Graduating Residents

Ping Li, MD  
Adult Neurology

Atul Mangla, MD  
Adult Neurology

Hemavathy Purushotham, MD  
Child Neurology

Tareq Kass-Hout, MD  
Adult Neurology

Omar Kass-Hout, MD  
Adult Neurology

Anil Neelakantan, MBBS  
Adult Neurology

PGY III-IV Residents 2012-2013

Salomi Salins, MBBS  
Adult Neurology

Novreen Shahdad, MBBS  
Adult Neurology

Alyssa Fiddler, DO  
Adult Neurology

Ahmad Abokhamis, MD  
Adult Neurology

Mahmoud Al Masy, MD  
Adult Neurology

Supriya Kohli, MBBS  
Adult Neurology

Nitin Agarwal, MBBS  
Child Neurology

Catherine Porter, MD  
Adult Neurology

Lynn Chouhfeh, MD  
Adult Neurology

Karanbir Singh, MBBS  
Adult Neurology

Rajesh K. Gupta, MBBS  
Adult Neurology

Michael E. Cohen
Residents
Research Day

2012

Friday, June 15, 2012
11:00 am—3:30 pm
Cummings Conference Center
Welcome:
Gil I. Wolfe, MD, FAAN

Introduction:
Robert Zivadinov, MD, PhD, FAAN

Welcome/Introduction
11:00 am  Gil I. Wolfe, MD, FAAN
           Robert Zivadinov, MD, PhD, FAAN

Presentation Session # 1
11:10 am  Anil Neelakantan, MBBS
11:30 am  Omar Kass-Hout, MD
11:50 am  Ping Li, MD
12:10 pm  Atul Mangla, MD
12:30 pm  Break/Photo Session

Presentation Session # 2
1:00 pm   Tareq Kass-Hout, MD
1:20 pm   Hemavathy Purushotham, MD
1:40 pm   Catherine Porter, MD
1:55 pm   Salomi Salins, MBBS
2:10 pm   Break

Presentation Session # 3
2:20 pm   Alyssa Fiddler, DO
2:35 pm   Nitin Agarwal, MBBS
2:50 pm   Novreen Shahdad, MBBS
3:05 pm   Lynn Chouhfeh, MD
3:20 pm   Emad Nourollah-zadeh, MD
Welcome to the Michael E. Cohen Resident Research Day, the annual event held by the University at Buffalo Department of Neurology staged in recognition of research projects conducted by our residents and fellows.

Our research day represents the culmination of months and even years of meticulous work by our neurology trainees. This work is now subjected to peer scrutiny and competition for awards.

Moreover, the research day recognizes the involvement of our faculty and fellows in the mentorship of residents. Experience and lessons learned are passed from each generation of physician researchers to the next in just this way.

Through the years, graduates of our program have repeatedly confirmed the invaluable experience of their participation in the Research Day. Their comments express an increased appreciation not only for the clinical research process itself but also for the positive impact it will always have on their clinical careers.

Today's presentations continue an established tradition of academic excellence. Please join the entire Department of Neurology in commending each resident and fellow for the innovation, scope and execution of their projects. On display are analytical skills, judgment and integrity. Please also accept my sincere appreciation to all of you for contributing to and sharing the day's events.

This is the ninth year of our revamped Resident Research Training Program, and the scope and variety of the projects presented today clearly demonstrate creativity, ambition, dedication and flair.

Whether our presenters' careers lead towards clinical work or further research, they are all true scholars, having exhibited the discernment, intuition and drive that will guide them deftly in future years. I congratulate each and every one of them for a job superbly done.

It has been my main objective in these last few years to foster and facilitate such an expansion in project diversity. As you see in our program today, although we continue to foster study in the areas of our strength and mainstays—multiple sclerosis and stroke—we have also increased the number of projects that explore other neurological disorders and diseases.

With these further innovations, we hope to open the door to the next level of research excellence. Projects that are increasingly comprehensive and creative will substantively boost the careers of our young doctors as well as enhance both the prominence and reputation of our Neurology Residency Program. What a wonderful undertaking to be part of!
Background Vitamin D3 (VitD3) deficiency has been implicated in a variety of neurological diseases. It has also been linked to the development of insulin resistance by causing low grade inflammation. VitD3 supplements reduce neuropathic pain in diabetic patients. VitD3 deficiency has been associated with development of diabetic neuropathy.

