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For adjunctive treatment of partial-onset seizures with or without secondarily generalized seizures and primary generalized tonic-clonic seizures in patients with epilepsy 12 years of age and older

Change the Course of Epilepsy Treatment for Your Patients

EXPERIENCE THE STRENGTH OF FYCOMPA®

FYCOMPA ACHIEVED A STATISTICALLY SIGNIFICANT 76% (n=81) median reduction in seizure frequency vs placebo (38%; n=81) (*P*<0.0001)[1,2]

48% (n=39) of patients taking FYCOMPA exhibited a **75% to 100% REDUCTION IN PGTC SEIZURE FREQUENCY** vs placebo (24%; n=19)[1,3]

31% (n=25) of patients taking FYCOMPA experienced **PGTC SEIZURE-FREE STATUS** in the maintenance phase of the trial vs placebo (12%; n=10)[2,3*]

*Prespecified exploratory endpoint.[2]

- 50% to <75%: FYCOMPA, 16% (n=13) of patients; placebo, 16% (n=13) of patients[3]
- 25% to <50%: FYCOMPA, 15% (n=12) of patients; placebo, 20% (n=16) of patients[3]
- 0 to <25%: FYCOMPA, 11% (n=9) of patients; placebo, 12% (n=10) of patients[3]
- Seizure frequency increase: FYCOMPA, 10% (n=8) of patients; placebo, 28% (n=23) of patients[3]

PHASE 3 STUDY DESIGN

Multicenter, randomized, double-blind, placebo-controlled, parallel-group study on effectiveness of FYCOMPA as adjunctive therapy in patients 12 years of age and older. The total treatment period was 17 weeks (4: titration; 13: maintenance). Inclusion criteria included taking 1 to 3 concomitant AEDs at baseline and ≥3 PGTC seizures experienced in 8-week baseline period.[1]

ADVERSE REACTIONS in PGTC seizure study[1]

The most frequently (≥4%) reported adverse reactions for placebo (n=82) and FYCOMPA 8 mg (n=81), respectively: dizziness (6% and 32%), fatigue (6% and 15%), headache (10% and 12%), somnolence (4% and 11%), irritability (2% and 11%), vertigo (2% and 9%), vomiting (2% and 9%), weight gain (4% and 7%), contusion (4% and 6%), nausea (5% and 6%), abdominal pain (1% and 5%), anxiety (4% and 5%), urinary tract infection (1% and 4%), ligament sprain (0% and 4%), balance disorder (1% and 4%), and rash (1% and 4%).

Learn more at the

American Epilepsy Society 70th Annual Meeting

BOOTH 501

George R. Brown Convention Center, Houston, TX. December 2-6, 2016

REFERENCES: 1. FYCOMPA US Prescribing Information.Woodcliff Lake, NJ: Eisai Inc. **2.** French JA, Krauss GL, Wechsler RT, et al. Perampanel for tonic-clonic seizures in idiopathic generalized epilepsy: a randomized trial. *Neurology.* 2015;85(11):950-957. **3.** Data on file. Eisai Inc., Woodcliff Lake, NJ; 2015.

Please see Important Safety Information, including **Boxed WARNING**, and Brief Summary of full US Prescribing Information on the following pages.

VISIT **FYCOMPA.COM/HCP** FOR MORE INFORMATION

INDICATION

FYCOMPA® (perampanel) is indicated as adjunctive therapy for the treatment of partial-onset seizures with or without secondarily generalized seizures and primary generalized tonic-clonic seizures in patients with epilepsy 12 years of age and older.

IMPORTANT SAFETY INFORMATION

WARNING: SERIOUS PSYCHIATRIC AND BEHAVIORAL REACTIONS

- **Serious or life-threatening psychiatric and behavioral adverse reactions including aggression, hostility, irritability, anger, and homicidal ideation and threats have been reported in patients taking FYCOMPA**
- **These reactions occurred in patients with and without prior psychiatric history, prior aggressive behavior, or concomitant use of medications associated with hostility and aggression**
- **Advise patients and caregivers to contact a healthcare provider immediately if any of these reactions or changes in mood, behavior, or personality that are not typical for the patient are observed while taking FYCOMPA or after discontinuing FYCOMPA**
- **Closely monitor patients particularly during the titration period and at higher doses**
- **FYCOMPA should be reduced if these symptoms occur and should be discontinued immediately if symptoms are severe or are worsening**

SERIOUS PSYCHIATRIC AND BEHAVIORAL REACTIONS In the partial-onset seizures clinical trials, hostility- and aggression-related adverse reactions occurred in 12% and 20% of patients randomized to receive FYCOMPA at doses of 8 mg and 12 mg per day, respectively, compared to 6% of patients in the placebo group. These effects were dose related and generally appeared within the first 6 weeks of treatment, although new events continued to be observed through more than 37 weeks. These effects in FYCOMPA-treated patients led to dose reduction, interruption, and discontinuation more frequently than placebo-treated patients. The combination of alcohol and FYCOMPA significantly worsened mood and increased anger. Homicidal ideation and/or threat have also been reported postmarketing in patients treated with FYCOMPA. Patients taking FYCOMPA should avoid the use of alcohol. Patients, their caregivers, and families should be informed that FYCOMPA may increase the risk of psychiatric events. Patients should be monitored during treatment and for at least one month after the last dose of FYCOMPA, and especially when taking higher doses and during the initial few weeks of drug therapy (titration period) or at other times of dose increases. Similar serious psychiatric and behavioral events were observed in the primary generalized tonic-clonic (PGTC) seizure clinical trial.

SUICIDAL BEHAVIOR AND IDEATION Antiepileptic drugs (AEDs), including FYCOMPA, increase the risk of suicidal thoughts or behavior in patients. Anyone considering prescribing FYCOMPA or any other AED must balance the risk of suicidal thoughts or behavior with the risk of untreated illness. Epilepsy and many other illnesses for which AEDs are prescribed are themselves associated with morbidity and mortality and an increased risk of suicidal thoughts and behavior. Patients, their caregivers, and families should be informed of the risk and advised to monitor and immediately report the emergence or worsening of depression, suicidal thoughts or behavior, thoughts about self-harm and/or any unusual changes in mood or behavior. Should suicidal thoughts and behavior emerge during treatment, consider whether the emergence of these symptoms in any given patient may be related to the illness being treated.

DIZZINESS AND GAIT DISTURBANCE FYCOMPA caused dose-related increases in events related to dizziness and disturbance in gait or coordination. Dizziness and vertigo were reported in 35% and 47% of patients in the partial-onset seizure trials randomized to receive FYCOMPA at doses of 8 mg and 12 mg per day, respectively, compared to 10% of placebo-treated patients. Gait disturbance related events were reported in 12% and 16% of patients in the partial-onset seizure clinical trials randomized to receive FYCOMPA at doses of 8 mg and 12 mg per day, respectively, compared to 2% of placebo-treated patients. These adverse reactions occurred mostly during the titration phase. These adverse reactions were also observed in the PGTC seizure clinical trial.

SOMNOLENCE AND FATIGUE FYCOMPA caused dose-dependent increases in somnolence and fatigue-related events. Somnolence was reported in 16% and 18% of patients in the partial-onset seizure trials randomized to receive FYCOMPA at doses of 8 mg and 12 mg per day, respectively, compared to 7% of placebo patients. Fatigue related events were reported in 12% and 15% of patients in the partial-onset seizure trials randomized to receive FYCOMPA at doses of 8 mg and 12 mg per day, respectively, compared to 6% of placebo patients. These adverse reactions occurred mostly during the titration phase. These adverse reactions were also observed in the PGTC seizure clinical trial. Patients should be advised against engaging in hazardous activities requiring mental alertness, such as operating motor vehicles or dangerous machinery, until the effect of FYCOMPA is known.

FALLS Falls were reported in 5% and 10% of patients in the partial-onset seizure clinical trials randomized to receive FYCOMPA at doses of 8 mg and 12 mg per day, respectively, compared to 3% of placebo-treated patients.

WITHDRAWAL OF AEDs A gradual withdrawal is generally recommended with AEDs to minimize the potential of increased seizure frequency, but if withdrawal is a response to adverse events, prompt withdrawal can be considered.

