year or two.

Does DICOM work with other standards-development organizations?

Absolutely! A joint DICOM/**HL7** working group has existed for many years to:

- Contribute to the development of the HL7 Reference Information Model.
- Propose extensions to the DICOM and HL7 standards where appropriate and
- Develop information linkages between the DICOM and HL7 standards.

DICOM is also an integral part of **Integrating the Healthcare Enterprise (IHE)**, which is an initiative to help both users and vendors develop approaches for integrating various medical imaging and information systems.

The DICOM Standards Committee has an active liaison to **ISO**'s TC 215. In fact, a joint DICOM/ISO working group produced a new standard enabling Web Access to DICOM Objects. The DICOM Standard is expected to become an ISO reference standard by the end of 2005. It has been a European Standard (**EN**) for years.

Wherever possible, DICOM utilizes relevant parts of other mature standards such as **LOINC**, **SNOMED**, **JPEG**, **MPEG**, **BI-RADS**, **TCP/IP** and other Internet Standards.

MORE ABOUT DICOM

The **DICOM Standard** is a product of the DICOM Standards Committee and its many international working groups. Day-to-day operations are managed by the National Electrical Manufacturers Association (NEMA), which holds the copyright to the Standard.

The **DICOM Standards Committee** consists of approximately:

- 25 – 30 Producers (companies)
- 10 – 12 User organizations (e.g., professional societies) with thousands of members and
- 6 – 8 General Interest members (e.g., government agencies or trade associations).

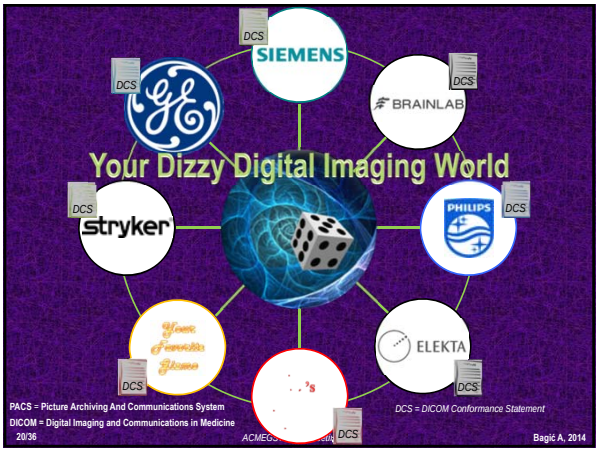
For more information, please visit:

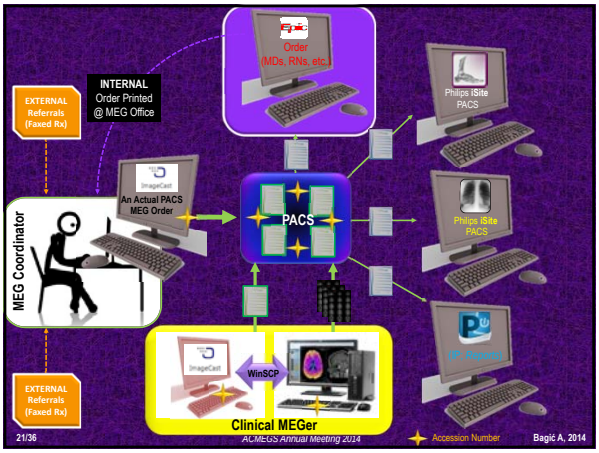
http://DICOM.nema.org

The official Dicom website gives you:

- A **free** download of the DICOM Standard
- Directions for ordering a **paper copy** of the Standard
- A list of **DICOM members**
- Details regarding **DICOM's strategic plan**
- Just about anything you need to know about DICOM.

http://medical.nema.org/dicom/geninfo/Brochure.pdf





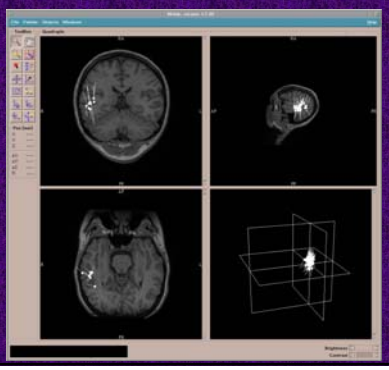
Recapitulation Of My Workflow

1. Complete MEG-EEG analysis (In my case using Neuromag software suite)
2. Push MSIs from **Mrilab** to PACS:
   1. "File => Print" => a pop up window "Print what, where and how?"
      1. **Viewer**: select a section being exported
      2. **Destination**: Export SELCTED (Stentor_SCP_PUH) (I can export also to CHP)
      3. **Size**: Constrain to square - SELECTED
      4. **Format**: Gray-scale, Image & LineArt, Binary – selected
   2. Click **Export** button at LLC => *Output Options* window pops up
   3. Select **Series** and TYPE IN DICOM Accession Number generated by ImageCast (DO NOT SELECT "Use DICOM overlay planes")
   4. Select **OK** at LLC
3. Finalize TYPING (dictating option exists!) report on an Office PC
4. Complete reporting in *ImageCast (a copy paste option used currently)*

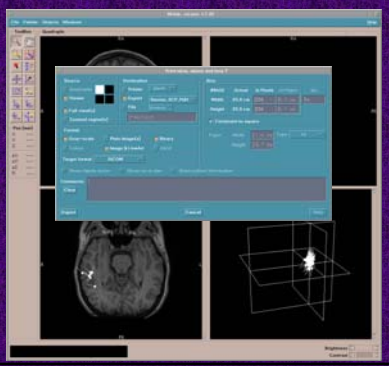
Outcome:
MSIs are available for exporting to any designated networked workstation as any other DICOM images...

PACS (Picture Archiving And Communications System) **DICOM (Digital Imaging and Communications in Medicine).**

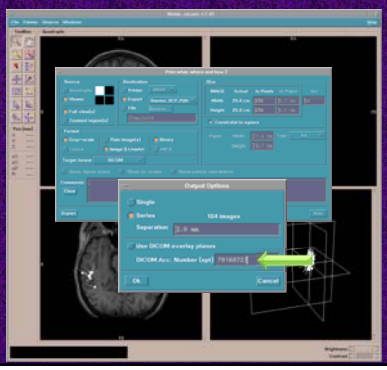
Exporting MSIs from Mrilab to PCAS



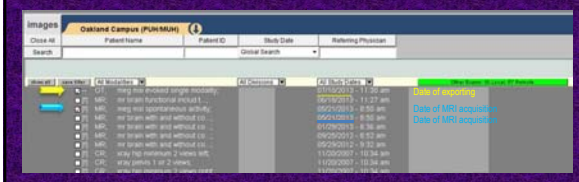
Exporting MSIs from Mrilab to PCAS



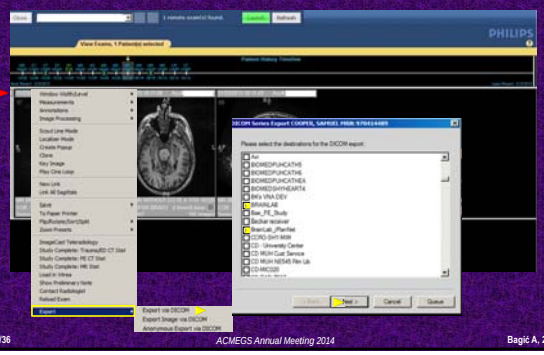
Exporting MSIs from Mrilab to PCAS



Some Intricacies Remain...



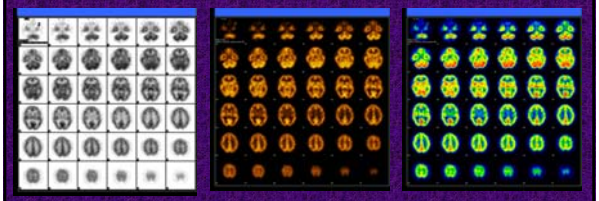
Example Of MSIs Exported to PACS



Example Of PSM Exported to PACS

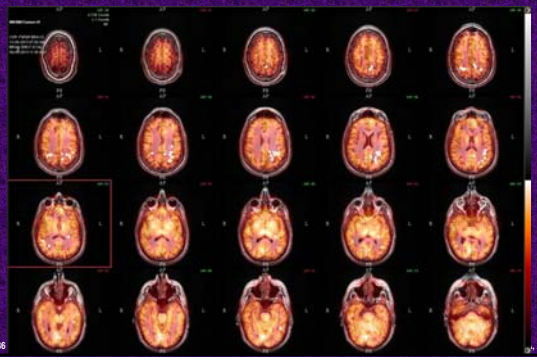
Example of Ictal SPECT

TV Inverse TV Gold TV Rainbow

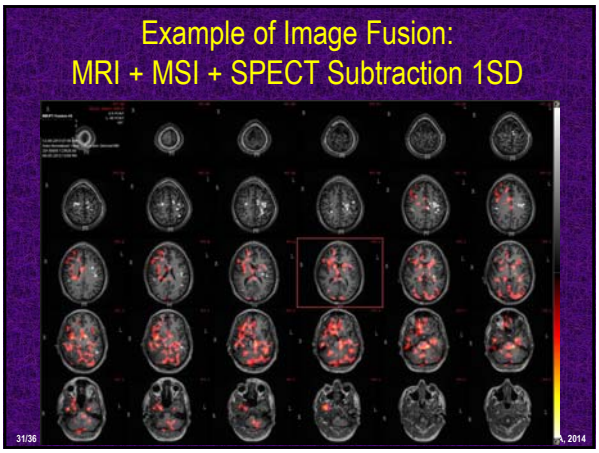


[Injection within 7 seconds of EEG onset]

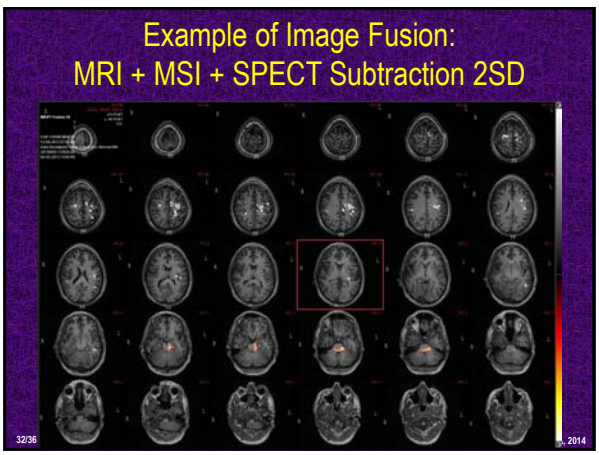
Example of Image Fusion: MRI + MSI + Ictal SPECT



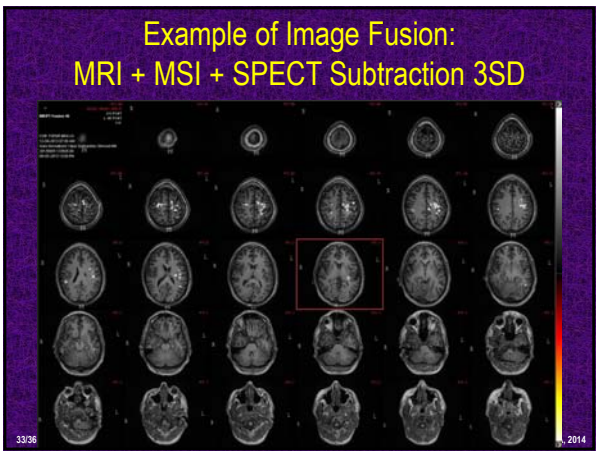
Example of Image Fusion: MRI + MSI + SPECT Subtraction 1SD



Example of Image Fusion: MRI + MSI + SPECT Subtraction 2SD



Example of Image Fusion: MRI + MSI + SPECT Subtraction 3SD



Summary & Conclusions (1/2)

- Rapid progress towards filmless and remote radiology opened many new possibilities in an effective and creative use of imaging in clinical practice.
- Inspired researchers eliminated cardinal obstacles in multimodal image integration.
- "Multiple solutions" do not translate into a streamlined logistics for an easy integration of MEG/MSIs into PACS.
- Technology *per se* is not an obstacle in most institutions and instances.
- Enormous amount of energy and time is wasted unnecessary on bringing the players to the table and making sure that everybody truly hears the same and commits.

Summary & Conclusions (2/2)

- Device vendors appear sub optimally disposed and "it is not that rare" that the devices produced by the same vendor don't communicate seamlessly.
- Currently, in most places, invested MEG clinicians have to facilitate focused team efforts on eliminating fatal "remaining trivialities".
- Your reasonable understanding of the big picture, and flexible persistence of forging a productive working relationship with hopefully equally flexible and enthusiastic local radiology staff coupled with a favorable IT environment remains necessary.
- At this point, there is no objective impassable obstacles for the complete and practical routine PACS integration of MSIs with all positive implications.
- This is one of the critical steps for further acceptance of MEG as a routine clinical tool among neurosurgeons that is necessary for the clinical MEG field's survival and advancement.