Objective To investigate the relationship of Compound Motor Unit Action Potential (CMAP) amplitude, conduction velocity, F-latency, presence of conduction block in peroneal/tibial nerves and sural nerve SNAP latency/amplitude to serum vitamin D3 levels in dysglycemic patients.

Methods The study was performed as a retrospective chart review at the Veterans Association Hospital in Buffalo, NY. Patients have diabetic neuropathy, have serum VitD3 levels and who have had EMG were identified and their results reviewed. The patients were divided into two categories (VitD3 deficient and normal) and the CMAP amplitudes, conduction velocities, F-latencies, sural SNAP latencies/amplitudes were compared using independent T-tests. The percentage of patients with conduction block were calculated in each group and analyzed with chi-square. Linear correlation model/graphs were plotted to analyze the relationship between serum levels of deseasonalized VitD3 with the parameters.

Results The study showed that tibial conduction velocity and median CMAP amplitude were significantly worse in the Vitamin D3 deficient group. Conduction block was present 3.6 to 6.6 times higher in patients with vitamin D3 deficiency. VitD3 deficient diabetic neuropathy patients were 3.1 times more likely to have an absent sural nerve response compared to diabetics with normal VitD3 level. Vitamin D3 levels showed inverse correlation with HbA1c levels, tibial and ulnar CMAP amplitudes.

Conclusions Compared to diabetics with normal Vitamin D3 levels, the patients with VitD3 deficiency were more likely to have conduction block and absent sural sensory nerve responses. Tibial and median nerve conduction parameters were worse in patients with Vitamin D3 deficiency.
Bridging with Intravenous Thrombolysis in Endovascular Therapy for Acute Ischemic Stroke

Omar Kass-Hout, MD MPH1, Kenneth V. Snyder, MD PhD,2 Vladan Radovic, MD MS1

1Department of Neurology, Jacobs Neurological Institute, SUNY at Buffalo, Buffalo, NY, USA; 2Department of Neurosurgery, SUNY at Buffalo, Buffalo, NY, USA

Background Large vessel occlusions with heavy clot burden are less likely to improve with intravenous (IV) thrombolysis alone. The purpose of this study was to show whether a combination of IV thrombolysis and endovascular therapy was superior to endovascular treatment alone.

Methods Data for 104 patients with acute large artery occlusion treated between 2005 and 2010 were reviewed. Forty-two received endovascular therapy in combination with IV thrombolysis (bridging group), and 62 received endovascular therapy only. Clinical outcome, mortality rate, and symptomatic intracranial hemorrhage (sICH) rate were compared between the two groups.

Results The two groups had similar demographic and vascular risk factor distribution, as well as National Institutes of Health Stroke Scale score on admission (mean ±SD: 14.8 ±4.7 and 16.0±5.3; P=0.23). No difference was found in Thrombolysis in Myocardial Infarction recanalization rates (score of 2 or 3) following combined or endovascular therapy alone (83.33% and 79.03%; p=0.585). Favorable outcome, defined as a modified Rankin Scale score of ≤2 at 90 days, also did not differ between the bridging group and the endovascular-only group (37.5% and 34.48%; P=0.643). There was no difference in mortality rate (22.5% and 31.03%; P=0.5618) and sICH rate (10.2% and 12.5% P=0.734). A significant difference was found in mean time from symptom onset to treatment in the bridging group and the endovascular-only group (227±88 min vs. 125±40 min; P<0.0001).

Conclusions Combining IV thrombolysis with endovascular therapy resulted in similar outcome, revascularization, sICH, and mortality rates compared with endovascular therapy alone. Prospective clinical studies comparing both treatment strategies in acute ischemic stroke are warranted.
Do omega-3 fatty acid ethyl esters (OFA) improve Clopidogrel-associated P2Y12 inhibition in stroke patients?