MOST COMMON ADVERSE REACTIONS The most common adverse reactions in patients receiving FYCOMPA (≥5% and ≥1% higher than placebo) include dizziness, somnolence, fatigue, irritability, falls, nausea, weight gain, vertigo, ataxia, headache, vomiting, confusion, abdominal pain, and anxiety.

DRUG INTERACTIONS FYCOMPA may decrease the efficacy of contraceptives containing levonorgestrel. Plasma levels of FYCOMPA were decreased when administered with carbamazepine, phenytoin, or oxcarbazepine. Concomitant use of FYCOMPA with other strong CYP3A inducers (e.g., rifampin, St. John's wort) should be avoided. Multiple dosing of FYCOMPA 12 mg per day enhanced the effects of alcohol on vigilance and alertness, and increased levels of anger, confusion, and depression. These effects may also be seen when FYCOMPA is used in combination with other CNS depressants.

PREGNANCY AND LACTATION Physicians are advised to recommend that pregnant patients taking FYCOMPA enroll in the North American Antiepileptic Drug (NAAED) Pregnancy Registry. Caution should be exercised when FYCOMPA is administered to pregnant or nursing women as there are no adequate data on the developmental risk associated with use in pregnant women, and no data on the presence of perampanel in human milk, the effects on the breastfed child, or the effects of the drug on milk production.

HEPATIC AND RENAL IMPAIRMENT Use in patients with severe hepatic or severe renal impairment is not recommended. Dosage adjustments are recommended in patients with mild or moderate hepatic impairment. Use with caution in patients with moderate renal impairment.

DRUG ABUSE AND DEPENDENCE FYCOMPA is a Schedule III controlled substance and has the potential to be abused and lead to drug dependence.

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Initial U.S. Approval: 2012

Brief Summary of Full Prescribing Information dated April 2016

WARNING: SERIOUS PSYCHIATRIC AND BEHAVIORAL REACTIONS

- **Serious or life-threatening psychiatric and behavioral adverse reactions including aggression, hostility, irritability, anger, and homicidal ideation and threats have been reported in patients taking FYCOMPA (5.1).**
- **These reactions occurred in patients with and without prior psychiatric history, prior aggressive behavior, or concomitant use of medications associated with hostility and aggression (5.1).**
- **Advise patients and caregivers to contact a healthcare provider immediately if any of these reactions or changes in mood, behavior, or personality that are not typical for the patient are observed while taking FYCOMPA or after discontinuing FYCOMPA (5.1).**
- **Closely monitor patients particularly during the titration period and at higher doses (5.1).**
- **FYCOMPA should be reduced if these symptoms occur and should be discontinued immediately if symptoms are severe or are worsening (5.1).**

WARNINGS AND PRECAUTIONS

Serious Psychiatric and Behavioral Reactions [illegible]

Suicidal Behavior and Ideation [illegible]

Table 1. Risk by Indication for Antiepileptic Drugs in the Pooled Analysis

Indication	Placebo Patients with Events per 1000 Patients	Drug Patients with Events per 1000 Patients	Relative Risk: Incidence of Events in Drug Patients/Incidence in Placebo Patients	Risk Difference: Additional Drug Patients with Events per 1000 Patients
Epilepsy	1.0	3.4	3.5	2.4
Psychiatric	5.7	8.5	1.5	2.9
Other	1.0	1.8	1.9	0.9
Total	2.4	4.3	1.8	1.9

[illegible]

Neurologic Effects [illegible]

Falls [illegible]

Withdrawal of Antiepileptic Drugs [illegible]

ADVERSE REACTIONS

Clinical Trials Experience [illegible]

Table 2. Adverse Reactions in Pooled Placebo-Controlled Trials in Patients with Partial-Onset Seizures (Studies 1, 2, and 3) (Reactions ≥2% of Patients in Highest FYCOMPA Dose (12 mg) Group and More Frequent than Placebo)

	Placebo n=442 %	FYCOMPA 4 mg n=172 %	FYCOMPA 8 mg n=431 %	FYCOMPA 12 mg n=255 %
Dizziness	9	16	32	43
Somnolence	7	9	16	18
Headache	11	11	11	13
Irritability	3	4	7	12
Fatigue	5	8	8	12
Falls	3	2	5	10
Ataxia	0	1	3	8
Nausea	5	3	6	8
Vertigo	1	4	3	5
Back pain	2	2	2	5
Dysarthria	0	1	3	4
Anxiety	1	2	3	4
Blurred vision	1	1	3	4
Gait disturbance	1	1	4	4
Weight gain	1	4	4	4
Cough	3	1	1	4
Upper respiratory tract infection	3	3	3	4
Vomiting	2	2	3	4
Hypersomnia	0	1	1	3
Anger	<1	0	1	3
Aggression	1	1	2	3
Balance disorder	1	0	5	3
Diplopia	1	1	1	3
Head injury	1	1	1	3
Hypoesthesia	1	0	0	3
Pain in extremity	1	0	0	3
Constipation	2	2	2	3
Myalgia	2	1	1	3
Coordination abnormal	0	1	<1	3

Table 2. Adverse Reactions in Pooled Placebo-Controlled Trials in Patients with Partial-Onset Seizures (Studies 1, 2, and 3) (Reactions ≥ 2% of Patients in Highest FYCOMPA Dose (12 mg) Group and More Frequent than Placebo) (cont.)

Euphoric mood	0	0	<1	2
Confusional state	<1	1	1	2
Hypersomnia	<1	0	0	2
Limb injury	<1	1	1	2
Mood altered	<1	1	<1	2
Arthralgia	1	0	2	2
Asthenia	1	1	2	2
Contusion	1	0	2	2
Memory impairment	1	0	1	2
Musculoskeletal pain	1	1	1	2
Oropharyngeal pain	1	2	2	2
Paresthesia	1	0	1	2
Peripheral edema	1	1	1	2
Skin laceration	1	0	2	2

Primary Generalized Tonic-Clonic Seizures: A total of 81 patients receiving FYCOMPA 8 mg once daily constituted the safety population in the placebo-controlled trial in patients with primary generalized tonic-clonic seizures (Study 4). Approximately 57% of patients were female, and the mean age was 27 years. In the controlled primary generalized tonic-clonic seizure clinical trial (Study 4), the adverse reaction profile was similar to that noted for the controlled partial-onset seizure clinical trials (Studies 1, 2, and 3). Table 3 gives the incidence of adverse reactions in patients receiving FYCOMPA 8 mg (≥4%) and higher than in the placebo group in Study 4. The most common adverse reactions in patients receiving FYCOMPA (≥10% and greater than placebo) were dizziness (32%), fatigue (15%), headache (12%), somnolence (11%), and irritability (11%). The adverse reactions most commonly leading to discontinuation in patients receiving FYCOMPA 8 mg (≥2% and greater than placebo) were vomiting (2%) and dizziness (2%).

Table 3. Adverse Reactions in a Placebo Controlled Trial in Patients with Primary Generalized Tonic-Clonic Seizures (Study 4) (Reactions ≥ 4% of Patients in FYCOMPA Group and More Frequent than Placebo)

	Placebo (n=82) %	FYCOMPA 8 mg (n=81) %
Dizziness	6	32
Fatigue	6	15
Headache	10	12
Somnolence	4	11
Irritability	2	11
Vertigo	2	8
Vomiting	2	8
Weight gain	4	7
Contusion	4	5
Nausea	5	6
Abdominal pain	1	5
Anxiety	4	5
Urinary tract infection	1	4
Ligament sprain	0	4
Balance disorder	1	4
Rash	1	4

Weight Gain: Weight gain has occurred with FYCOMPA. In controlled partial-onset seizure clinical trials, FYCOMPA-treated adults gained an average of 1.1 kg (2.5 lbs) compared to an average of 0.3 kg (0.7 lbs) in placebo-treated adults with a median exposure of 19 weeks. The percentages of adults who gained at least 7% and 15% of their baseline body weight in FYCOMPA-treated patients were 9.1% and 0.9%, respectively, as compared to 4.5% and 0.2% of placebo-treated patients, respectively. Clinical monitoring of weight is recommended. Similar increases in weight were also observed in adult and pediatric patients treated with FYCOMPA in the primary generalized tonic-clonic seizure clinical trial. Elevated triglycerides: Increases of triglycerides occurred with FYCOMPA use. Comparison of Sex and Race: No significant sex differences were noted in the incidence of adverse reactions. Although there were few non-Caucasian patients, no differences in the incidence of adverse reactions compared to Caucasian patients were observed. **Postmarketing Experience** The following adverse reactions have been identified during post-approval use of FYCOMPA. Because these reactions are reported voluntarily from a population of uncertain size, it is not always possible to reliably estimate their frequency or establish a causal relationship to drug exposure.
Dermatologic: Drug Reaction with Eosinophilia and Systemic Symptoms (DRESS).
Psychiatric: acute psychosis, hallucinations, delusions, paranoia, delirium, confusional state, disorientation, memory impairment.