Most Sincere Thanks To

Michaela N. Lionetti RT(R)(M), CIIP

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Matti Leiniö
Terri Martin
Antti Telio
Matti Kajola

Tao Song, PhD

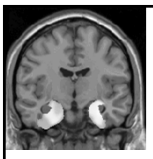
Many others who got stress ulcers on this since 2006 on both sides of Atlantic!

Towards a New Biomarker in Dementia

Why and What Biomarkers are Ideally Needed

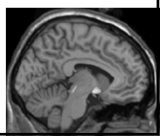
Jim Becker, Pittsburgh, PA

HIV disease includes a set of conditions referred to as HIV-Associated Neurocognitive Disorder (HAND); even a mild form of HAND can result in significant alterations in employment, medication adherence, driving ability and other aspects of daily life. Identifying the earliest indications of neuropathology is critical for the development of therapies. Unfortunately, there is no acknowledged neuroimaging biomarker that can identify the pathological substrate of HAND; the identification and differential diagnosis of HAND is limited to the clinical signs and symptoms. Our research team has been exploring the relative merits of magnetoencephalography (MEG) as a potential HAND biomarker, because it measures neuronal activity directly from the magnetic fields induced by neuronal currents. MEG does not rely on the blood-oxygen level dependent response to generate responses, and has the best tradeoff between spatial and temporal resolution of any current neuroimaging technology. MEG can identify individuals with HIV Disease, the MEG responses change with recovery from HIV-Associated Dementia, and the findings are stable over 6-months. Because MEG directly measures the activity of neuronal populations, it provides unique information regarding the pathophysiology of HAND that cannot be obtained from other neuroimaging modalities. Consequently, MEG should detect brain functional abnormalities prior to clinical symptomatology.



Brain Structure, Cognitive Function and HIV Disease

James T. Becker, Ph.D.
University of Pittsburgh
Multicenter AIDS Cohort Study



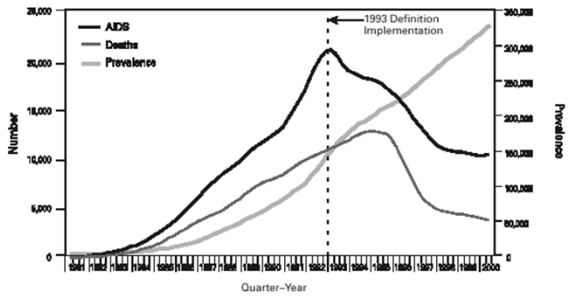
Conclusions

- In areas of the world with access to medical resources, the face of the epidemic of HIV Disease is changing.
- Among patients with appropriate medical care, factors other than HIV Disease are at least as important in determining the state of their brain health.
- Imaging biomarkers may provide an avenue to identify CNS dysfunction prior to the development of HIV-associated cognitive dysfunction.

HIV Disease – The First Report

- **June 1981**
  ***Pneumocystis* pneumonia—Los Angeles. *MMWR* 1981;30:250–252**
- From October 1980 through May 1981, 5 homosexual men, who did not know each other and had no known common contacts, were treated for *Pneumocystis carinii* pneumonia (PCP) in Los Angeles. All 5 patients had previous or current cytomegalovirus infection and candidal mucosal infection.

FIGURE 1. Estimated AIDS incidence*, deaths, and prevalence, by quarter-year of diagnosis/death — United States, 1981–2000



* Adjusted for reporting delays.

The Multicenter AIDS Cohort Study (MACS)

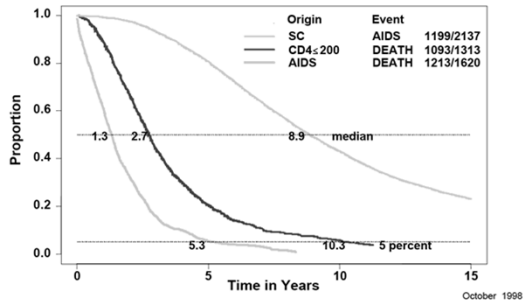
- The Multicenter AIDS Cohort Study (MACS) is a four-site study of the natural and treated history of HIV infection among gay/bisexual men.
- Study participants were enrolled in three waves: 1984/85, 1987/90, and 2001/03. The MACS has tracked cognitive test performance among the study participants since 1984 using screening tools, and a sub-cohort has been followed with more detailed testing for 25 years.

MACS Cohort(s)

- Enrolled men at 4 study sites: Baltimore/Washington, Chicago, Los Angeles, and Pittsburgh.
- A total of 6972 men have been enrolled since April 1984 (4954 in 1984–1985, 668 in 1987–1991, and 1350 in 2001–2003).
- Cohort 1 was the original sample of 4,954 men, and Cohort 2 was a “New Recruit Cohort” that focused on enrolling minority and special target groups such as the partners of the men in C1. Cohort 3 enrollment took place between October 2001 and August 2003 again focusing on recruiting racial/ethnic minorities as well as a special target group of uninfected men who had been censored from C1 in 1995.

Progression of HIV-1 Infection Prior to Potent Antiretroviral Therapy

Muñoz, Xu. Stat Med 1996; Enger et al. JAMA 1996; Jacobson et al. AJE 1993 (update)

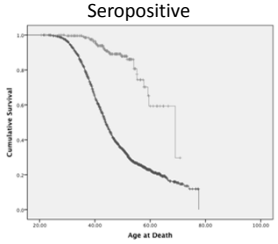


October 1998

Age at Death as a Function of HIV Serostatus and Recruitment Cohort

Seronegative

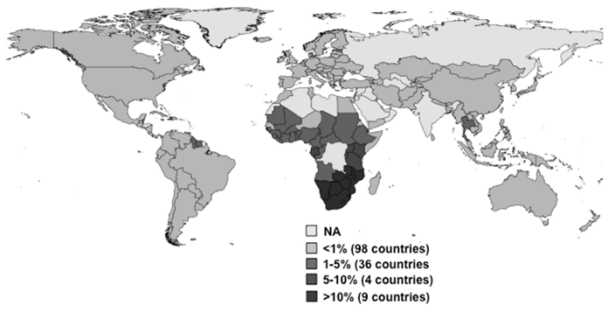
Seropositive



Median Age at Death:		
Cohorts 1 & 2 :	81.8 (78-85)	43.9 (43-44)
Cohort 3:	71.7 (67-76)	69.1 (55-83)

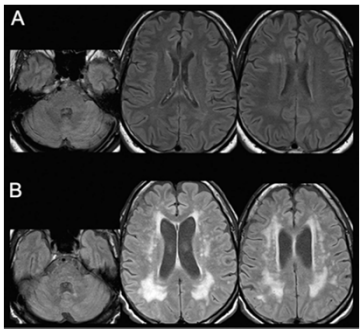
Adult HIV Prevalence Rate, 2011

Global HIV Prevalence Rate = 0.8%



NOTES: Data are estimates. Prevalence rates include adults ages 15-49. The estimate for Sudan represents data for South Sudan. An estimate was provided for Sudan and is <1%.
SOURCE: Kaiser Family Foundation, www.GlobalHealthFacts.org, based on UNAIDS, Report on the Global AIDS Epidemic; 2012.

HIV Encephalitis is Uncommon



Research Nosology for HIV-Associated Cognitive Disorders

Table Revised research criteria for HIV-associated neurocognitive disorders (HAND) (modified from HIV Neurobehavioral Research Center criteria[18])

HIV-associated asymptomatic neurocognitive impairment (ANI)*

1. Acquired impairment in cognitive functioning, involving at least two ability domains, documented by performance of at least 1, 0 SD below the mean for age-education-appropriate norms on standardized neuropsychological tests. The neuropsychological assessment must survey at least the following abilities: verbal/language, attention/working memory, abstraction/executive, memory (learning, recall), speed of information processing, sensory-perceptual, motor skills.
2. The cognitive impairment does not interfere with everyday functioning.
3. The cognitive impairment does not meet criteria for delirium or dementia.
4. There is no evidence of another preexisting cause for the ANI.†

*If there is a prior diagnosis of ANI, but currently the individual does not meet criteria, the diagnosis of ANI in remission can be made.

†If the individual with suspected ANI also satisfies criteria for a major depressive episode or substance dependence, the diagnosis of ANI should be deferred to a subsequent examination conducted at a time when the major depression has remitted or at least 1 month after cessation of substance use.

HIV-1–associated mild neurocognitive disorder (MND)*

1. Acquired impairment in cognitive functioning, involving at least two ability domains, documented by performance of at least 1, 0 SD below the mean for age-education-appropriate norms on standardized neuropsychological tests. The neuropsychological assessment must survey at least the following abilities: verbal/language, attention/working memory, abstraction/executive, memory (learning, recall), speed of information processing, sensory-perceptual, motor skills.

   Typically, this would correspond to an MSK scale stage of 0.5 to 1.0.
2. The cognitive impairment produces at least mild interference in daily functioning (at least one of the following):
   a) Self-report of reduced mental acuity, inefficiency in work, homemaking, or social functioning.
   b) Observation by knowledgeable others that the individual has undergone at least mild decline in mental acuity with resultant inefficiency in work, homemaking, or social functioning.
3. The cognitive impairment does not meet criteria for delirium or dementia.
4. There is no evidence of another preexisting cause for the MND.†

*If there is a prior diagnosis of MND, but currently the individual does not meet criteria, the diagnosis of MND in remission can be made.

†If the individual with suspected MND also satisfies criteria for a severe episode of major depression with significant functional limitations or psychotic features, or substance dependence, the diagnosis of MND should be deferred to a subsequent examination conducted at a time when the major depression has remitted or at least 1 month after cessation of substance use.

HIV-1–associated dementia (HAD)*

1. Marked acquired impairment in cognitive functioning, involving at least two ability domains, typically the impairment is in multiple domains, especially in learning of new information, attention/working memory, abstraction/executive, memory (learning, recall), speed of information processing, and motor skills, documented by neuropsychological testing with at least two domains 2 SD or greater than demographically corrected means. Note that where neuropsychological testing is not available, standard neurological evaluation and simple bedside testing may be used, but this should be done as indicated in algorithm, see below).

   Typically, this would correspond to an MSK scale stage of 2.0 or greater.
2. The cognitive impairment produces marked interference with day-to-day functioning (work, home life, social activities).
3. The pattern of cognitive impairment does not meet criteria for delirium (e.g., clouding of consciousness is not a prominent feature), or, if delirium is present, criteria for dementia need to have been met on a prior examination when delirium was not present.
4. There is no evidence of another, preexisting cause for the dementia (e.g., other CNS infection, CNS neoplasm, cerebrovascular disease, preexisting neurologic disease, or severe substance abuse compatible with CNS disorder).†

*If there is a prior diagnosis of HAD, but currently the individual does not meet criteria, the diagnosis of HAD in remission can be made.

†If the individual with suspected HAD also satisfies criteria for a severe episode of major depression with significant functional limitations or psychotic features, or substance dependence, the diagnosis of HAD should be deferred to a subsequent examination conducted at a time when the major depression has remitted or at least 1 month has elapsed following cessation of substance use. Note that the consensus was that even when major depression and HAD occurred together, there is little evidence that pseudodementia exists and the cognitive deficits do not generally improve with treatment of depression.

CNS Abnormalities Persist in the Era of HAART

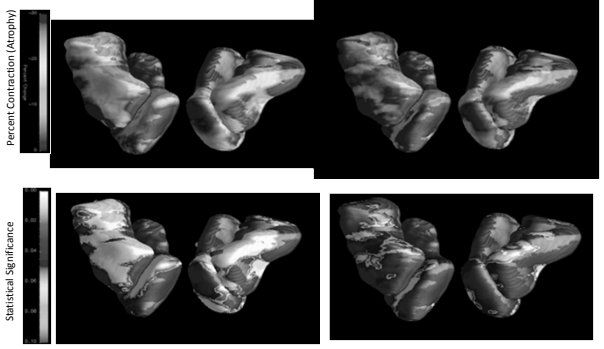
- Among patients with appropriate medical care, factors other than HIV Disease are at least as important in determining the state of their brain health.
- Elevated risk of all neurological diagnoses in HIV Disease.
- Subcortical *and* cortical tissue loss.
- Effects are independent of age.
- Independent effects of CVD and lung function on brain and cognition.
- Can alter cognitive test performance.