Ping Li¹, Haris Kamal¹, Melissa Baxter², and Bijal Mehta³

¹Department of Neurology, Jacob Neurological Institute, SUNY at Buffalo, Buffalo, NY; ²Department of Pharmacy, Buffalo General Medical Center, SUNY at Buffalo, Buffalo, NY; ³Department of Neurology, University of California Los Angeles, Harbor-UCLA Medical Center, Torrance, CA

Background  The action of OFA in preventing cerebrovascular disease (CVD) remains unknown. In addition to altering lipid profiles, OFA may have platelet aggregation inhibitory effects, as previously reported. Clopidogrel inhibits platelets via the P2Y12 receptor. OFA may alter clopidogrel-associated platelet inhibition via a possible synergistic effect on P2Y12 inhibition.

Objective  To determine if OFA affects clopidogrel-associated P2Y12 receptor inhibition by comparing the percentage of responders: 1) in CVD patients who were taking both clopidogrel and OFA with patients only taking clopidogrel; 2) in CVD patients given loading doses of clopidogrel along with OFA and patients receiving only loading doses of clopidogrel.

Methods  This is a retrospective chart review study of adult patients with CVD or cerebral aneurysms and taking clopidogrel, who were seen at a single hospital between March 2010 to September 2011. Patients who were taking clopidogrel only for cardiac reason, whose P2Y12 inhibition tests were done prior to the administration of clopidogrel, those who did not have the test, and those who did not have medication list on record were excluded. Statistical analysis was performed using Fisher’s exact test for the categorical variables and student’s t-test for the continuous variables.

Results  478 subjects were included in the study. For the 70 subjects who received loading doses of both clopidogrel and OFA, 67.1% had a positive P2Y12 inhibition response. For the 65 subjects who received just clopidogrel loading doses, 64.6% of subjects were responders. There were 69.4% responders in the 294 subjects who were taking clopidogrel alone at home, and 75.5% responders in the 49 subjects who were taking both clopidogrel and OFA at home. However, these percentage differences were not statistically significant.

Conclusion  This study did not find additional P2Y12 platelet inhibition when patients were given OFA, although they had a trend toward having adequate platelet inhibition. However, OFA might inhibit platelet aggregation through other mechanisms.

Dr. Li is originally from China. After completing medical school at Beijing Medical University, she went to Canada and pursued her Masters Degree in Neuroscience at the University of British Columbia. She subsequently did research in stroke at the University of British Columbia and had published several papers in peer-reviewed journals. She joined the Neurology Residency at University at Buffalo in 2008 and is currently a PGY4 neurology resident. Her interest is to pursue a clinical neurophysiology fellowship.
Statin Induced Myopathy and Vitamin D Insufficiency

Atul Mangla, MD, Barbara Teter, PhD, MPH, Nicholas Silvestri, MD

Department of Neurology, Jacobs Neurological Institute, SUNY at Buffalo, Buffalo, NY, USA

Originally from New Delhi, India, Dr. Mangla completed his medical training at Calicut Medical College in India. Afterwards, he worked as a junior resident in the Neurology department in New Delhi. He finished his Internal medicine year at Westlake Hospital in Chicago and entered the Neurology residency program at UB in 2009. Currently, he is a PGY4 resident and is graduating this year. His interest is to pursue a fellowship in Neuromuscular medicine.

**Background and Purpose** The vitamin D receptor is present in skeletal muscle and vitamin D deficiency has been known to cause myalgia. Myopathy has been known to be associated with statins and there is limited data to show interaction between vitamin D insufficiency and statin induced myopathy.

**Objectives** We aim to find a significant association between vitamin D insufficiency and statin induced myopathy.

**Methods** We retrospectively collected data on 1089 consecutive patients who were on a statin with available baseline vitamin D levels, seen at our institution between Jan 2001 and Dec 2011.

Patients were assigned into two groups based on presence of myopathy, which was defined as presence of myalgia or serum CK levels > 3 x ULN. Patients with no baseline vitamin D levels and with myalgia or high serum CK secondary to other reasons were excluded. Using multivariate logistic regression we evaluated potential predictors of statin-induced myopathy.

**Results** 44 patients in the myopathy group and 845 patients in the control group were comparable for age (mean= 70.1/71.08 year, p<0.29), statin dose (mean= 37.58/38.36 mg, p<0.38) with significant difference in vitamin D level (mean= 20.9/26.13 ng/dl, p<0.05) and serum CK levels (mean = 484.09/126.24 U/L, p<0.05). Among 244 patients in the myopathy group, 207(84.8%) had low vitamin D (OR 3.176; 95% CI 2.17- 4.63). Patients who were younger than 60 years, male or black in race showed a trend towards higher incidence of myopathy but were not significant (p=>0.05). By multivariate analysis low vitamin D level was the only independent predictor of statin-induced myopathy.