DRUG INTERACTIONS

Contraceptives With concomitant use, FYCOMPA at a dose of 12 mg per day reduced levonorgestrel exposure by approximately 40% [see Clinical Pharmacology (12.3)]. Use of FYCOMPA with oral or implant contraceptives containing levonorgestrel may render them less effective. Additional non-hormonal forms of contraception are recommended. **Cytochrome P450 Inducers** The concomitant use of known moderate and strong CYP3A inducers including carbamazepine, phenytoin, or oxcarbazepine with FYCOMPA decreased the plasma levels of perampanel by approximately 50-67% [see Clinical Pharmacology (12.3)]. The starting doses for FYCOMPA should be increased in the presence of enzyme-inducing AEDs [see Dosage and Administration (2.3)]. When these enzyme-inducing AEDs are introduced or withdrawn from a patient's treatment regimen, the patient should be closely monitored for clinical response and tolerability. Dose adjustment of FYCOMPA may be necessary [see Dosage and Administration (2.3)]. Concomitant use of FYCOMPA with other strong CYP3A inducers (e.g., rifampin, St. John's wort) is not recommended. **Alcohol and Other CNS Depressants** The concomitant use of FYCOMPA and CNS depressants, including alcohol, may increase CNS depression. A pharmacodynamic interaction study in healthy subjects found that the effects of FYCOMPA on complex tasks such as driving ability were additive or supra-additive to the impairment effects of alcohol [see Clinical Pharmacology (12.2)]. Multiple dosing of FYCOMPA 12 mg per day also enhanced the effects of alcohol to interrupt vigilance and alertness, and increased levels of anger, confusion, and depression. These effects may also be seen when FYCOMPA is used in combination with other CNS depressants. Care should be taken when administering FYCOMPA with these agents. Patients should limit activity until they have experience with concomitant use of CNS depressants (e.g., benzodiazepines, narcotics, barbiturates, sedating antihistamines). Advise patients not to drive or operate machinery until they have gained sufficient experience on FYCOMPA to gauge whether it adversely affects these activities.

USE IN SPECIFIC POPULATIONS

Pregnancy To provide information regarding the effects of in utero exposure to FYCOMPA, physicians are advised to recommend that pregnant patients taking FYCOMPA enroll in the North American Antiepileptic Drug (NAAED) Pregnancy Registry. This can be done by calling the toll free number 1-888-233-2334, and must be done by patients themselves. Information on the registry can also be found at the website http://www.aedpregnancyregistry.org/. There are no adequate and well-controlled studies in pregnant women. In animal reproduction studies, perampanel induced developmental toxicity in pregnant rat and rabbit at clinically relevant doses. Oral administration of perampanel (1, 3, or 10 mg/kg/day) to pregnant rats throughout organogenesis resulted in an increase in visceral abnormalities (diverticulum of the intestine) at all doses tested. In a dose-ranging study at higher oral doses (10, 30, or 60 mg/kg/day), embryo lethality and reduced fetal body weight were observed at the mid and high doses tested. The lowest dose tested (1 mg/kg/day) is similar to a human dose of 8 mg per day based on body surface area (mg/m²). Upon oral administration of perampanel (1, 3, or 10 mg/kg per day) to pregnant rabbits throughout organogenesis, embryo lethality was observed at the mid and high doses tested; the no-effect dose for embryo-fetal developmental toxicity in rabbit (1 mg/kg/day) is approximately 2 times a human dose of 8 mg per day based on body surface area (mg/m²). Oral administration of perampanel (1, 3, or 10 mg/kg per day) to rats throughout gestation and lactation resulted in fetal and pup deaths at the mid and high doses and delayed sexual maturation in males and females at the highest dose tested. No effects were observed on measures of neurobehavioral or reproductive function in the offspring. The no-effect dose for pre- and postnatal developmental toxicity in rat (1 mg/kg/day) is similar to a human dose of 8 mg per day based on body surface area (mg/m²). **Lactation** There are no data on the presence of perampanel in human milk, the effects of perampanel on the breastfed infant, or the effects of the drug on milk production. Perampanel and/or its metabolites are present in rat milk, and are detected at concentrations higher than that in maternal plasma. The developmental and health benefits of breastfeeding should be considered along with the mother's clinical need for FYCOMPA and any potential adverse effects on the breastfed infant from FYCOMPA or from the underlying maternal condition. **Females and Males of Reproductive Potential** Use of FYCOMPA with oral or implant contraceptives containing levonorgestrel may render them less effective. Additional non-hormonal forms of contraception are recommended [see Clinical Pharmacology (12.3)]. **Pediatric Use** The safety and efficacy of FYCOMPA for the adjunctive therapy of partial-onset seizures in pediatric patients 12 years of age and older was established by three randomized double-blind, placebo-controlled, multicenter studies, which included 72 pediatric patients between 12 and 16 years of age exposed to FYCOMPA. The safety and efficacy of FYCOMPA for the adjunctive therapy of primary generalized tonic-clonic seizures in pediatric patients 12 years of age and older was established in a single randomized double-blind, placebo-controlled, multicenter trial (n=164), which included 11 pediatric patients 12 to 16 years of age exposed to FYCOMPA; an additional 6 patients were treated with FYCOMPA in the open-label extension of the study. The safety and effectiveness of FYCOMPA in pediatric patients less than 12 years of age have not been established [see Clinical Pharmacology (12.3), Clinical Studies (14.1)]. Juvenile Animal Data: Oral administration of perampanel (1, 5, 5/10/30 mg/kg/day; high dose increased on postnatal days [PND] 28 and 56) to young rats for 12 weeks starting on PND 7 resulted in reduced body weight, reduced growth, neurobehavioral impairment (water maze performance and auditory startle habituation) at the mid and high doses, and delayed sexual maturation at the high dose. CNS signs (reduced activity, incoordination, excessive grooming/pawing/licking, pup death), decreased hindlimb splay, and decreased hindlimb grip strength were observed at all doses. Effects on pup body weight, pup growth, hindlimb splay, impairment in the water maze performance, and auditory startle persisted after dosing was stopped. A no-effect dose for postnatal developmental toxicity was not identified in this study. Oral administration of perampanel (1, 5, 5/10 mg/kg/day; high dose increased on PND 56) to juvenile dogs for 33 weeks, starting on PND 42, resulted in CNS signs (incoordination, excessive grooming/licking/scratching, spatial disorientation, and/or ataxic gait) at all doses tested. **Geriatric Use** Clinical studies of FYCOMPA did not include sufficient numbers of patients aged 65 and over to determine the safety and efficacy of FYCOMPA in the elderly population. Because of increased likelihood for adverse reactions in the elderly, dosing titration should proceed slowly in patients aged 65 years and older [see Dosage and Administration (2.5)]. **Hepatic Impairment** Use of FYCOMPA in patients with severe hepatic impairment is not recommended, and dosage adjustments are recommended in patients with mild or moderate hepatic impairment [see Dosage and Administration (2.4), Clinical Pharmacology (12.3)]. **Renal Impairment** Dose adjustment is not required in patients with mild renal impairment. FYCOMPA should be used with caution in patients with moderate renal impairment, and slower titration may be considered. Use in patients with severe renal impairment or patients undergoing hemodialysis is not recommended [see Dosage and Administration (2.6), Clinical Pharmacology (12.3)].