3-D Surface Maps of Basal Ganglia from MACS MRI Study

A

Effects of HIV Serostatus

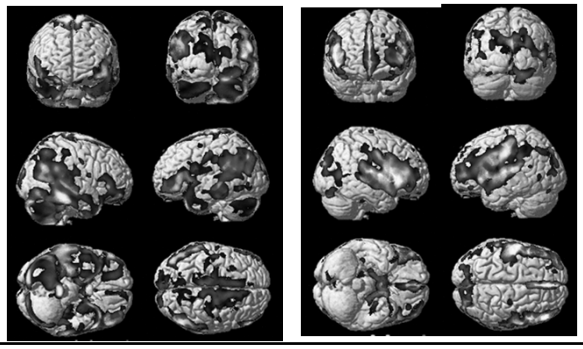
Effects of Time Since Seropositivity



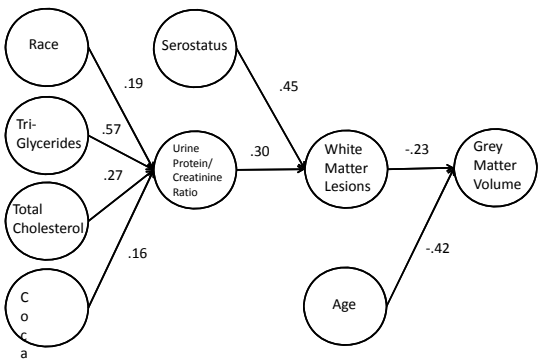
Surface Rendering of the Independent Effects of HIV Serostatus and Age on Grey Matter Volume

HIV Serostatus

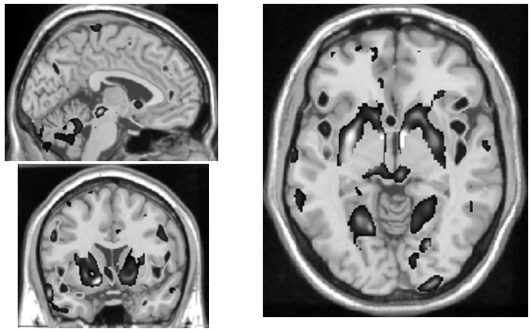
Age



Predictors of Grey Matter Volume



Lung Function, HIV Status and Brain Structure (Morris, et al.)



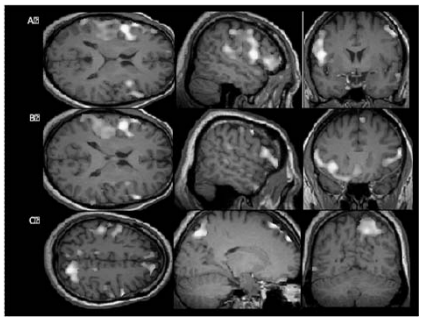
Cardiovascular Risk Factors and Cognition in HIV Disease

	Psychomotor Speed	Delayed Recall
Race	4.8, (3.13 – 7.34)	3.0, (1.98 – 4.49)
Depression	2.0, (1.28 – 3.27)	1.5, (.95 – 2.32)
Education	0.52, (.34 - .79)	.41, (.274 - .618)
Carotid IMT	1.7, (1.08 – 2.74)	
GFR	1.6, (1.00 – 2.66)	
Glucose	.66, (.44 – .99)	
Coronary Calcium		2.3, (.921 – 5.61)
HIV Serostatus	.78, (.51 – 1.20)	.59 (.381 - .903)
HIV Viral Load		2.1, (1.30 – 3.45)

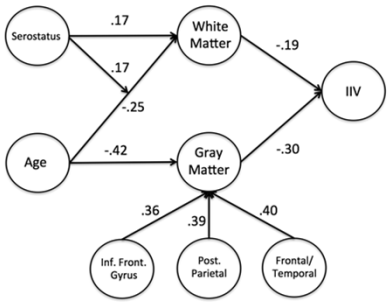
Intra-Individual Variability in HIV Disease (Hines, et al.)

- Focus on the study of *within-person* variability in cognitive functioning, otherwise termed intra-individual variability (IIV).
- Within-person differences in test performance observed across tasks at a single time point (dispersion), or on a single task across multiple time points (inconsistency).
- We examined *dispersion* in 147 MACS participants with MRI scans.

Regional Brain Atrophy Associated with Intra-Individual Variability



Intra-Individual Variability in HIV Disease



Regional Cerebral Blood Flow Measured with Arterial Spin Labeling MRI

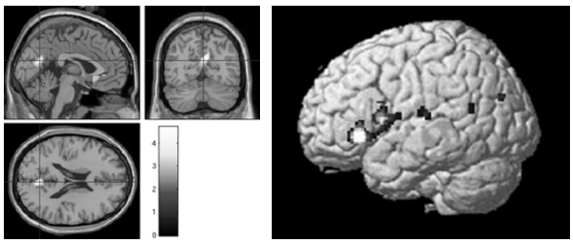
Drug Use Increases

HIV Increases

Cerebrovascular Reactivity to 5% CO_2 (relative to room air)

Age Decreases

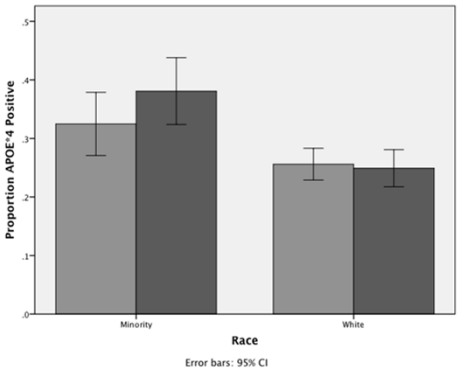
Drug Use Increases



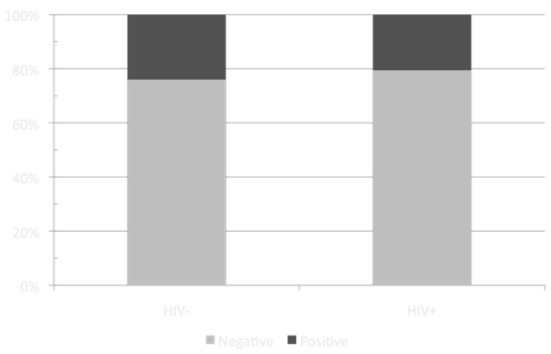
With Increased Survival Comes Older Average Age

- Concerns about prevalence of APOE ε4 allele
- Concerns about increased rate of β-amyloid deposition.
- The good news, so far:
  - Don't worry, they're not issues.

APOE*4 Allele Frequency in 2846 MACS Participants



Global PiB Retention by Serostatus



Summary

- Elevated risk of all neurological diagnoses in HIV Disease.
- Subcortical *and* cortical tissue loss.
- Effects are independent of age.
- Independent effects of CVD and lung function on brain and cognition.
- Mediated pathways from HIV Disease to non-specific brain alterations and increased IIV.
- No increase in rate of APOE ε4 allele.
- No increase in rate of PiB retention.

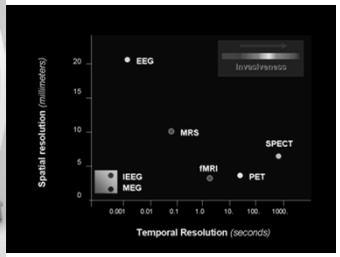
Summary

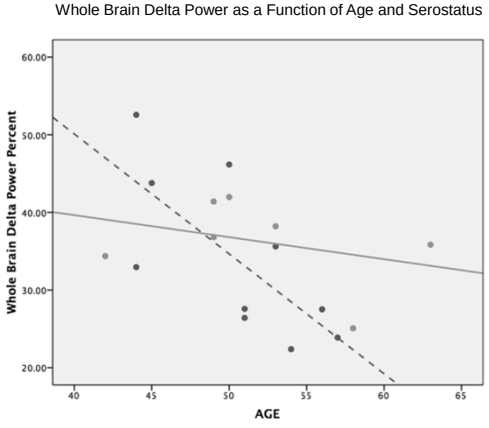
- Abnormalities in brain structure and function persist in the era of HAART.
- What remains unclear is the extent to which this is a function of prior experience with uncontrolled viral replication, or whether these abnormalities put the individual at increased *clinical* risk for subsequent expression of other neurodegenerative diseases (i.e., reduction of brain reserve).

Biomarkers for HAND

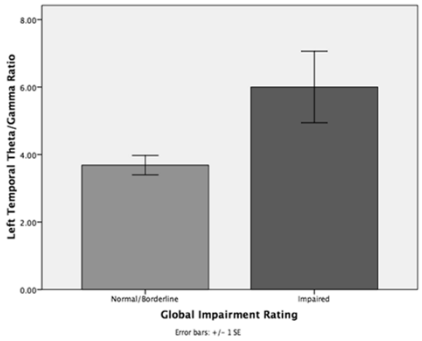
- "current approaches to classification and diagnosis of this [CNS] dysfunction rely on syndromic definitions or measures of abnormality on neuropsychological testing in the background context of HIV-1 infection." Thus, "supplanting or augmenting these approaches with objective biologic measurements related to underlying disease processes would provide a major advance in classification, diagnosis, epidemiology, and treatment assessment". (Price, et al., 2007)
- We need to consider that structure follows function follows pathology.
- We may want to consider alternative methods to our current imaging technologies for identifying the earliest phases of HIV-related changes in the brain.

Magnetoencephalography (MEG)

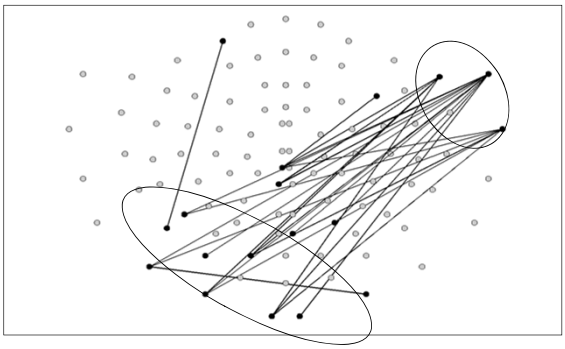




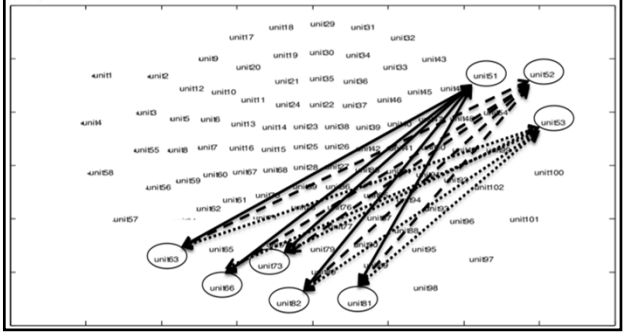
Theta:Gamma Ratio as a Function of Global Impairment Rating



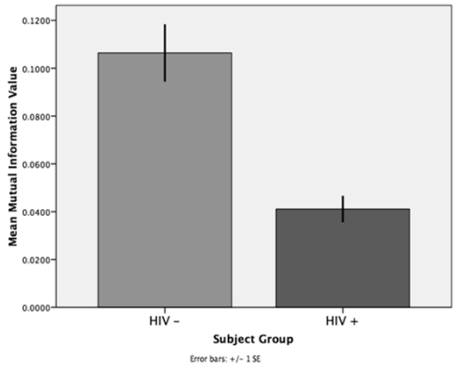
Focusing on two groups of sensors, where we found significant statistical differences



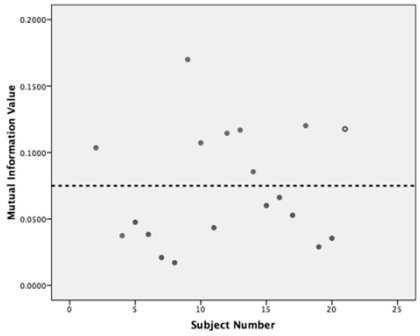
Functional Neuronal Network that Distinguished Serostatus



Mutual Information Values from Connectivity Analysis of Resting MEG



MI Values by Participant and Serostatus



Medical History

- 52 year old, right-handed black man
- HIV+ in October 2009 after diagnosis of PCP
- September 2009 first went to hospital with pneumonia
  - Fired from job
  - Became homeless
  - Not on treatment when came to Pittsburgh in March 2010
  - First seen at PACT 3/4/2010

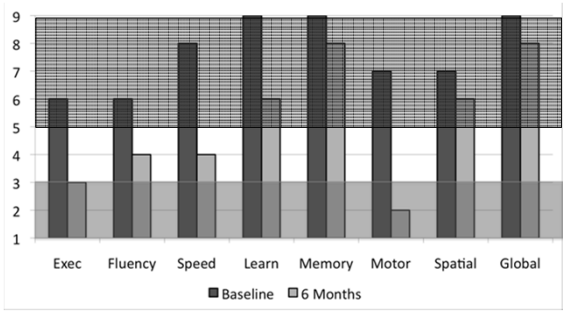
Medical History, Con't

- Initial diagnoses in March 2010 included:
  - PCP
  - Peripheral Neuropathy
  - HIV encephalopathy
  - Brain lesions secondary to PML, toxo or crypto
    - MRI scan read as normal for age
  - Diagnosed with HAD 4/21/2010
    - Enrolled in MEG study in May 2010