**Conclusion** The study demonstrates the odds of having statin-induced myopathy in patients with low vitamin D levels. Symptomatic myalgia in statin-treated patients with concurrent vitamin D deficiency may reflect a reversible interaction between vitamin D deficiency and statins on skeletal muscle.
Wake Up Symptomatic Stroke in Acute Brain Ischemia (WASSABI) Trial

Tareq Kass-Hout, MD, Omar Kass-Hout, MD MPH, Maxim Mokin, MD PhD, Emad Nourallah-zadeh, MD, Mahmoud Al Masry, MD, Vladan Radovic, MD, Robert Sawyer, MD

Department of Neurology, Jacobs Neurological Institute, SUNY at Buffalo, Buffalo, NY, USA

Background Acute stroke is the fourth leading cause of mortality and the major cause of long-term disability in the developed world. Approximately 25% of ischemic stroke patients awaken with their symptoms. This is called a Wake Up Stroke (WUS). Perfusion studies help identify the patients who still have penumbra and are eligible to receive revascularization therapy.

Methods Data were collected from 21 US centers of 97 WUS patients presented with considerable salvageable tissue on perfusion studies and a NIHSS 8-22 were enrolled in the study. Patients with hemorrhagic stroke were excluded. Patients were randomized into one of two prospective groups: best medical therapy (standard of care) and intravenous thrombolysis. The primary end point, mRs-90 days of 2 or less, was compared between the two groups. Mantel-Hanzel test was used to compare the proportion of patients with a good outcome. Safety measurement, represented by the symptomatic intracranial hemorrhage, was compared in the same way following the ECASS III criteria.

Results A total of 97 patients were enrolled in the final analysis. Fifty-one patients were in the IV tPA arm and 46 in the control arm. The treatment group showed a higher chance of better outcome with OR 1.46, 95% CI(1.37-1.98). Symptomatic intracranial hemorrhage was higher in the IV tPA group (3.9% vs. 2.2% p-value < 0.0001). Mortality at three months in the control group was 19% vs. 10% in the IV tPA group (p-value=0.095).

Conclusion Utilization of perfusion studies might be a useful tool to determine eligibility for intravenous thrombolysis in patients with unknown time of onset.
Effect of cooling therapy on Phenobarbital levels in newborns

Hemavathy Purushotham, MD1 Kelly Michienzi,2 Pharm D, E. Ann Yeh, MD,3 Murali Ramanathan, PhD2, Jonathan Knights2, Satyanarayana Lakshminrusimha,4 MD

1Department of Neurology, Jacobs Neurological Institute, SUNY at Buffalo, Buffalo, NY, USA; 2Department of Pharmaceutical Sciences, SUNY at Buffalo, Buffalo, NY, USA; 3Department of Neurology, Hospital for Sick Kids, Toronto, Canada; 4Department of Pediatrics, SUNY at Buffalo, Buffalo, NY, USA

Background Seizures are common in newborns with Hypoxic Ischemic Encephalopathy (HIE). Phenobarbital is the most widely used antiepileptic drug (AED) in this subgroup of patients. Therapeutic hypothermia has become the standard of practice for newborns with HIE. The effect of therapeutic hypothermia on the metabolism and pharmacokinetics of Phenobarbital is not clear.

Objectives To evaluate the effect of therapeutic hypothermia on Phenobarbital. To determine if the clearance of Phenobarbital from the body is decreased among newborns that have undergone cooling compared to the newborns that have not undergone cooling.

Methods All newborns admitted consecutively to Women and Children’s Hospital of Buffalo’s neonatal intensive care unit (NICU) and treated with Phenobarbital between January 2009 and January 2012 will be identified. These subjects will be categorized in 3 groups. The first group will include asphyxiated neonates who had undergone cooling. The second group will comprise asphyxiated/HIE newborns. The third group consists of newborns without perinatal asphyxia who received Phenobarbital for seizures. The time to reach steady state and the time to maintain a steady state level of Phenobarbital will be compared among the three arms.