DRUG ABUSE AND DEPENDENCE

Controlled Substance FYCOMPA contains perampanel and is listed as a Schedule III controlled substance. **Abuse** Prescription drug abuse is the intentional non-therapeutic use of a drug, even once, for its rewarding psychological or physiological effects. Drug addiction, which develops after repeated drug abuse, is characterized by a strong desire to take a drug despite harmful consequences, difficulty in controlling its use, giving a higher priority to drug use than to obligations, increased tolerance, and sometimes physical withdrawal. Drug abuse and drug addiction are separate and distinct from physical dependence (for example, abuse may not be accompanied by physical dependence) [see Drug Abuse and Dependence (9.3)]. Studies of human abuse potential were performed to evaluate the abuse potential of FYCOMPA (8 mg, 24 mg, and 36 mg) as compared to alprazolam C-IV (1.5 mg and 3 mg), and oral ketamine C-III (100 mg) in recreational polydrug users. Supra-therapeutic doses of FYCOMPA 24 and 36 mg produced responses for "Euphoria" that were similar to ketamine 100 mg and alprazolam 3 mg. For "High," FYCOMPA 24 mg and 36 mg produced responses comparable to ketamine 100 mg and significantly higher than both doses of alprazolam on a visual analog scale (VAS). "Drug Liking," "Overall Drug Liking," and "Take Drug Again" for FYCOMPA were each statistically lower than ketamine 100 mg. In addition, for "Bad Drug Effects," FYCOMPA 24 and 36 mg produced responses significantly higher than ketamine 100 mg. For "Sedation," FYCOMPA 24 and 36 mg produced responses similar to alprazolam 3 mg and lower than ketamine 100 mg. Additionally, on VAS measures related to dissociative phenomena such as "Floating," "Spaced Out," and "Detached," FYCOMPA at supra-therapeutic doses produced responses similar to ketamine 100 mg and greater than both doses of alprazolam tested. Of note, due to somnolence a number of subjects had missing data around T_{max} of FYCOMPA. The above described data might represent an underestimate of FYCOMPA's effects. The duration of effects of higher doses of FYCOMPA on the majority of measures was much greater than alprazolam 3 mg and ketamine 100 mg. In the study, the incidence of euphoria following FYCOMPA administration 8 mg, 24 mg, and 36 mg was 37%, 46%, 46%, respectively, which was higher than alprazolam 3 mg (13%) but lower than ketamine 100 mg (89%). **Dependence** Physical dependence is characterized by withdrawal symptoms after abrupt discontinuation or a significant dose reduction of a drug. The potential for FYCOMPA to produce withdrawal symptoms has not been adequately evaluated.

OVERDOSAGE

There is limited clinical experience with FYCOMPA overdose. The highest reported overdose (approximately 264 mg) was intentional. This patient experienced serious adverse reactions of altered mental status, agitation, and aggressive behavior and recovered without sequelae. In general, the adverse reactions associated with overdoses were similar to the reactions at therapeutic doses, with dizziness reported most frequently. There were no reported sequelae. There is no available specific antidote to the overdose reactions of FYCOMPA. In the event of overdose, standard medical practice for the management of any overdose should be used. An adequate airway, oxygenation, and ventilation should be ensured; monitoring of cardiac rhythm and vital sign measurement is recommended. A certified poison control center should be contacted for updated information on the management of overdose with FYCOMPA. Due to its long half-life, the reactions caused by FYCOMPA could be protracted.

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References: 1. Qudexy XR [package insert]. Maple Grove, MN: Upsher-Smith Laboratories, Inc.; March 2015.
2. Topamax Tablets [package insert]. Titusville, NJ: Janssen Pharmaceuticals, Inc.; December 2014.

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HOUSTON WELCOME LETTER

CITY OF HOUSTON
Office of the Mayor

Sylvester Turner
Mayor
P.O. Box 1562
Houston, Texas 77251-1562
Telephone – Dial 311
www.houstontx.gov

December 1, 2016

Greetings;

On behalf of the city of Houston, I welcome you to our great city.

It is truly a pleasure to have the American Epilepsy Society in Houston for the 70th Annual Meeting.

Houston is wonderfully diverse in its people, cultures and ideas. More than 30 million people visit the Greater Houston area every year to experience our award-winning restaurants, world class museums, thrilling sports arenas and attractions.

Houston is home to the Texas Medical Center, the largest life sciences destination in the world. With 106,000 employees, 54 institutions, and thousands of volunteers and patient visits, over 160,000 people visit Texas Medical Center each day. Over the course of the year, the campus welcomes more than 7.2 million visitors.

Today, people come from across the country and all over the world to receive care from the best doctors in their field, including epilepsy, at Texas Medical Center. Incredible breakthroughs in diagnosis and treatment happen at the campuses eight different academic and research institutions, not to mention the 21 different hospitals.

It's my hope that all of you enjoy your time in Houston, and you have a productive and very successful annual meeting.

Warm regards,

Sylvester Turner
Mayor

PRESIDENT'S WELCOME LETTER

On behalf of the American Epilepsy Society (AES) Board of Directors, the Annual Meeting program committees, and AES staff: Welcome to the 70th AES Annual Meeting!

We are here in Houston to discuss where our work in epilepsy stands today, what our roadblocks are, and what we would like to see from our field for the persons with epilepsy we serve in the future. We've brought the world's most brilliant epilepsy professionals together under one roof for this Annual Meeting, and I look forward to seeing the results of the learning that will take place, the partnerships that will be formed, and the inspired ideas that will take root.

I am incredibly proud of the society's recent accomplishments. This year alone, we released a new guideline on status epilepticus, placing many of the newer treatments in a modern context. Also on the clinical front, we have partnered with the American Academy of Neurology (AAN), Child Neurology Foundation and others on performance measures in adult and pediatric epilepsy and with AAN on updating guidelines on AED efficacy and tolerability. Our journal, *Epilepsy Currents*, achieved an astonishing impact factor of 5.615, making it one of the highest-rated epilepsy journals—a tribute to the hard work of our editors and authors.

Additionally, an exceptional new "Ask the Experts" webinar series launched this year, and the recordings are available on the website. New self-assessment modules continue to provide members with an easy way to meet MOC requirements and prepare for the certification exam. On that front, watch soon for new PI-CME activities to meet certification requirements and measurably improve patient care. A new suite of tools to address psychosocial comorbidities was developed. And AES is now a partner in the Managing Epilepsy Well (MEW) network of universities, community-based organizations, and the Centers for Disease Control and Prevention, helping to disseminate validated patient self-management tools.

We launched the Fellow of the American Epilepsy Society (FAES) program designed to recognize those members who have made exceptional commitments to AES and the field of epilepsy. We have increased our support for ground-breaking, early-career, and trainee-focused research. And finally, our major awards programs for achievement in epilepsy had quite the challenge this year because of the large number of remarkable clinicians and scientists who have boldly forged new advances in the field.

The bottom line is this: we are changing lives and I am honored to join such amazing colleagues and contribute to such influential work!

I would like to personally invite you to join me at this year's Presidential Symposium—Epilepsy Care: A Futurist View. I have challenged each of the speakers to describe what the future may hold for epilepsy research and treatment—join me to learn what they foresee. It will take place at 8:30 a.m. on Saturday, December 3. I look forward to seeing you there.