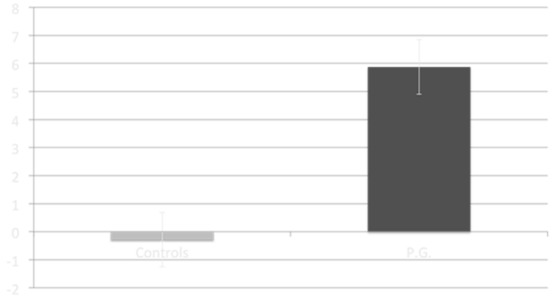
HIV Lab Values

	Visit		
Measure	March 2010	May 2010	September 2010
CD4+ Cell Count	12	232	256
Log10 HIV RNA	5.81	2.62	<50
CD4 Nadir	12		
RNA Peak	5.81		

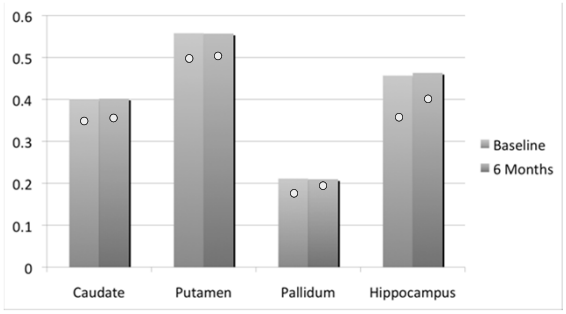
Changes in Neuropsychological Test Performance During Acute Recovery



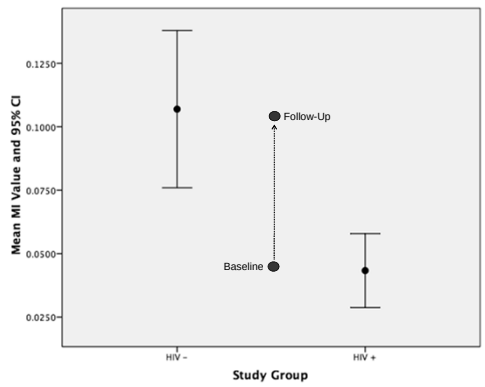
Percent Brain Volume Change During Recovery - *SIENA*



Changes in Regional Brain Volumes

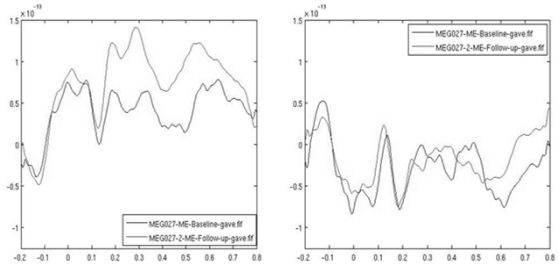


Change in MEG Identified Network Connectivity in P.G.



Temporal lobe

Left Hemisphere **Right Hemisphere**



Summary

- MEG is reliable and stable in the absence of clinical change
- Memory task is reliable and stable in the absence of clinical change.
- Resting State MEG signal shows mild changes related to HIV serostatus
  - But, shows larger and more pervasive links to cognitive status

Summary

- Functional connectivity analysis using mutual information reveals a network of connections that are significantly linked to HIV status, but not to cognitive functions
- The network shows recovery of function with effective HAART in a single case.
- MEG appears to meet important prerequisites to serve as a biomarker for HAND

Conclusions

- In areas of the world with access to medical resources, the face of the epidemic of HIV Disease is changing.
- Among patients with appropriate medical care, factors other than HIV Disease are at least as important in determining the state of their brain health.
- Imaging biomarkers may provide an avenue to identify CNS dysfunction prior to the development of HIV-associated cognitive dysfunction.

Thank you very much.

Towards a New Biomarker in Dementia

First Results of the Multi Center MAGIC AD Study

Fernando Maestu, Madrid, ES

In the last years, MEG field is experienced tremendous advances in its new clinical aplications. Dementia is one of those where greater advances are taking place, especially in Alzheimer´s Disease (AD). In fact functional connectivity measures are being testing AD as a dysconection syndromes. Thus, in the early stages of the disease Mild Cognitive Impairment patients showed increased synchronization and those that developed dementia showed higher synchronization than those that did not develop dementia. Correlations with anatomical conectivity and amyloidosis has been found as well. Despite of all these scientific evidence it was needed an international blind study. In an international multicenter study, we used magnetoencephalography and functional connectivity metrics to evaluate the ability to differentiate Mild Cognitive Impairment (MCI) from normal aging at the individual level. Data mining techniques were using for extracting features (links) to classify participants as MCI or controls using samples of already known patients and controls (learning stage) and from unseen data from five different centers. We identified a pattern of neuronal hypersynchronization; the features of the network that best discriminated MCI were fronto-parietal and interhemispheric. When this model was tested in an unseen sample the sensitivity was 1.00, specificity of .69 and overall total accuracy of .83. We report here the first use of neuronal functional connectivity data to discriminate between MCI patients and healthy elderly subjects at the individual level. The hypersynchronization pattern found in the MCI patients may be considered an early sign of synaptic disruption and a possible preclinical biomarker for MCI/AD.

First Results of the Multi-Center MAGIC-AD Study

CTB Center For Biomedical Technology

MEG!!

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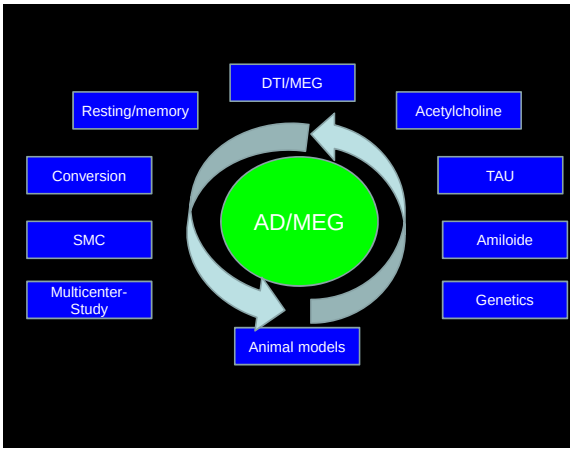
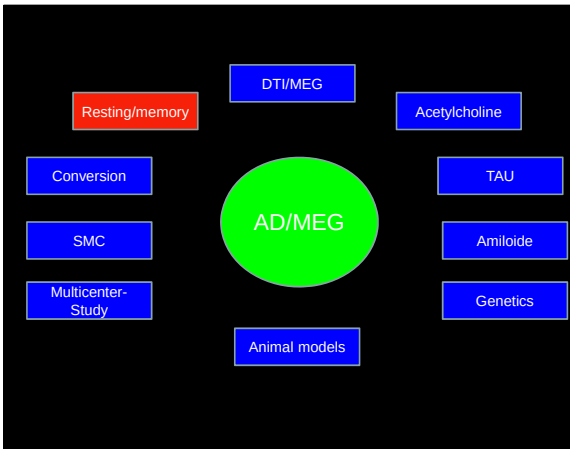
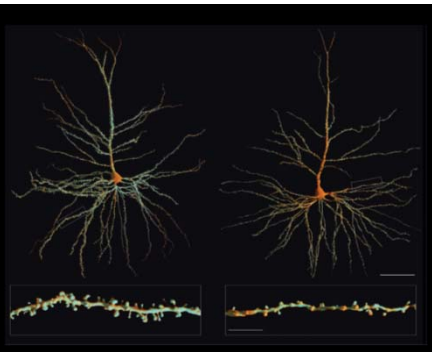
Laboratory of Cognitive and Computational Neuroscience
(Center for Biomedical Technology) UPM-UCM





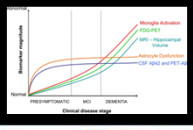
Functional connectivity in Mild Cognitive Impairment: Evaluating the Disconnection Hypothesis

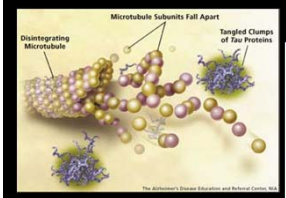
Bajo R, et al Journal of Alzheimer Disease , 2010
Bajo R et al, Age, 2011
Buldu J, et al, PLOSone, 2011
Bajo R et al, Brain Connectivity, 2012

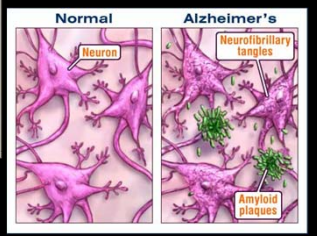


Morrison JH and Baxter M, Nat Rev Neurosci, 2012

Disconexion Hypothesis



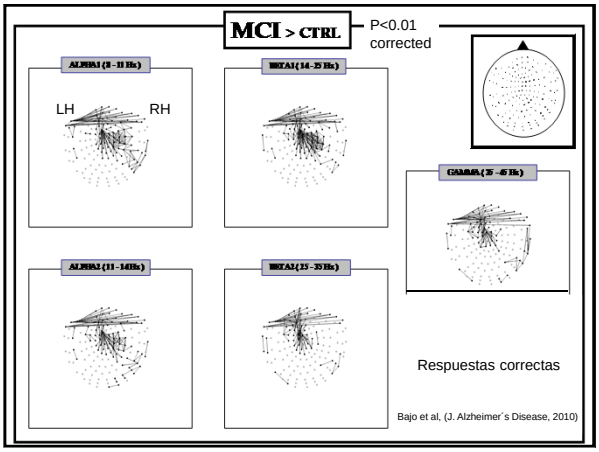


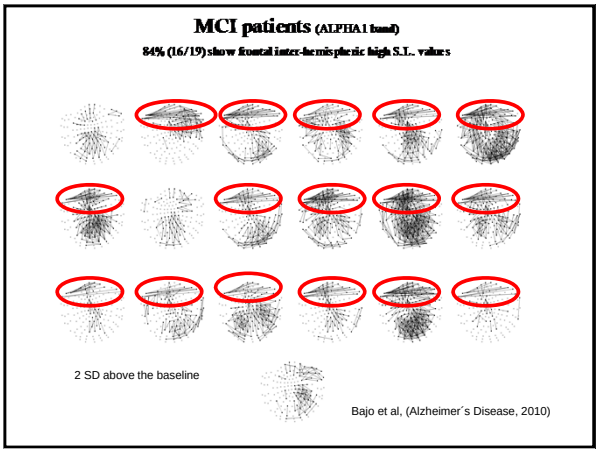


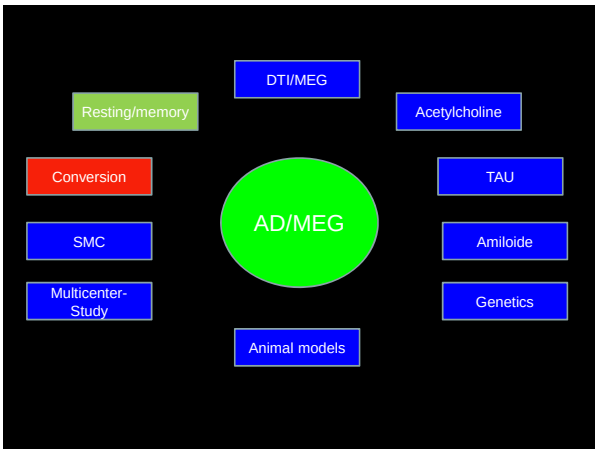
Journal of Alzheimer's Disease 22 (2010) 183–193
DOI 10.3233/JAD-2010-100177
IOS Press

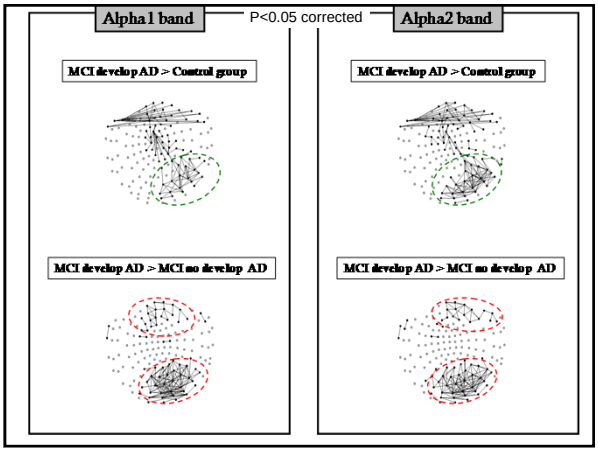
Functional Connectivity in Mild Cognitive Impairment During a Memory Task: Implications for the Disconnection Hypothesis