Expected results Asphyxia and cooling will cause delayed clearance of Phenobarbital from the body. This may be important from a pharmacological perspective particularly in reducing the potential toxicity of the drug. We may also gain perspective on adjusting the dosage, frequency of administration and monitoring the drug levels.

Dr. Purushotham attended medical school in India and completed her Pediatric residency in Brooklyn. She is board-certified in pediatrics and is currently in a three-year child neurology program. On completion she will pursue her interest in working with children. She is married to a wonderful husband and is a mother of two adorable girls.
Olfactory Testing for Biomarkers in Alzheimer’s Dementia

Catherine Porter, MD, Kinga Szigeti, MD, PhD, Deepika Lal
Department of Neurology, Jacobs Neurological Institute, SUNY at Buffalo, Buffalo, NY, USA

Background Alzheimer’s disease (AD) is the most prevalent of the dementias, currently affecting 5.4 million in the US and some 36 million worldwide (ADI, 2011). By 2050, the direct US cost of AD and other dementias is projected to increase to over 1.1 trillion dollars and worldwide prevalence to exceed 115 million (AA, 2012).

Early identification of at-risk individuals and targeted intervention strategies could result in a significant reduction in public health burden and improved quality of life for Alzheimer’s patients and caregivers.

Objectives To expand upon recent investigations identifying an olfactory receptor copy number variant (OR CNV) on Chromosome 14 associated with age at onset (AAO) of AD (Shaw, Szigeti, et al, 2011) by (1) Screening olfactory receptor regions with a custom microarray for AD associations; (2) Assessing if identified OR CNVs correspond to a change in olfaction, and; (3) Evaluating relationships between OR CNVs and specific neuropsychological profiles.

Methods The proposed project is a pilot study of 150 subjects (50 AD, 50 amnestic MCI, and 50 normal controls) associated with a larger study (“Olfactory Receptor Copy Number Variation Association with Age at Onset of Alzheimer Disease.”). Pilot study components include (1) clinical neurological evaluation; (2) neuropsychological testing; (3) smell testing with the University of Pennsylvania Smell Identification Test (UPSIT); (4) obtaining DNA for olfactory receptor array; (5) customized olfactory receptor array testing, and; (6) data analysis.

Expected Results/Conclusions We expect to find several atypical CNV OR regions in the human genomes with AD associations. The identified CNVs will be further studied in larger replication cohorts and, if confirmed, could serve as biomarkers in increasing diagnosis accuracy, allowing earlier diagnosis, or aiding in risk stratification for conversion to AD, all of which can help in clinical trial designs for disease-modifying therapy.
Dr. Salomi Salins attended medical school at Rajiv Gandhi Medical College in India and graduated in 2008 after completing a year of internship. She then completed the first year of internal medicine at the University of Buffalo in 2009. She is currently in the third year of Neurology and will graduate in 2013; she will be Co-Chief Resident for the next academic year, 2012-2013. Dr. Salins has been awarded a Neurophysiology fellowship at the University of Buffalo for 2013-2014.

**Background** Once a fatal disease, MG has a much more favorable prognosis and outcome with the introduction of effective pharmacologic therapy. It reaches a maximum level of severity in the first 2 years, followed by improvement. Many eventually progress to a state where their symptoms stabilize and likely will stay in a state of chronic remission.

**Hypothesis** The disease course of MG appears to show a pattern of chronic remission. Over time, the functional status improves (MGFA) as the medication requirement decreases.

**Objectives**
1. Find out the disease course of MG over 10 years or longer since diagnosis.
2. Find out the medication requirement each year over the number of years since diagnosis.
3. Compare functional status (MGFA status) amongst those on low dose medications vs high dose medications.

**Statistical methods**
1. This is a retrospective chart review. A post-intervention MGFA status will be obtained for each year from diagnosis. The specific doses for Prednisone, Azathioprine, Mycofenolate, Cyclosporine and Pyridostigmine will be obtained for each year from diagnosis.
2. A time trend analysis between the MGFA status and medication doses will be done.
3. A comparative analysis will be done, comparing the MGFA post-intervention status in patients on high doses of above pharmacologic agents versus low doses or no agents. The sample size being small, we will do a Chi-Square Analysis.