Sincerely,

Michael D. Privitera, M.D.
2016 President, American Epilepsy Society

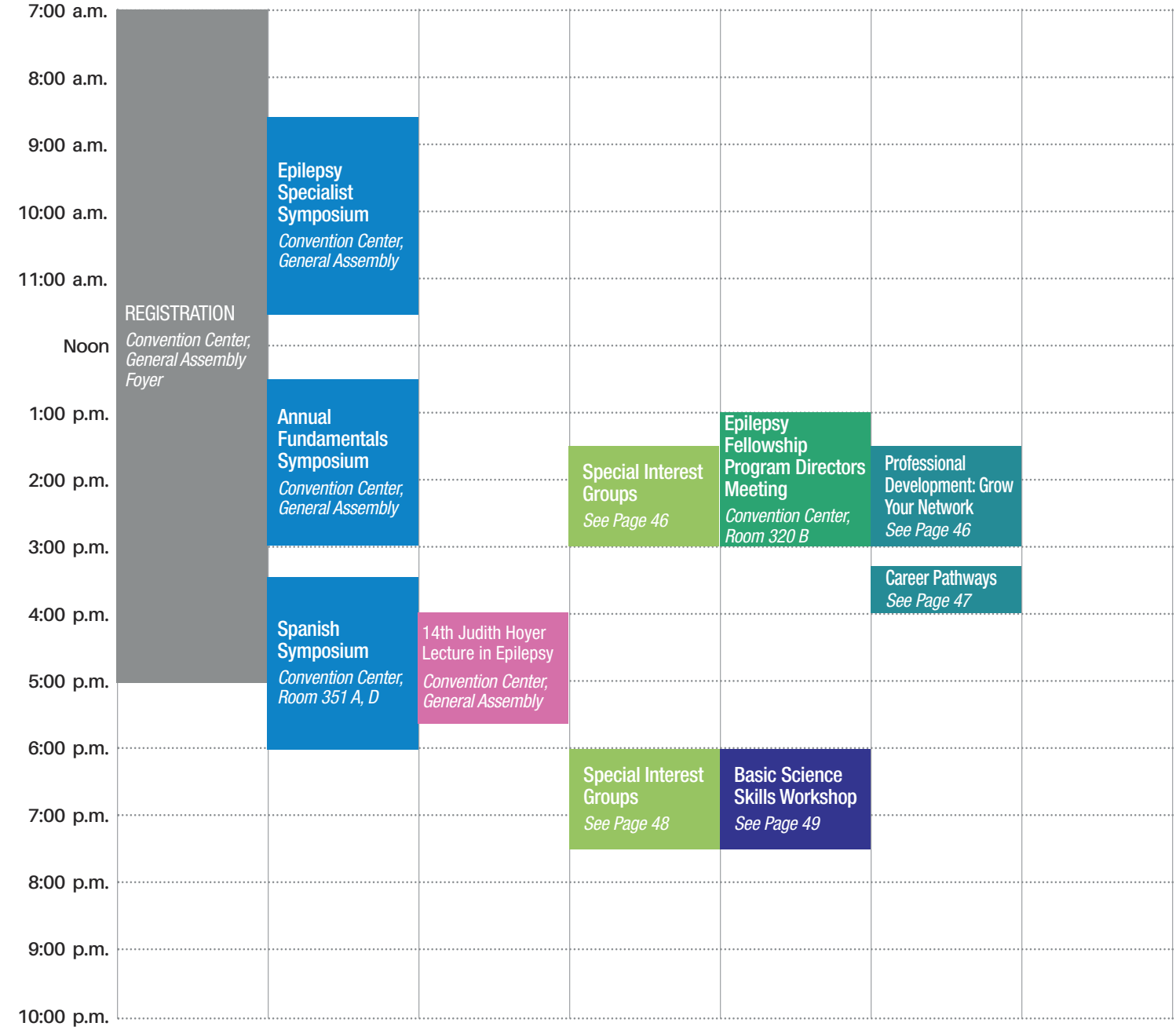
SCHEDULE AT A GLANCE

THURSDAY, DECEMBER 1

REGISTRATION

Thursday, December 1 | 5:00 p.m. - 7:00 p.m.
Convention Center, General Assembly Foyer

FRIDAY, DECEMBER 2



SYMPOSIA

Epilepsy Specialist Symposium | Choosing from the Feast of Epilepsy Surgery Procedures: How Do We Decide the Best Course for Each Patient?

Annual Fundamentals Symposium | The New Definition and Classification of Epilepsy

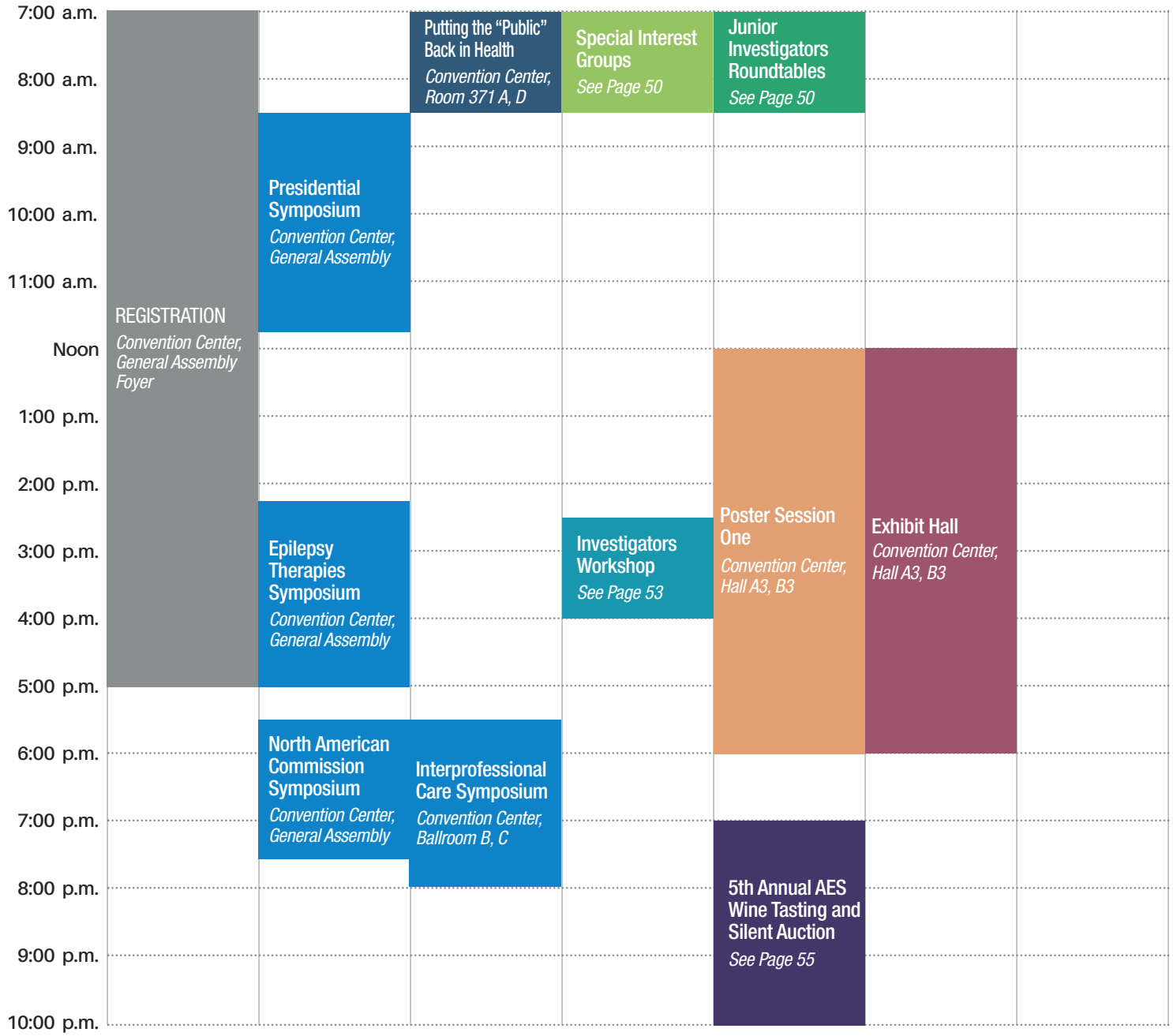
Spanish Symposium | How to Evaluate and Ameliorate the Treatment Gap in Epilepsy: Important Considerations for Spanish-speaking Countries. *Presented in Spanish.*

LECTURES AND MORE

14th Judith Hoyer Lecture in Epilepsy | The SUDEP Movement: From Inception to the Goal

SCHEDULE AT A GLANCE

SATURDAY, DECEMBER 3



amRUTaMAD

U9u:2ru6,2n4B|587:2.5 | Epilepsy Care: A Futurist View

Epilepsy Therapies Symposium | Risky Business: From Repetitive Seizures to Status

North American Commission Symposium | Controversies in the Management of Epilepsy During Pregnancy