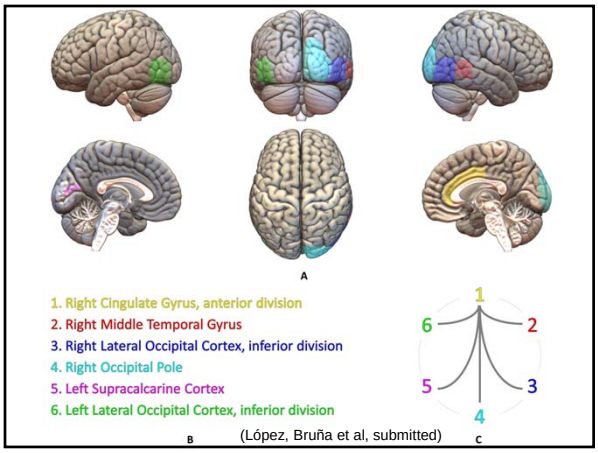
Ricardo Bajo[a,1,*], Fernando Maestú[a,b,1], Angel Nevado[a,b], Miguel Sancho[c], Ricardo Gutiérrez[a], Pablo Campo[a], Nazareth P. Castellanos[a], Pedro Gil[d], Stephan Moratti[a,e], Ernesto Pereda[f] and Francisco del-Pozo[a]



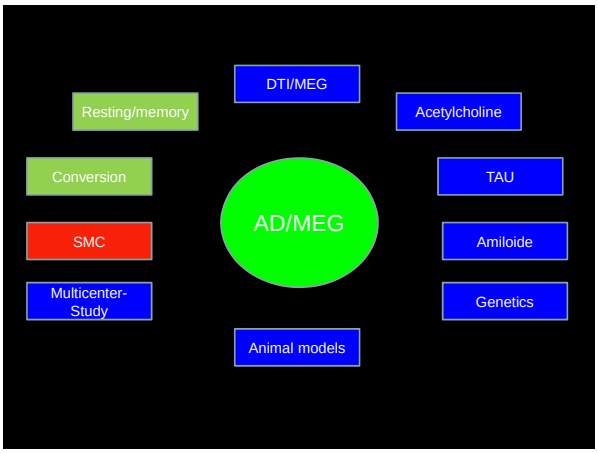


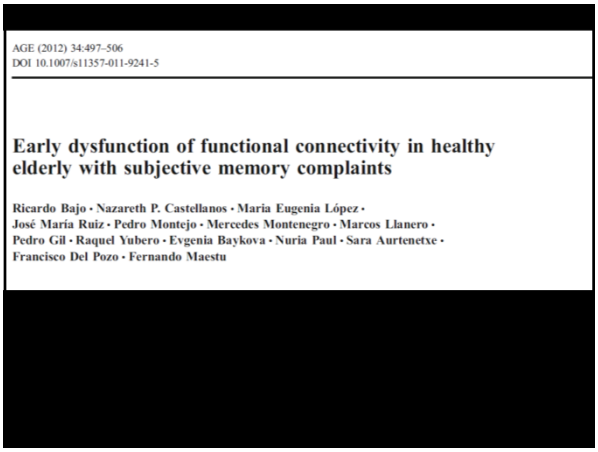






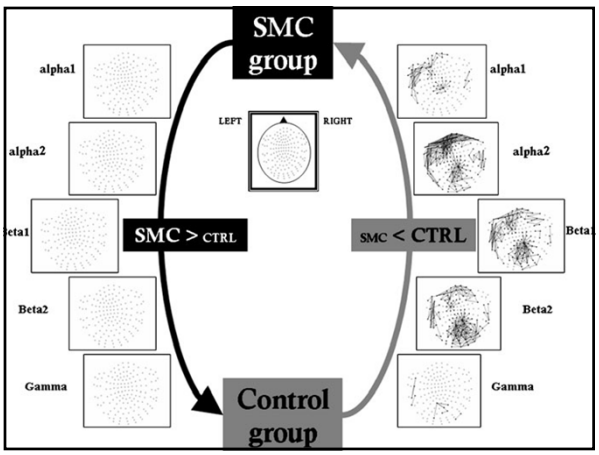
(López, Bruña et al, submitted)

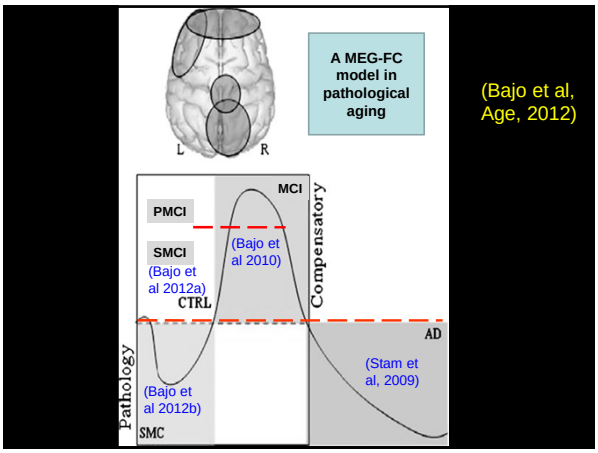



AGE (2012) 34:497–506
DOI 10.1007/s11357-011-9241-5

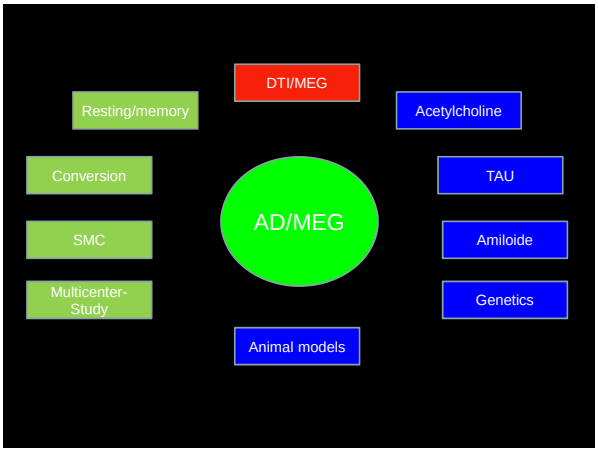
Early dysfunction of functional connectivity in healthy elderly with subjective memory complaints

Ricardo Bajo · Nazareth P. Castellanos · Maria Eugenia López · José María Ruiz · Pedro Montejo · Mercedes Montenegro · Marcos Llanero · Pedro Gil · Raquel Yubero · Evgenia Baykova · Nuria Paul · Sara Aurtenetxe · Francisco Del Pozo · Fernando Maestu





(Bajo et al, Age, 2012)



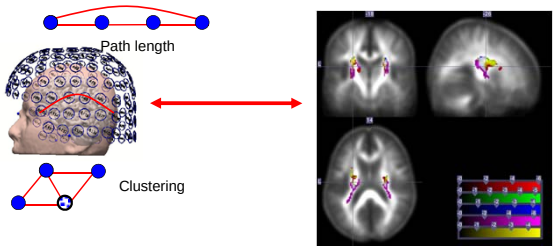
ANATOMO-FUNCTIONAL CONNECTIVITY :

A MEG/DTI STUDY USING GRAPH THEORY ANALYSIS

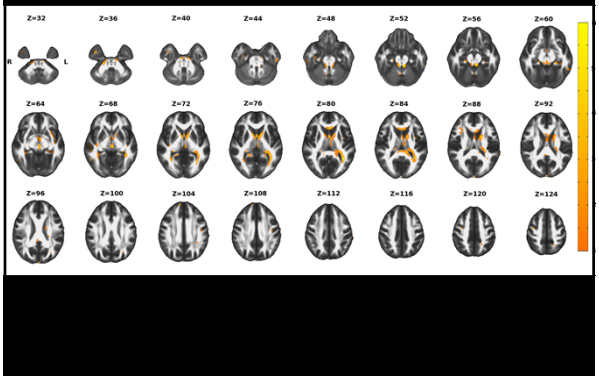
(Pineda et al, submitted)

Graph theory:MEG/DTI

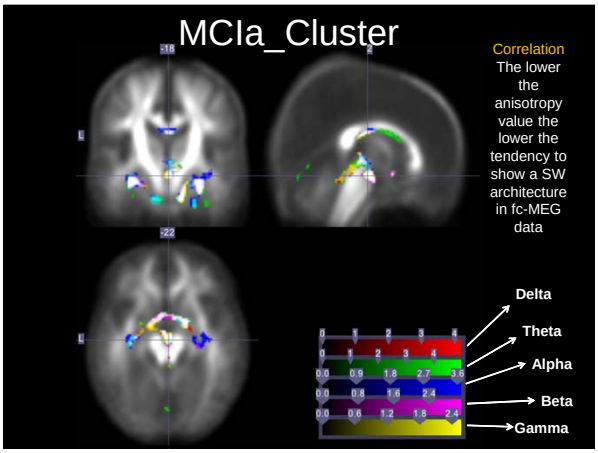
- **Combination of anatomo functional connectivity**
- **Is the functional connectivity architecture depending on the integrity of the white matter?**

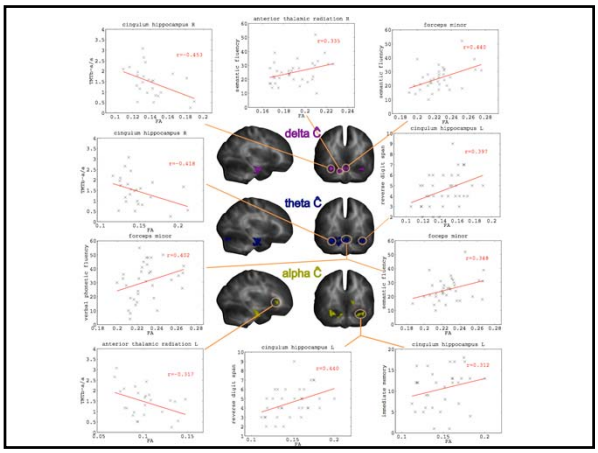


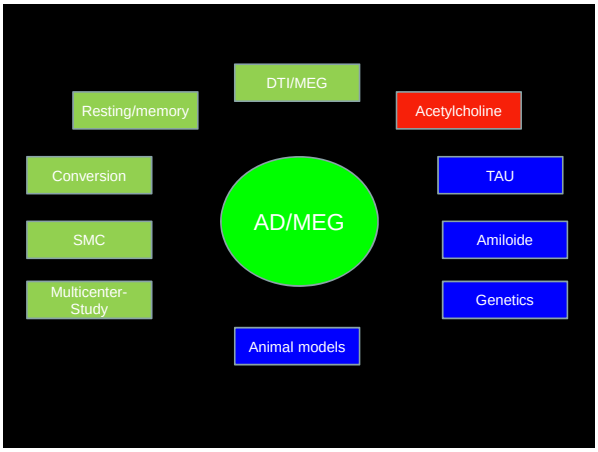
White Matter Integrity HA > MCIa

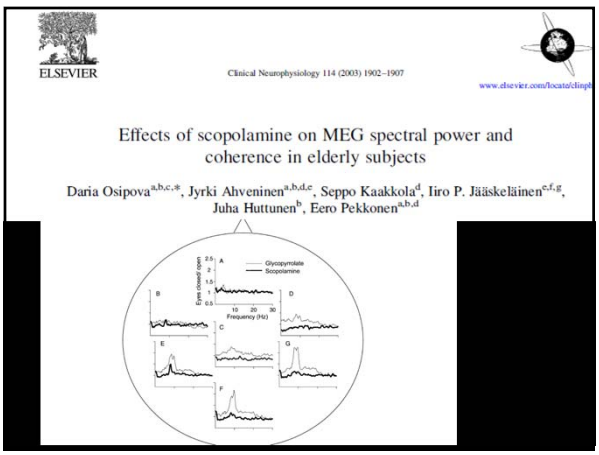


MCIa_Cluster



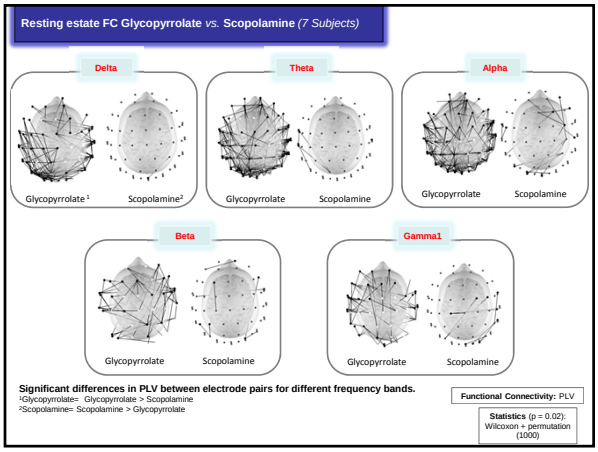

delta Ć
theta Ć
alpha Ć

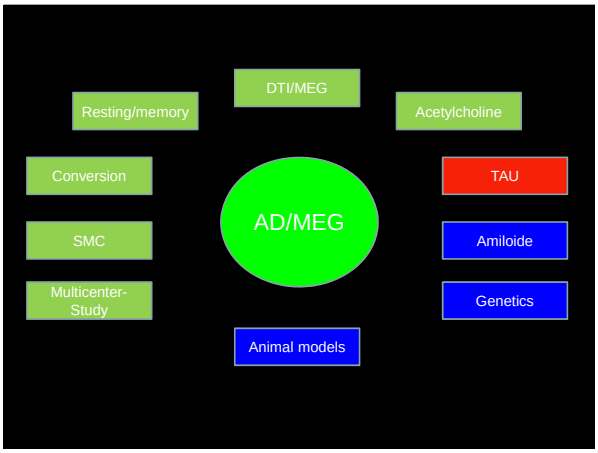

DTI/MEG
Resting/memory
Acetylcholine
Conversion
AD/MEG
TAU
SMC
Amiloide
Multicenter-Study
Genetics
Animal models


ELSEVIER
Clinical Neurophysiology 114 (2003) 1902–1907
www.elsevier.com/locate/clinph
Effects of scopolamine on MEG spectral power and coherence in elderly subjects
Daria Osipova[a,b,c,*], Jyrki Ahveninen[a,b,d,e], Seppo Kaakkola[d], Iiro P. Jääskeläinen[e,f,g], Juha Huttunen[h], Eero Pekkonen[a,b,d]

PHARMACOLOGICAL MODEL OF DEMENTIA: SCOPOLAMINE

1. Scopolamine is an acetylcholine muscarinic receptor antagonist.
2. Produces transient cognitive deficits: amnesia (episodic memory), resembling those observed in AD
3. The drugs were administered 1 h before the measurements and the subjects were supervised for at least 8 h after the drug administration (glycopyrrolate was administered as well in a separate session)
4. All recordings were conducted between 8 a.m. and 12 a.m. with 1 week interval between the sessions to minimize the possible effects of circadian rhythms.