**Expected Results**
1. We expect a sigmoid upsloping trend between the MGFA post-intervention status and the number of years from diagnosis.
2. In stable disease, patients on low/no dose medications do not have a functional status different from those who are on high dose medications.
3. Over time, the post intervention MGFA status improves as the medication requirement decreases.
Alyssa Fiddler is a PGY3 resident in the neurology program of SUNY at Buffalo. A native of Hamilton, Ontario, in Canada, Alyssa received her medical training at Lake Erie College of Osteopathic Medicine in Erie, PA. Her undergraduate training includes a Bachelor of Science degree from McMaster University in Hamilton, ON. After completing her residency, Alyssa will be joining the neurophysiology fellowship with JNI.

**Background** Triphasic waves have been shown to co-exist with other abnormal EEG patterns, i.e., alpha coma, but the correlation between typical triphasic waves and epileptiform discharges in the same EEG has yet to be investigated.

**Objective** In this retrospective study, we will try to evaluate how often triphasic waves and epileptiform activity occur together on electroencephalogram in an adult epilepsy population.

**Methods** This will be a retrospective chart review of the Women and Childrens Hospital of Buffalo EEG database. Charts from 2007-2010 will be reviewed. The diagnosis of triphasic waves and epileptiform activity will be done by the neurologist assigned to read electroencephalograms who will not be aware of the study being performed at the time of study interpretation.

Statistical analysis will be performed using STATA (Statacorp, TX). Planned tests include the Chi-square test and/or Fisher’s exact test.

**Expected results** There is likely an increased chance of epileptiform activity on EEG in patients with triphasic waves. This would be important for neurologists to be aware of, as often the encephalopathy of these patients is attributed to the underlying etiology of the triphasic waves. The epileptiform activity, with resultant seizures, may be contributing to the encephalopathy seen in many of these patients.


**Clobazam Effect on Seizure Control and Quality of life**

Nitin Agarwal, MBBS, Joy Parrish, MD, Sarah G. Finnegan, MD, PhD, Susan L. Kerr, MD, Thomas J. Langan, MD, Mary Jo Elgie, BSMT, Arie Weinstock, MD

Department of Neurology, Jacobs Neurological Institute, SUNY at Buffalo, Buffalo, NY, USA

---

**Introduction**  Clobazam (CLB) is a 1, 5-benzodiazepine that has been used as an adjunctive anticonvulsant drug in treatment of epilepsy. The FDA has recently approved it for use in drop seizures associated with Lennox Gastaut Syndrome. Patients with chronic medical conditions like epilepsy have significant worsening of quality of life, which can be studied by various standard measures. The Quality-of-Life Childhood Epilepsy Questionnaire (QOLCE) and Aberrant behavior checklist (ABC) are two such measures and consist of various subdomains which assess overall QOL, general health physical activities, emotional well-being, social functioning, behavioral and cognitive functioning, speech and stereotypic behavior.

**Objectives** We aim to compare the change in seizure profile and quality of life pre and post Clobazam treatment using standardized questionnaires.

**Methods** The study will be a four-phased, prospective, observational study of 30 pediatric patients meeting inclusion criteria, and consenting to participation. In the evaluation phase, after a baseline neurologic examination, seizure count will be recorded in a seizure diary. After one week of seizure count, Clobazam will be initiated at the pre-determined dose based on weight of the patient and titrated up during the follow-up visit at a 4-week interval, if needed. Patients will continue the drug for another 4-week period and be evaluated again at the end of the study. QOLCE and ABC questionnaire will be completed at each visit. Patients who improve on Clobazam and are willing to continue will do so and be followed up in the clinic. Anticipated time for completion of the study is about 12 months. Collected data will be analyzed at the conclusion of the study with the help of a statistician using paired t tests.

**Expected Results** At the conclusion of the study, we expect to see significant reduction in daily seizure frequency and overall improvement in QOL in our patients.

---

Dr. Nitin Agarwal was born and brought up in New Delhi, India. After finishing medical school at the University College of Medical Sciences in Delhi, he moved to the US for post-grad education. He is currently training in pediatric neurology and plans to go for an epilepsy fellowship on finishing his residency. He loves being outdoors, playing cricket and watching sports.
Photic Induced Seizures and Photoparoxysmal Response

Novreen Shahdad, MBBS, Arie Weinstock, MD

Department of Neurology, Jacobs Neurological Institute, SUNY at Buffalo, Buffalo, NY, USA

Background Seizures induced by visual stimuli can be observed in a wide variety of seizure types, both generalized and focal. They are characterized by changes in cortical excitability in response to photic stimulation. The cortical excitability is seen as abnormal spikes or spike and wave discharges on EEG known as Photoparoxysmal response (PPR). People with epilepsy have a 2-14% chance of having seizures precipitated by light or some sort of visual stimuli such as television, movie screen images, videogames, etc. Photo-sensitivity, an abnormal EEG response to light or pattern consisting of PPR, is seen in 0.3 to 3% of population.