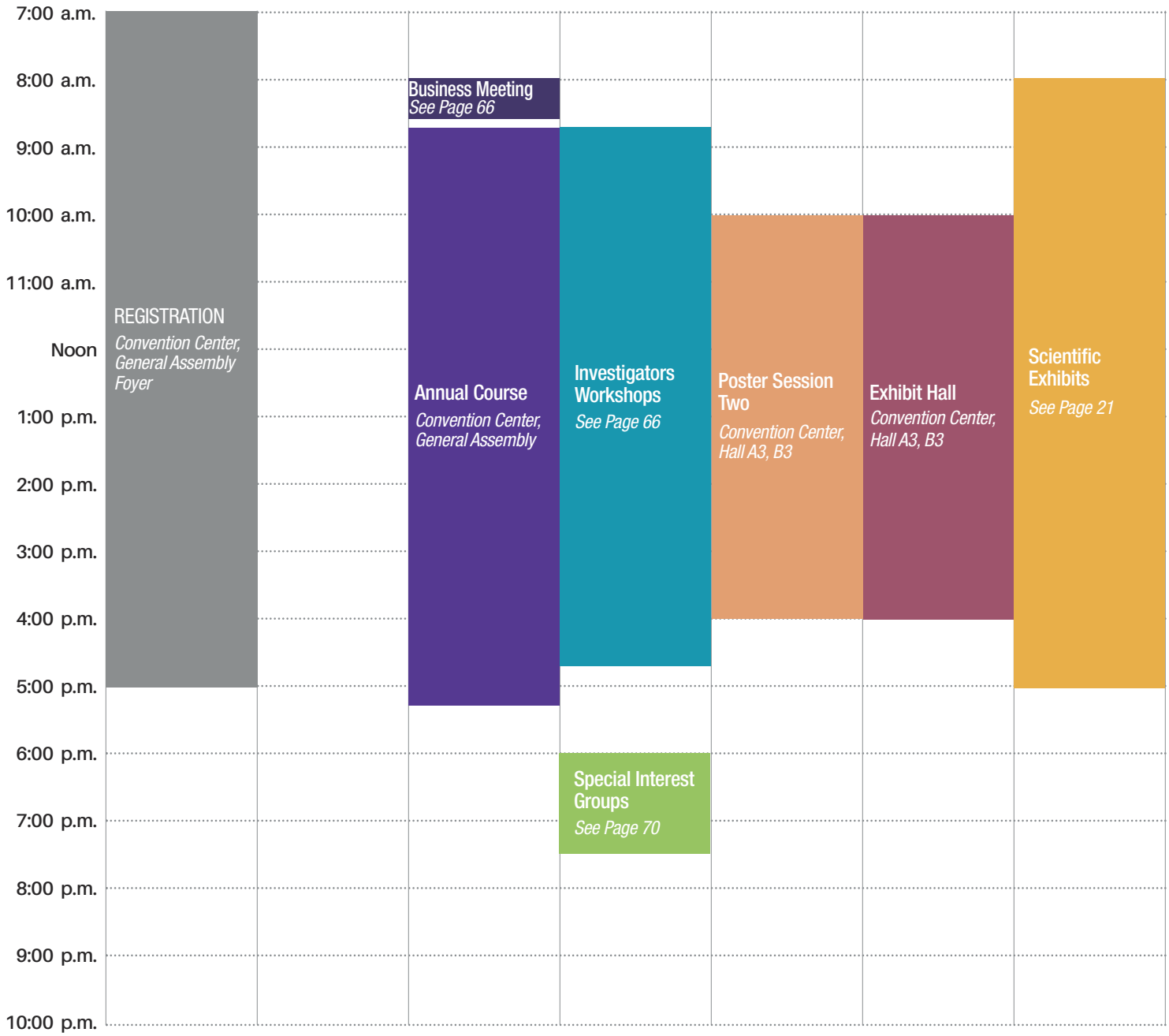
Interprofessional Care Symposium | Interprofessional Assessment and Intervention of the Psychosocial Comorbidities of Epilepsy

LECTURES AND MORE

Putting the "Public" Back in Health | Resources and Opportunities Available to People with Epilepsy

SCHEDULE AT A GLANCE

SUNDAY, DECEMBER 4



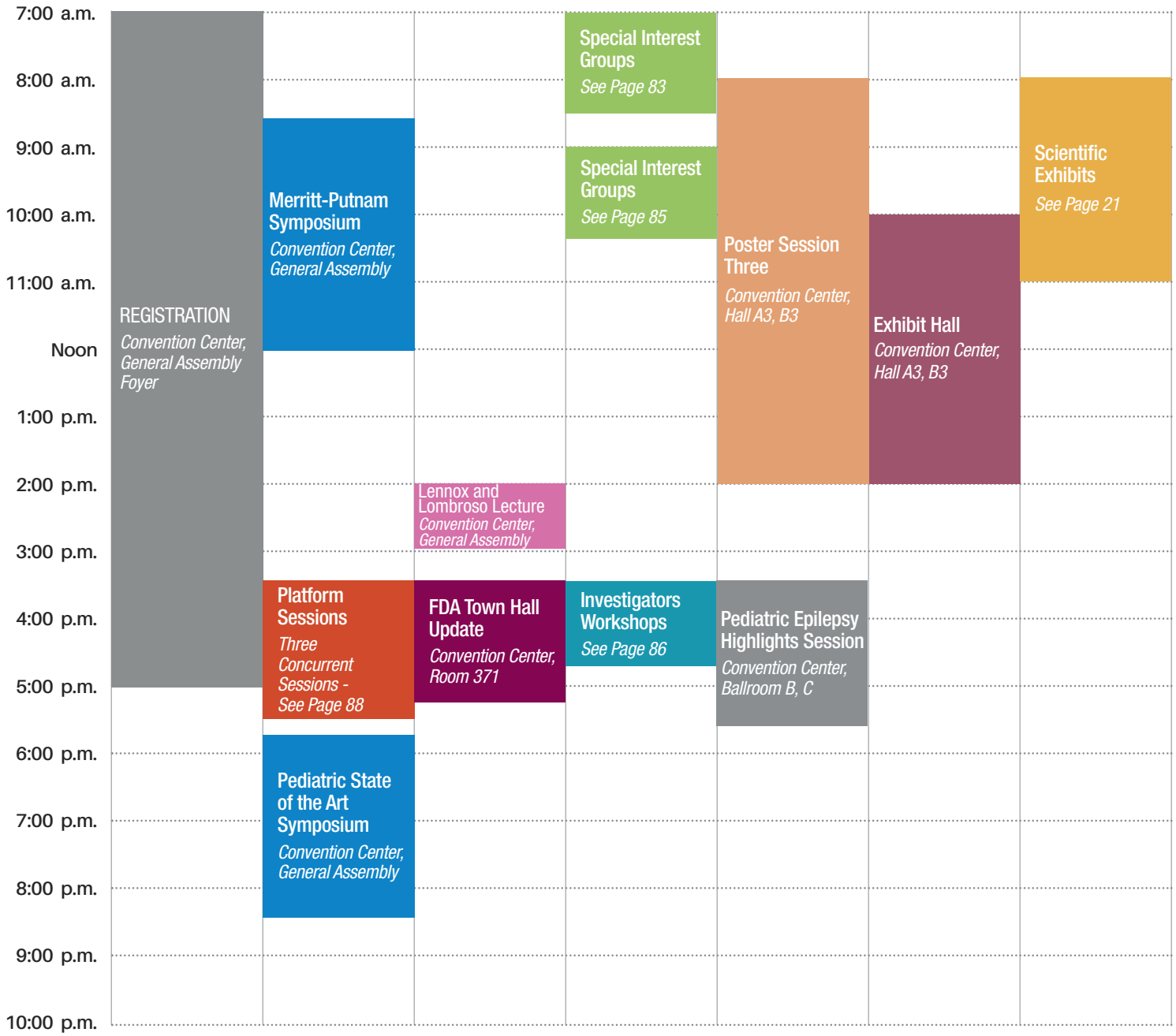
ANNUAL COURSE

Annual Course | When All Else Fails: Intractable Epilepsy — Pathophysiology to Treatment

#AES2016

SCHEDULE AT A GLANCE

MONDAY, DECEMBER 5



SYMPOSIA

Merritt-Putnam Symposium | Multiscale Imaging of Seizures and Epilepsy

Pediatric State of the Art Symposium | Tuberous Sclerosis Complex (TSC): Understanding and Modifying Epileptogenesis