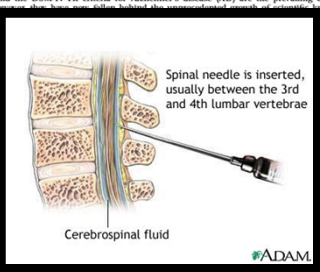
Position Paper

Research criteria for the diagnosis of Alzheimer's disease: revising the NINCDS–ADRDA criteria

Bruno Dubois*, Howard H Feldman*, Claudia Jacova, Steven T DeKosky, Pascale Barberger-Gateau, Jeffrey Cummings, André Delacourte, Douglas Galasko, Serge Gauthier, Gregory Jicha, Kenichi Meguro, John O'Brien, Florence Pasquier, Philippe Robert, Martin Rossor, Steven Salloway, Yaakov Stern, Pieter J Visser, Philip Scheltens

Lancet Neurol 2007; 6:734–46 The NINCDS–ADRDA and the DSM-IV-TR criteria for Alzheimer's disease (AD) are the prevailing diagnostic

TAU concentration has been associated with neuronal damage and cognitive impairment

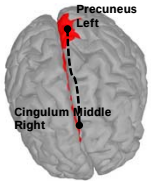


p-TAU in the CSF

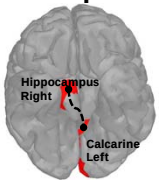
And

MEG-FC

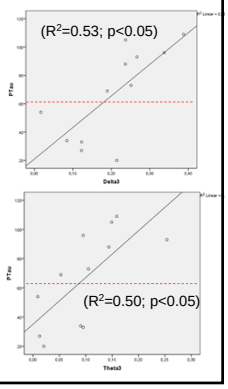
Delta pTau

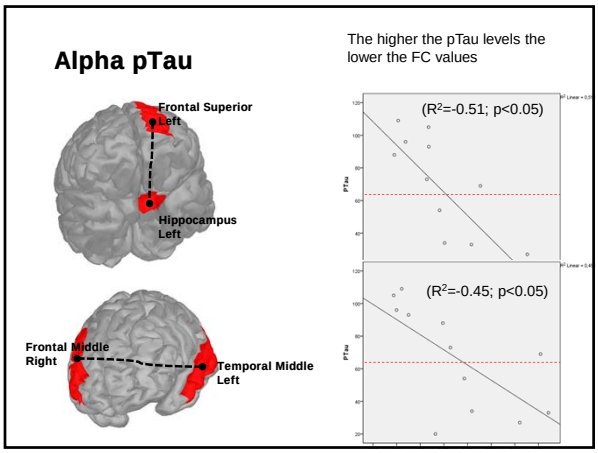


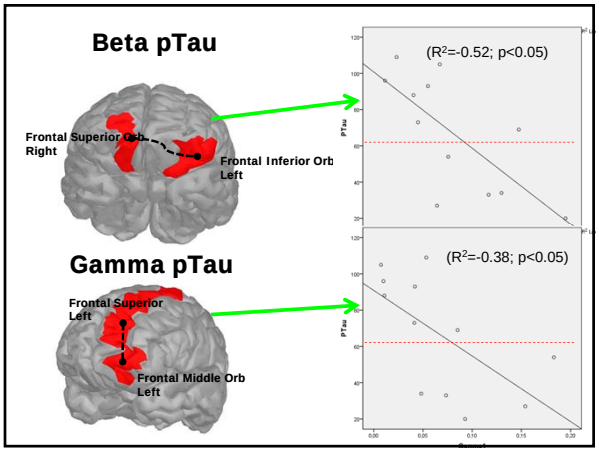
Theta pTau

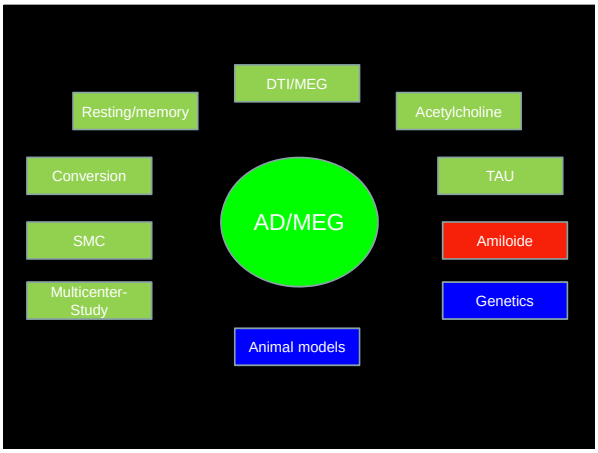


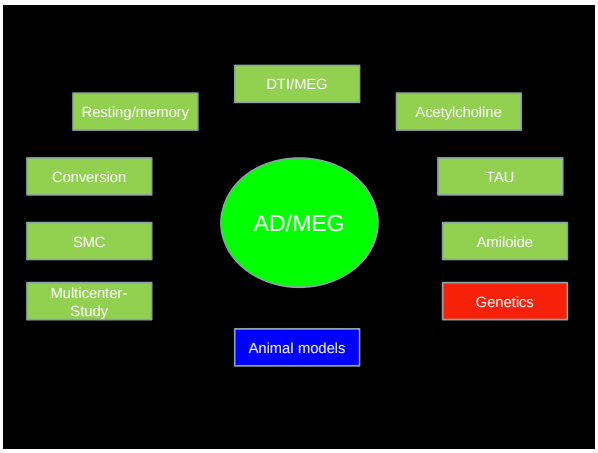
The higher the pTau levels the higher the FC values




Alpha pTau
The higher the pTau levels the lower the FC values
Frontal Superior Left
Hippocampus Left
Frontal Middle Right
Temporal Middle Left
(R²=-0.51; p<0.05)
(R²=-0.45; p<0.05)
PTau


Beta pTau
Frontal Superior Orb Right
Frontal Inferior Orb Left
Gamma pTau
Frontal Superior Left
Frontal Middle Orb Left
(R²=-0.52; p<0.05)
(R²=-0.38; p<0.05)
PTau


DTI/MEG
Resting/memory
Acetylcholine
Conversion
TAU
AD/MEG
SMC
Amiloide
Multicenter-Study
Genetics
Animal models

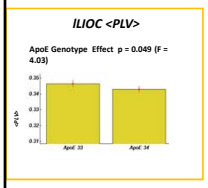


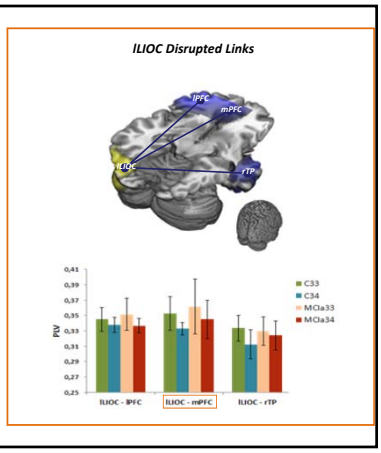
FUNCTIONAL CONNECTIVITY AND GENETIC PROFILES

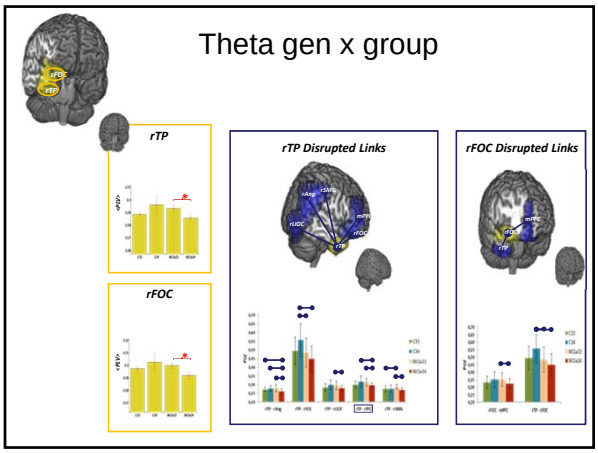
CARRIERS VERSUS NON CARRIERS OF APOE 3/4

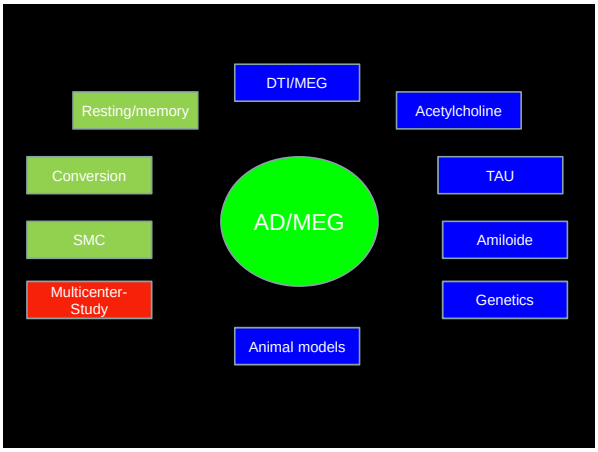
(Cuesta et al, submitted)

Delta Band APOE effect






Theta gen x group
rTP
rFOC
rTP Disrupted Links
rFOC Disrupted Links


DTI/MEG
Resting/memory
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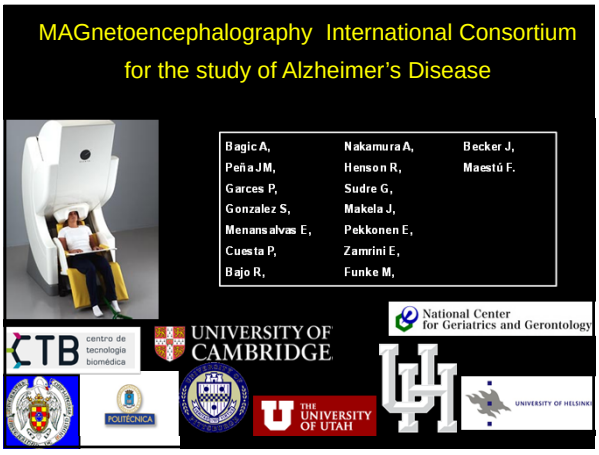
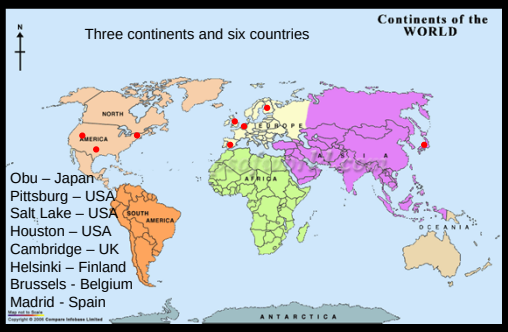
Genetics
Animal models


MAGnetoencephalography International Consortium for the study of Alzheimer's Disease
Bagic A, Peña JM, Garces P, Gonzalez S, Menanslvas E, Cuesta P, Bajo R,
Nakamura A, Henson R, Sudre G, Makela J, Pekkonen E, Zamrini E, Funke M,
Becker J, Maestú F.
CTB centro de tecnologia biomédica
UNIVERSITY OF CAMBRIDGE
National Center for Geriatrics and Gerontology
POLITÉCNICA
THE UNIVERSITY OF UTAH
UNIVERSITY OF HELSINKI

DISCLOSURE

- Elekta-Neuromag supported the annual meeting of the consortium

Magnetoencephalography International Consortium on AD
MAGIC-AD



SAGE-Hindawi Access to Research
International Journal of Alzheimer's Disease
Volume 2011, Article ID 280289, 10 pages
doi:10.4061/2011/280289

Review Article

Magnetoencephalography as a Putative Biomarker for Alzheimer's Disease

Edward Zamrini,[1] Fernando Maestu,[2] Eero Pekkonen,[3] Michael Funke,[1] Jyrki Makela,[4] Myles Riley,[1] Ricardo Bajo,[2] Gustavo Sudre,[5] Alberto Fernandez,[2] Nazareth Castellanos,[2] Francisco del Pozo,[2] C. J. Stam,[6] Bob W. van Dijk,[7] Anto Bagic,[8] and James T. Becker[8, 9, 10, 11]

SUMMARY

- It has been examined differences in functional connectivity between MCI and healthy controls with MEG **at the group level**.
- In order for MEG to be useful, it must be able to detect abnormal function at the **level of the individual patient.**

- There were **two goals** to the present study:
  - **To develop a model**, using data mining techniques, that reliably distinguishes between MCI patients and healthy controls.
  - **Test this model** using an unseen dataset of MCI and control subjects acquired by the MAGIC-AD consortium.