It is not clear whether the patients with visually evoked seizures have a greater incidence of positive response on photic stimulation on EEG. The results of previous studies have been quite variable. In a retrospective study done at Washington University, PPR was found in 53% of patients with video game-induced seizures. In yet another European study done on video game-induced epilepsy, PPR was quoted at 85% of the subjects studied.

Objectives (1) To assess the yield of EEG in patients with photic induced seizures. (2) To determine the clinical significance of PPR on EEG.

Method This study will be performed as a retrospective database analysis of EEGs at WCHOB, Buffalo, NY. From the EEG database containing more than 50,000 EEGs, we will identify the EEGs of patients with photic-induced seizures and EEGs with PPR. These two patient populations will be studied with respect to their seizure type, clinical course, response to therapy, EEG findings and risk factors.

Conclusion Our goal is to describe the potential correlation of PPR in patients with photic-induced seizures. We will also study patterns of the risk factors of patients with PPR on EEG and their predilection to develop seizures and response to therapy.
Evaluating the risk of development of autoimmune diseases in multiple sclerosis patients treated with immunomodulatory therapies

Lynn Chouhfeh, MD, Barbara Teter, PhD, MPH, Bianca Weinstock-Guttman, MD

Department of Neurology, Jacobs Neurological Institute, SUNY at Buffalo, Buffalo, NY, USA

**Background**
An increased association of different autoimmune diseases (AID) was reported in multiple sclerosis (MS) patients. The relationship between a specific disease modifying therapy (DMT) and the risk of developing autoimmune diseases in MS patients is still unclear.

**Objectives**
1. To ascertain and compare the frequencies and types of AID between MS patients: DMT users vs. DMT naïve;
2. To investigate whether specific AID occur after particular DMT initiation;
3. Determine the most relevant demographic characteristics associated with comorbid autoimmune disorders and the relation to DMT use;
4. To investigate whether disability is influenced by AID comorbidity occurring in DMT naïve or after use of a specific DMT.

**Methods**
Retrospective case control study using data extracted from the NY State MS Consortium registry including patients with 5 years or more of follow-up available data. DMT users with AID will be individually examined to determine timing of AID vs. DMT initiation to identify the case group (AID after DMT initiation) compared to the “control” group. Bivariate testing to identify group differences in demographic and clinical characteristics will be performed using the chi-square and t-tests and ANOVA. Prediction of disability worsening will be conducted using logistic regression to adjust for covariates for the EDSS analysis and linear regression including adjustment for covariates for the MSSS analysis.

**Preliminary Results**
The study sample includes 85.2% DMT users (n=2,130) at the time of most recent follow-up, whereas 14.8% (n=370) remained DMT naïve. The prevalence of AID was 10.1% at the time of registration and 20.2% across the sample at time of most recent follow-up.

**Conclusion**
Comorbid AID may be triggered by specific immunomodulatory therapies in MS patients. Identifying risk factors associated with the development of AID in the context of a specific DMT may help in providing a more appropriate personalized therapeutic management of MS patients.
Graduation Dinner

Graduation Dinner
Westwood Country Club
772 North Forest Road
Buffalo, NY 14221
June 15, 2012

6:00 pm
Cocktails

7:00 pm
Dinner

Chairman’s Address:
Gil I. Wolfe, MD, FAAN

Michael E. Cohen Research Day Awards Presentation:
Michael E. Cohen, MD

Message from Outgoing Chief Resident:
Anil Neelakantan, MBBS

Message by Incoming Co-Chief Residents:
Salomi Salins, MBBS
Catherine Porter, MD

9:30 pm
End of Reception

This program was produced with the support of:
The Jacobs Neurological Institute (JNI) and Buffalo Neuroimaging Analysis Center (BNAC).