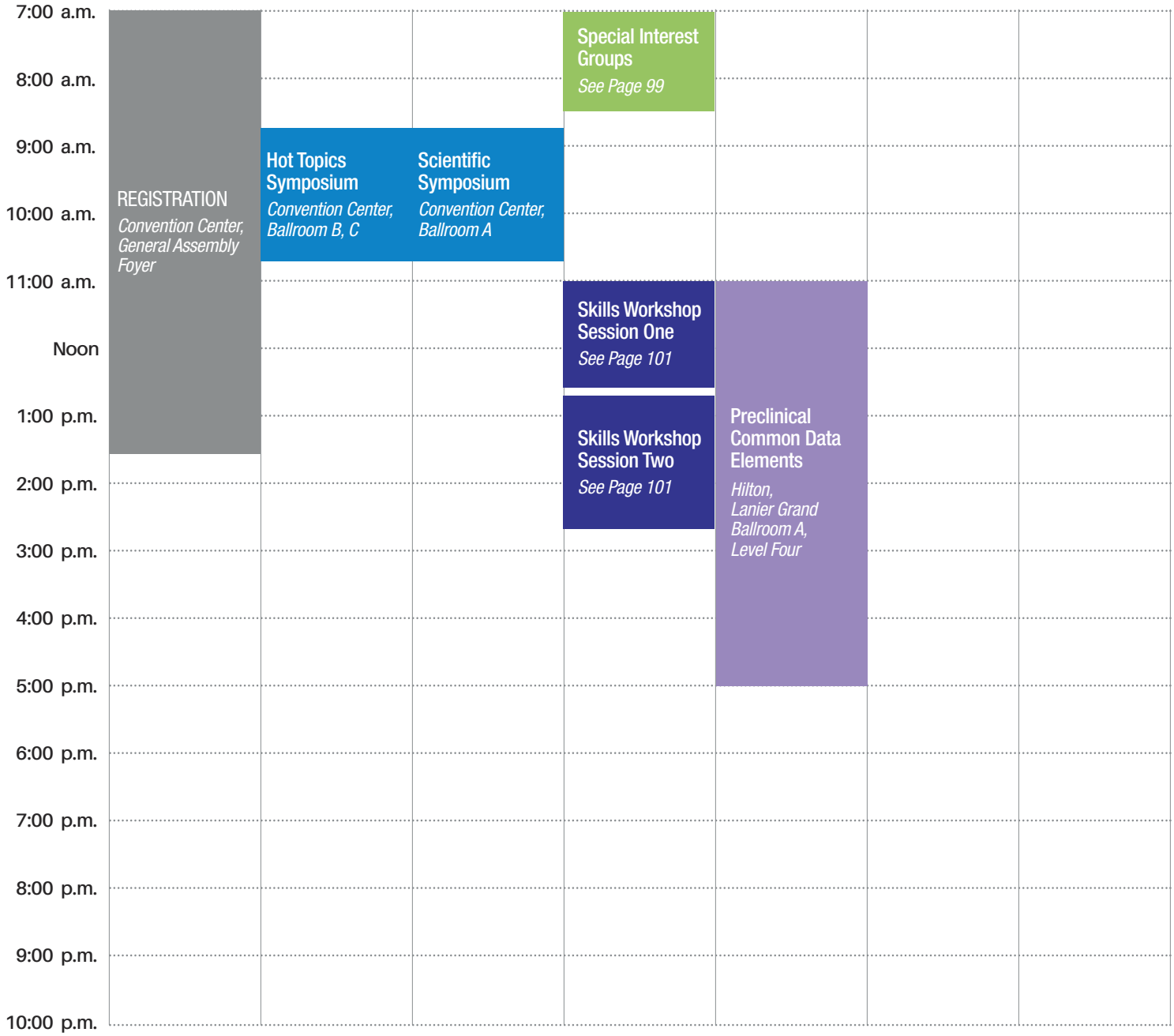
81-HLF1GDA0D9BF1D

8doorBocBrnatrurBdbvytd | EEG, the New Frontier

FDA Town Hall Update | Therapeutic Equivalence of Generic Antiepileptic Drugs

SCHEDULE AT A GLANCE

TUESDAY, DECEMBER 6



SYMPOSIA

Hot Topics Symposium

Scientific Symposium | The Neurobiology of Brain Stimulation in Epilepsy: Targets, Networks and Cascades

AESnet.org

SPECIAL INTEREST GROUP SCHEDULES

FRIDAY, DECEMBER 2

1:30 p.m. – 3:00 p.m.	**Epidemiology** *Convention Center, Room 360 A, D*	6:00 p.m. – 7:30 p.m.	**Ictal Semiology** *Convention Center, Room 361 A, D*
1:30 p.m. – 3:00 p.m.	**Global Health in Epilepsy** *Convention Center, Room 330 A*	6:00 p.m. – 7:30 p.m.	**Neuroendocrinology** *Convention Center, Room 330 A*
1:30 p.m. – 3:00 p.m.	**Neurostimulation/Epilepsy and Engineering** *Convention Center, Room 361 A, D*	6:00 p.m. – 7:30 p.m.	**Neuroimaging** *Convention Center, Room 371 A, D*
1:30 p.m. – 3:00 p.m.	**Surgery** *Convention Center, Room 371 A, D*	6:00 p.m. – 7:30 p.m.	**Nursing** *Convention Center, Room 330 B*
1:30 p.m. – 3:00 p.m.	**Translational Research** *Convention Center, Room 370 A, D*	6:00 p.m. – 7:30 p.m.	**Seizure and Cerebrovascular Disease** *Convention Center, Room 310 A*
6:00 p.m. – 7:30 p.m.	**Epilepsy and Aging** *Convention Center, Room 310 B*		

SATURDAY, DECEMBER 3

7:00 a.m. – 8:30 a.m.	**Basic Mechanisms and Neuroscience** *Convention Center, Room 360 A, D*	7:00 a.m. – 8:30 a.m.	**Frontal Lobe** *Convention Center, Room 310 B*
7:00 a.m. – 8:30 a.m.	**Critical Care Epilepsy** *Convention Center, Room 330 A*	7:00 a.m. – 8:30 a.m.	**Genetics** *Convention Center, Room 361 A, D*
7:00 a.m. – 8:30 a.m.	**Dietary Therapies for Epilepsy** *Convention Center, Room 370 A, D*	7:00 a.m. – 8:30 a.m.	**Neonatal Seizures** *Convention Center, Room 320 B*

SUNDAY, DECEMBER 4

6:00 p.m. – 7:30 p.m.	**Children's Hour** *Convention Center, Room 361 A, D*	6:00 p.m. – 7:30 p.m.	**Sleep and Epilepsy** *Convention Center, Room 320 A*
6:00 p.m. – 7:30 p.m.	**Psychosocial Comorbidities** *Convention Center, Room 310 B*	6:00 p.m. – 7:30 p.m.	**Temporal Lobe Club** *Convention Center, Room 371 A, D*
6:00 p.m. – 7:30 p.m.	**Quality, Value and Safety in Epilepsy** *Convention Center, Room 310 A*		

MONDAY, DECEMBER 5

7:00 a.m. – 8:30 a.m.	**NIH and Non-Profit Research Resources/Junior Investigator Workshop** *Convention Center, Room 320 A*	7:00 a.m. – 8:30 a.m.	**Tumor-related Epilepsy** *Convention Center, Room 310 B*
7:00 a.m. – 8:30 a.m.	**Practice Management** *Convention Center, Room 310 A*	7:00 a.m. – 8:30 a.m.	**Women with Epilepsy** *Convention Center, Room 320 B*
7:00 a.m. – 8:30 a.m.	**Private Practice Epilepsy** *Convention Center, Room 370 A, D*	9:00 a.m. – 10:30 a.m.	**Cognitive and Behavioral Treatments** *Convention Center, Room 320 B*
7:00 a.m. – 8:30 a.m.	**SUDEP** *Convention Center, Room 361 A,D*	9:00 a.m. – 10:30 a.m.	**MEG/MSI** *Convention Center, Room 361 A, D*
		9:00 a.m. – 10:30 a.m.	**Pediatric Epilepsy Case Discussions** *Convention Center, Ballroom A*

TUESDAY, DECEMBER 6

7:00 a.m. – 8:30 a.m.	**EEG** *Convention Center, Room 310 B*	7:00 a.m. – 8:30 a.m.	**Psychogenic Nonepileptic Seizures (PNES)** *Convention Center, Room 370 A, D*
7:00 a.m. – 8:30 a.m.	**Neuropharmacology** *Convention Center, Room 361 A, D*	7:00 a.m. – 8:30 a.m.	**Tuberous Sclerosis** *Convention Center, Room 320 B*
7:00 a.m. – 8:30 a.m.	**Neuropsychology** *Convention Center, Room 371 A, D*		

POSTER SESSION SCHEDULES

SATURDAY, DECEMBER 3

Poster Session One

Noon – 6:00 p.m.
Convention Center, Hall A3, B3

Authors Present: Noon – 2:00 p.m.
Poster Walking Tours: 12:15 p.m. – 1:45 p.m.