Stages of the study

1. Training datasets (known subjects)

- All data recorded (resting state) in Madrid
- MEG Datasets: 83 MCI and 54 controls

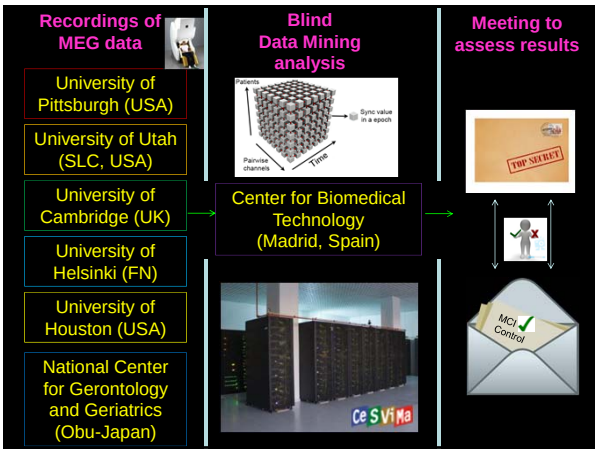
2. Validation datasets (Unseen/ blind study)

- Data recorded at five different MEG labs
- MEG data sets: 24 MCI and 28 controls

Results of the data mining classification
Madrid data

Internal validation

	Real class: MCI	Real class: Control		
Predicted class: MCI	65	15	81,25%	PPV
Predicted class: Control	13	39	75,00%	NPV
	83,33%	72,22%	78,79%	Accuracy
	Sensitivity	Specificity		

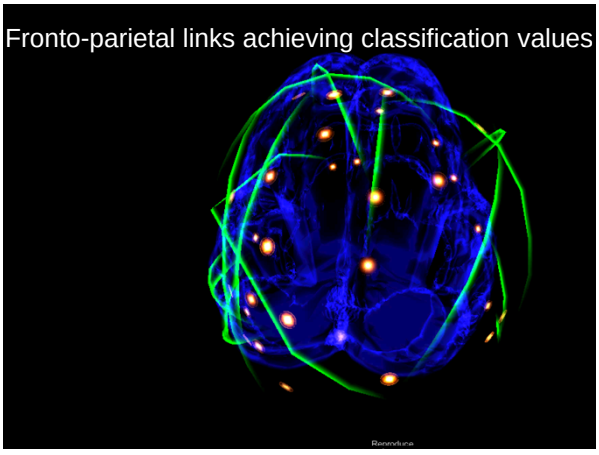


Results of the data mining blind classification

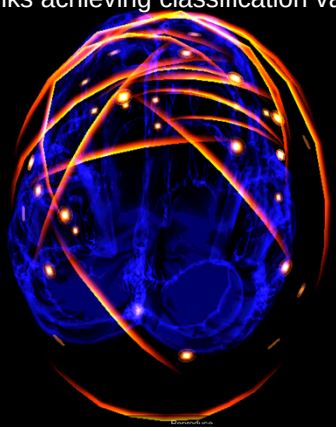
MAGIC-AD DATA

External validation

	Real class: MCI	Real class: Control		
Predicted class				
MCI	12	4	75,00%	PPV
Control	1	11	91,67%	NPV
	92,31%	73,33%	82,14%	Accuracy
	Sensitivity	Specificity		



Inter-hemisph links achieving classification values



Results of the data mining blind classification
MAGIC-AD DATA

External validation (Second Round)

	Real class: MCI	Real class: Control		
Predicted class: MCI	11	4	73,33%	PPV
Predicted class: Control	0	9	100,00%	NPV
	100,00%	69,23%	83,33%	Accuracy
	Sensitivity	Specificity		

CONCLUSIONS

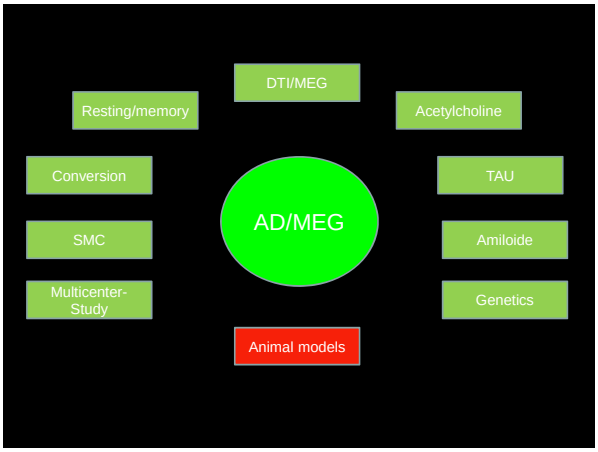
1.MEG is able to detect differences in functional connectivity profiles between MCI and control subjects across laboratories

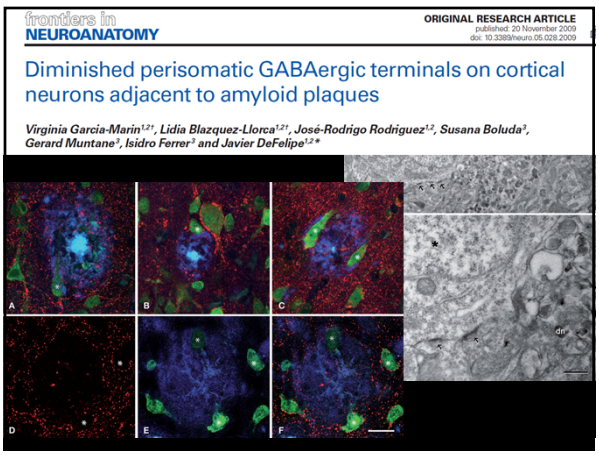
2.In the validation study a sensitivity of 96% and specificity of 72%, with a total accuracy of 83% (taking together the two MAGIC-AD samples) indicating the utility of MEG as a clinical tool

CONCLUSIONS

3. MCI showed hypersynchronization of the fronto-parietal networks and interhemispheric connections

4. Where we are: increase the sample, conversion study, combination with biomarkers

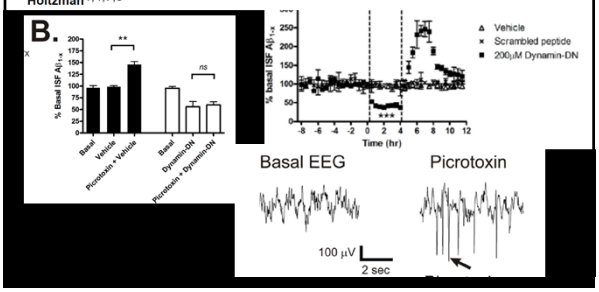




Published in final edited form as:
Neuron. 2008 April 10; 58(1): 42–51.

Endocytosis is required for synaptic activity-dependent release of amyloid-β in vivo

John R. Cirrito[1,2,7,8], Jae-Eun Kang[1,7], Jiyeon Lee[5,7], Floy R. Stewart[1], Deborah K. Verges[1], Luz M. Silverio[1], Guojun Bu[5,6,7], Steven Mennerick[2,3,7], and David M. Holtzman[1,4,7,8]



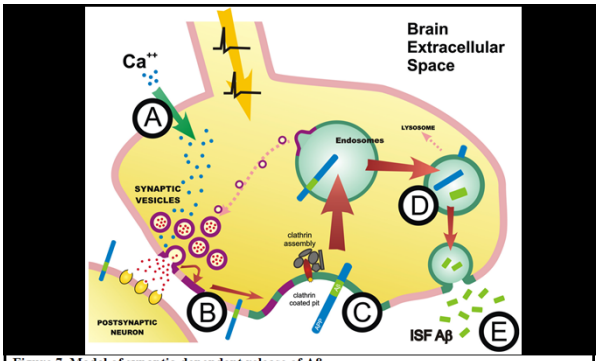
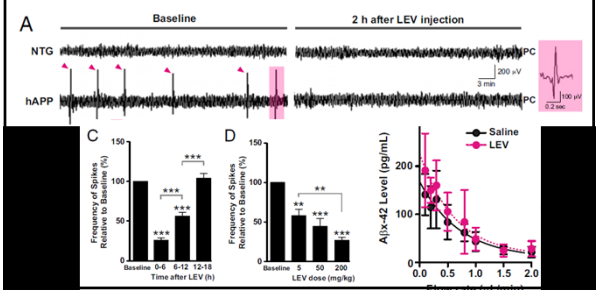


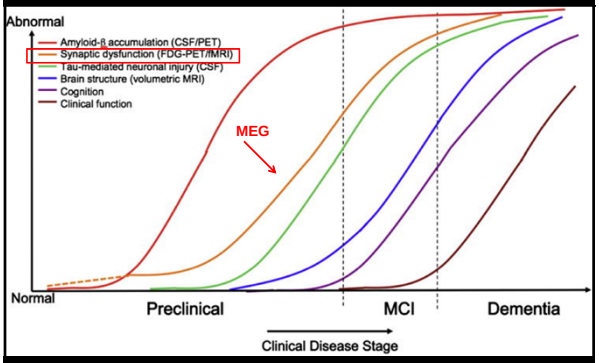
Figure 7. Model of synaptic-dependent release of Aβ
(A) Depolarization of the synaptic terminal causes calcium influx leading to synaptic vesicle release. (B) Synaptic vesicle membrane recycling from the cell surface via clathrin-mediated endocytosis causes more APP to internalized (C). Within endosomes, BACE and β-secretase cleave APP to produce Aβ (D) which is secreted from the neuron into the brain ISF (E).

Levetiracetam suppresses neuronal network dysfunction and reverses synaptic and cognitive deficits in an Alzheimer's disease model

Pascal E. Sanchez[a,b], Lei Zhu[a,b], Laure Verret[a,b], Keith A. Vossel[a,b], Anna G. Orr[a,b], John R. Cirrito[c], Nino Devidze[a], Kaitlyn Ho[a], Gui-Qiu Yu[a], Jorge J. Palop[a,b], and Lennart Mucke[a,b,1]

[a]Gladstone Institute of Neurological Disease, San Francisco, CA 94158; [b]Department of Neurology, University of California, San Francisco, CA 94158; and [c]Department of Neurology, Washington University School of Medicine, St. Louis, MO 63110





(Jack et al, 2010; Sperling et al, 2011)

CONCLUSIONS

1. MCI showed higher synchronization than controls
2. Multicenter study was able to classify MCI and controls
3. Reduction of the acetylcholine activity lead to a profile of AD.
4. APO-4 allele decrease synchronization
5. Increases of the Tau protein and beta amiloide disrupts the organization of the functional networks
6. The white matter damage affects the functional networks

ACMEGS
AMERICAN CLINICAL MEG SOCIETY

Update on Educational Initiatives

Update on MEG Fellowship Curriculum

Rick Burgess, Cleveland, OH

Update on MEG Fellowship Curriculum:
Beyond ACMEGS and other short courses ?

ACMEGS Annual Meeting
Atlanta Georgia
February 6, 2014

Consequences of the publication of the Clinical Practice Guidelines.

- The publication of CPGs* has helped to establish referring physicians' expectations for a high level of quality in the interpretation and for practical utility from the results.
- Centers with MEGs are striving to practice according to these guidelines.

*Bagic, Knowlton, Rose, Ebersole. CPG #1. J Clin Neurophysiol, 2011.
Burgess, Funke, Bowyer, Lewine, Kirsch, Bagic. CPG #2. J Clin Neurophysiol, 2011.
Bagic, Knowlton, Rose, Ebersole. CPG #3. J Clin Neurophysiol, 2011.
Bagic, Barkley, Rose, Ebersole. CPG #4. J Clin Neurophysiol, 2011.

Number of MEG installations is expanding.

- The number of MEG centers has increased over the two years since publication of the guidelines.
- MEG laboratories can now be found even in community hospitals.
- There has been considerable migration of MEG laboratory directors (the musical chairs involved Albuquerque, Birmingham, Chicago, Houston, McGovern, Memphis, Milwaukee, Montreal, NIMH, Omaha, Orlando, St. Louis, USC, New Jersey, and Utah).
- Some clinical positions for MEG laboratories remain unfilled.

Trained individuals to run these new MEG laboratories are in short supply.

- Decision makers (department chairs, administrators, C-suite officials) are concerned about this shortage.
- Especially those contemplating the new purchase of a MEG system want to know where will the future magnetoencephalographers come from.
- This apprehension is one of the major obstacles to the growth of the field.

What is ACMEGS's interest in these educational and staffing concerns?

- ACMEGS is actively involved in the education of people interested in MEG.
- What should ACMEGS do to encourage further education that will ensure that there is an adequate pool of qualified magnetoencephalographers?

Educational Training Opportunities: Definitions

- Fellowships
  - –Postresidency or Second Fellowship, generally for young trainees
  - –Requires some organizing, involvement of GME, and funding
  - –↳ ACMEGS likely has an important role
- Ad hoc education for potential MEG center directors
  - –Short progams/observerships for seasoned EEGers/epileptologists
  - –Usually arranged unofficially, person to person, customized to the time available (both teacher's and student's schedule)
  - –↳ ACMEGS unlikely to have any effect

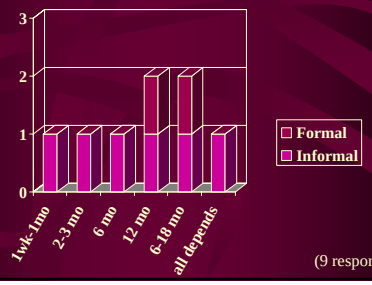
Centers with MEG Training Programs.

Results of anonymous surveys sent to all centers who are institutional members of the ACMEGS (16)

	Formal progams	Informal programs
No. of centers	2	7
No. of trainees/yr	0 - 2	0 - 3
No. Spont MEGs	50 - 100	"all depends"
No. SEFs	20 - 50	0 - 100

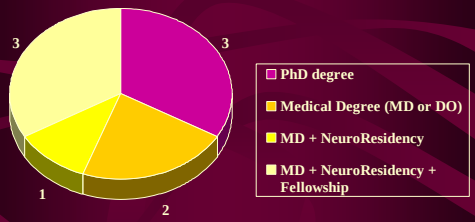
Duration of Training

6 / 9 (67%) adjust the duration of training to suit the experience of the trainee

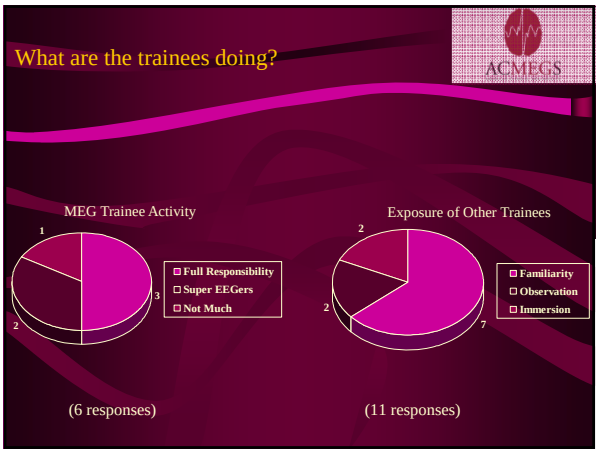


(9 responses)

Minimum qualifications required for acceptance into training program.



What are the trainees doing?

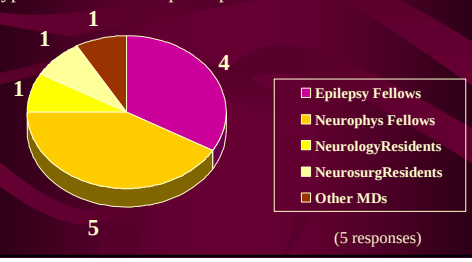


(6 responses) (11 responses)

Who is doing the clinical work?

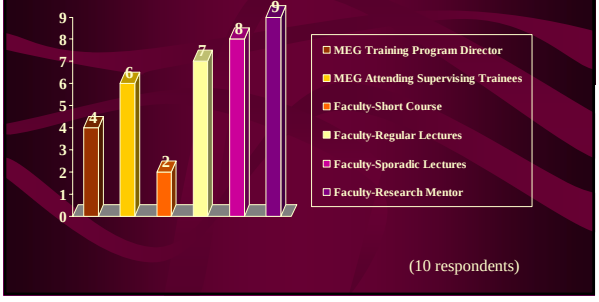
No help from trainees 7/11 (64%)

Types of trainees who participate:



(5 responses)

What educational activities is the MEG laboratory director doing?



(10 respondents)

Survey Free -Text Comments.

- "Clinical training programs will proliferate when MEG centers become financially self-sustaining"
- "Would like to be a training site"
- "Would love to start a training program here"
- "MEG community should put major effort and resources into formalizing physician training"
- "Fully support the formation of 1-year ACGME-approved fellowship"
- "Survey fails to address particular needs of fully qualified directors or fellows on elective rotations who only want/need to learn certain aspects or gain some appreciation"
- "MEG community should create a consensus as to training content, expectations, and program accreditation"

(7 responses)

What can ACMEGS do to promote the availability of qualified magnetoencephalographers?

- Present courses (such as our 'Annual Course')?
- Provide multi-day on-site training programs?
- Assist the manufacturers with start-up training?
- Develop guidelines for clinical fellowships?
- Continue to agitate / encourage appropriate individuals?

Does the additional training required demand a subspecialty fellowship?

"Additional background training of physicians interpreting clinical MEG and MEG–EEG studies should meet the minimal requirements for examination by the American Board of Clinical Neurophysiology (www.abcn.org) or the American Board of Psychiatry and Neurology Added Qualifications in Clinical Neurophysiology (www.abpn.com)."

A Bagic. ACMEGS Annual Course, February 2013, Miami Fla

Where do we go next?

- Just let things evolve for awhile and pick up later?
- Expand (double) the survey to non-ACMEGS centers?
- Obtain consensus within ACMEGS?
- Develop and publish fellowship guidelines?
- Put Training Guidelines on the web?
- Include MEG track in ABCN exam?
- Officially pursue development of non-ACGME fellowships?

Should ACMEGS generate Fellowship Training Goals?

- Young physicans find this technology clinically powerful and intellectually fascinating, and they are looking for good training programs.
- The most basic definitions of who, what, where, and how long, about MEG training do not exist.
- If non-ACGME fellowships are further developed will this have a positive influence:
  - On hiring?
  - On the entire field of clinical magnetoencephalography?

Key questions for Fellowship Training Goals.

- **Eligibility ---**
  - Who are these clinical fellowships for?
- **Duration ---**
  - What's the minimum duration? What about requests for mini-fellowships?
- **Content ---**
  - Practice? Number of studies?
  - Apprenticeship? Didactic?
- **Other Components ---**
  - Just magnetoencephalography or coupled with epilepsy, etc?
- **Certification ---**
  - Formal certification in future?
  - At present, what does it take to get a letter of completion?

Formal Fellowship Options: UCNS.

- ACGME-approved MEG Fellowship extremely unlikely.
- UCNS: Unified Council on Neurological Subspecialties.
- It provides unified accreditation for non-ACGME fellowship programs.
- They have established patterns for accreditation and certification, and can provide guidance.
- Subspecialties may progress to ACGME, or remain under UCNS, e.g. Headache Medicine.

Crumrine P. UPMC, Personal Communication, March 2013, Cleveland, OH

Speculating Based on Analogous UCNS Examples.

- Because of the possible/historical pathways followed by physicians into MEG, our sub-specialty may emulate the trajectories taken by Sleep and Interventional Neurology.
  - Sleep fellows come from Neurology, Psychiatry, or Pulmonary.
  - Interventional fellows come from Neurology, Neurosurgery, or Radiology.
- Usually one or more of the primary boards will have to accommodate those fellows.
- Current ACMEGS leanings (at least as documented by CPGs) are that MEG will eventually be populated by neurologists with fellowship training in CNP or Epilepsy Medicine.

Crumrine P. UPMC, Personal Communication, March 2013, Cleveland, OH

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ACMEGS
AMERICAN CLINICAL MEG SOCIETY

Update on Educational Initiatives

Update on MEG/EEG Technologist Activities

Janice Walbert, ABRET & Judy Ahn-Ewing, ASET

ACMEGS
AMERICAN CLINICAL MEG SOCIETY

Update on Educational Initiatives

Update on Clinical Startup Recommendations

Paul Ferrari, Austin TX & Ron Gordon, Vancouver BC

AMERICAN CLINICAL MAGNETOENCEPHALOGRAPHY SOCIETY
2013 Annual Conference ♦ February 7, 2013

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:

	Most Effective				**Least Effective**	
Faculty						**Comments**
Dr. Bagic	5	4	3	2	1	
Dr. Paetau	5	4	3	2	1	
Dr. Ebersole	5	4	3	2	1	
Dr. Baillet	5	4	3	2	1	
Dr. Von Allmen	5	4	3	2	1	
Dr. Knowlton	5	4	3	2	1	
Dr. Burgess	5	4	3	2	1	
Ms. Ahn-Ewing	5	4	3	2	1	
Ms. Walbert						
Dr. Alexopoulos	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

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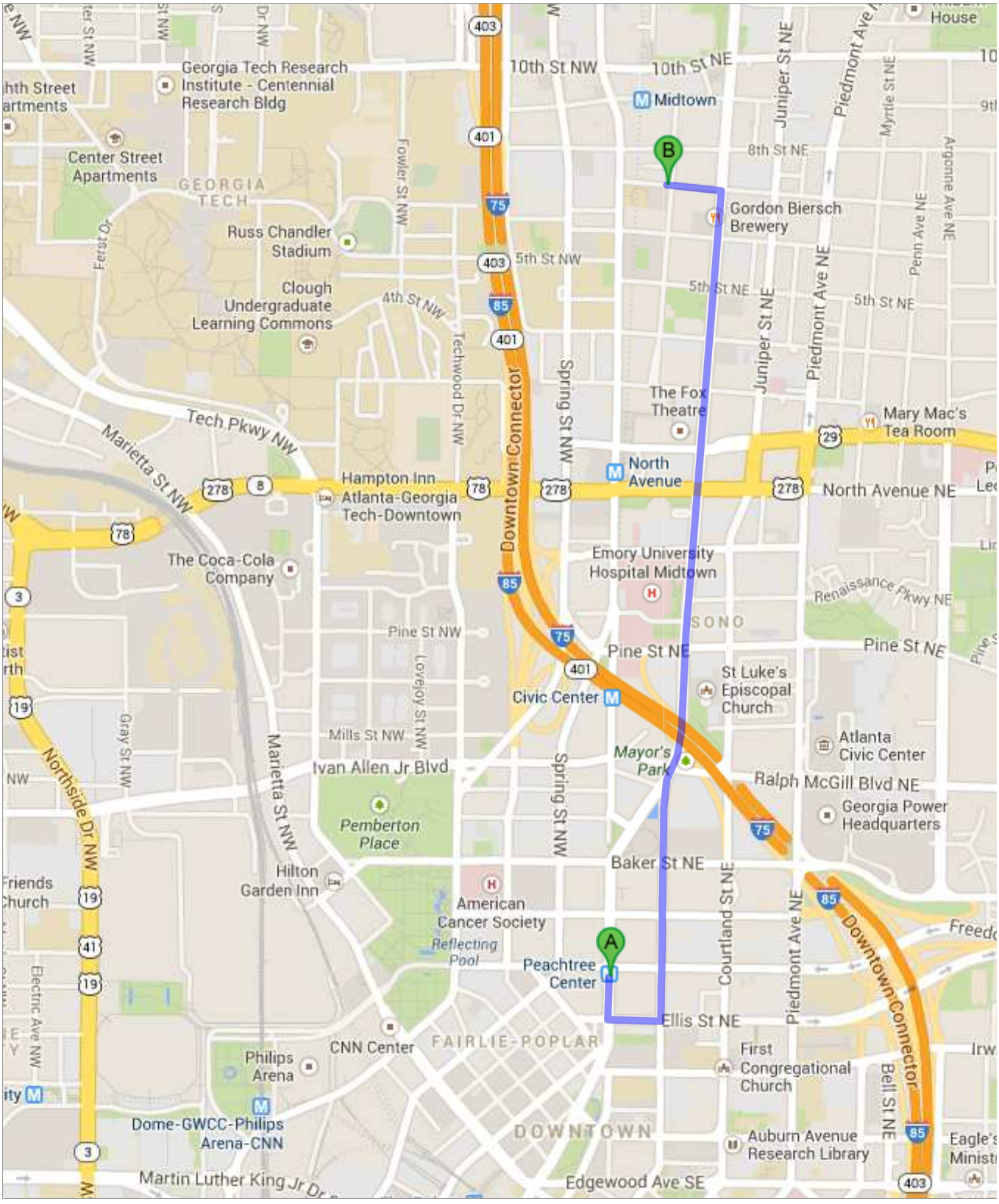

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