Translational Research	1.001 – 1.089
Interprofessional Care/Professionals in Epilepsy	1.090 – 1.099
Neurophysiology	1.100 – 1.154
Clinical Epilepsy	1.155 – 1.215
Neuroimaging	1.216 – 1.244
Comorbidities (Somatic and Psychiatric)	1.245 – 1.254
Antiepileptic Drugs	1.255 – 1.290
Surgery	1.291 – 1.314
Behavior / Neuropsychology / Language	1.315 – 1.333
Genetics	1.334 – 1.342
Health Services	1.343 – 1.347
Case Studies	1.348 – 1.359
Saturday Late Breaking	1.360 – 1.377

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Saturday, Sunday and Monday at 12:15 p.m.
Convention Center, Hall A3, B3

Join leading experts as they spotlight interesting posters and facilitate discussion with authors, gaining new and different perspectives on the data presented.

To join a Walking Tour, gather at the poster information table behind booth #433 near poster board #001. A schedule of topics and tour leaders will be available.

POSTER SESSION 1: SATURDAY, DECEMBER 3
Tour Leaders: Steve Roper, M.D., Andres Kanner, M.D., Joseph Sirven, M.D., and Tallie Z. Baram, M.D., Ph.D.

POSTER SESSION 2: SUNDAY, DECEMBER 4
Tour Leaders: Jackie French, M.D., Ilo Leppik, M.D., Bruce Hermann, Ph.D., Andrew Cole, M.D., and Peter Crino, M.D.

POSTER SESSION 3: MONDAY, DECEMBER 5
Tour Leaders: Jean Gotman, Ph.D., Greg Bergey, M.D., Dennis Spencer, M.D., Anne Anderson, M.D., Kimford Meador, M.D., and Eric Kossoff, M.D.

Please note: Poster tour leaders are subject to change.

SUNDAY, DECEMBER 4

Poster Session Two

10:00 a.m. – 4:00 p.m.
Convention Center, Hall A3, B3

Authors Present: Noon – 2:00 p.m.
Poster Walking Tours: 12:15 p.m. – 1:45 p.m.

Neurophysiology	2.001 – 2.064
Clinical Epilepsy	2.065 – 2.119
Neuroimaging	2.120 – 2.154
Comorbidities (Somatic and Psychiatric)	2.155 – 2.178
Antiepileptic Drugs	2.179 – 2.230
Non-AED/Non-Surgical Treatments	2.231 – 2.239
Surgery	2.240 – 2.263
Behavior / Neuropsychology / Language	2.264 – 2.280
Genetics	2.281 – 2.290
Health Services	2.291 – 2.311
Neuropathology of Epilepsy	2.312 – 2.318
Practice Resources	2.319 – 2.323
Epidemiology	2.324 – 2.333
Public Health	2.334 – 2.346
Case Studies	2.347 – 2.360
Sunday Late Breaking	2.361 – 2.368
Sunday Camelice Posters	2.369 – 2.378

MONDAY, DECEMBER 5

Poster Session Three

8:00 a.m. – 2:00 p.m.
Convention Center, Hall A3, B3

Authors Present: Noon – 2:00 p.m.
Poster Walking Tours: 12:15 p.m. – 1:45 p.m.

Translational Research	3.001 – 3.089
Neurophysiology	3.090 – 3.147
Clinical Epilepsy	3.148 – 3.192
Neuroimaging	3.193 – 3.222
Antiepileptic Drugs	3.223 – 3.259
Surgery	3.260 – 3.284
Dietary	3.285 – 3.302
Behavior / Neuropsychology / Language	3.303 – 3.321
Genetics	3.322 – 3.331
Health Services	3.332 – 3.338
Epidemiology	3.339 – 3.348
Case Studies	3.349 – 3.360
Monday Late Breaking	3.361 – 3.378

Investigators Workshop Poster Session

Sunday, December 4 | Noon – 1:30 p.m. | *Convention Center, Ballroom Prefunction, between Rooms 310 and 320*
See pages 66-67

EXHIBIT HALL AND SCIENTIFIC EXHIBITS SCHEDULES

Exhibit Hall

SATURDAY, DECEMBER 3 | Noon - 6:00 p.m.

Convention Center, Hall A3, B3

Noon .....Grand Opening
Noon – 6:00 p.m. .....Cyber Café
Noon – 6:00 p.m. .....Attendee Lounge
Noon – 1:00 p.m. .....Lunch
2:30 p.m. - 3:30 p.m .....Beverage Break
4:30 p.m. - 5:30 p.m. .....Symposia Break
5:30 p.m. .....Passport to Prizes Drawing

SUNDAY, DECEMBER 4 | 10:00 a.m. - 4:00 p.m.

Convention Center, Hall A3, B3

10:00 a.m. – 4:00 p.m. .....Cyber Café
Noon – 6:00 p.m. .....Attendee Lounge
10:00 a.m. – 11:00 a.m. .....Beverage Break
Noon – 1:00 p.m. .....Lunch
3:00 p.m. - 4:00 p.m. .....Beverage Break
3:30 p.m. .....Passport to Prizes Drawing

MONDAY, DECEMBER 5 | 10:00 a.m. - 2:00 p.m.

Convention Center, Hall A3, B3

10:00 a.m. – 2:00 p.m. .....Cyber Café
Noon – 6:00 p.m. .....Attendee Lounge
10:00 a.m. – 11:00 a.m. .....Beverage Break
Noon – 1:00 p.m. .....Lunch
1:30 p.m. .....Passport to Prizes Drawing

#AES2016

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Scientific exhibits will be on display in Rooms 330 A, 340 A, 360 A and D, 370 A and D on Level 3 of the George R. Brown Convention Center. These exhibits will provide meeting attendees an opportunity to update themselves on the latest research. Authors will be present throughout the exhibit.

SUNDAY, DECEMBER 4 | 8:00 a.m. – 11:00 a.m.

Eisai Inc.
Research Updates from Eisai
Convention Center, Room 370 A

Lundbeck, LLC.
Research Updates on Clobazam, IV Carbamazepine, and Vigabatrin: Maintaining Lundbeck's Partnership in Epilepsy
Convention Center, Room 330 A

Sunovion Pharmaceuticals Inc.
Aptiom® (eslicarbazepine acetate) Scientific Exhibit Room
Convention Center, Room 360 A, D

SUNDAY, DECEMBER 4 | 8:00 a.m. - 5:00 p.m.

UCB, Inc.
UCB Connecting You to Solutions for Your Patients
Convention Center, Room 370 D

Zogenix, Inc.
Evolution of Low-Dose Fenfluramine in the Treatment of Epileptic Encephalopathies: New Understandings of Mechanisms, Basic Science and Clinical Data
Convention Center, Room 340 A

SUNDAY, DECEMBER 4 | 2:00 p.m. – 5:00 p.m.

Sage Therapeutics
Neuroactive steroids in the treatment of Status Epilepticus (SE), Super Refractory SE (SRSE) and Orphan Epilepsies
Convention Center, Room 360 A, D

MONDAY, DECEMBER 5 | 8:00 a.m. - 11:00 a.m.

GW Pharmaceuticals
Advancing Cannabidiol as a Treatment in Epilepsy: GW's Development Program and Initial GWPCARE Phase III Results
Convention Center, Room 330 A

The AES 2016 Annual Meeting

The AES Annual Meeting is the largest meeting and exhibition in the world for those who share the common scientific and clinical interests of epilepsy and clinical neurophysiology. The meeting attracts more than 5,000 attendees dedicated to improving the quality of life for those living with epilepsy. AES is one of 108 chapters of the International League Against Epilepsy(ILAE). In 2016 AES is proud to also host the 6th Biennial North American Regional Epilepsy Congress.

Learn at Sessions Tailored to Your Experience

The AES Annual Meeting offers high-quality educational programming designed for diverse work settings, professional roles and experience levels. Whether you are just starting with the specialty, have a limited background in epilepsy or are highly fluent with complex topics, you will find sessions and content relevant to your needs.

Benefit from a Comprehensive Program

From instructive lectures to hands-on learning, the AES Annual Meeting offers extensive learning opportunities.

Symposia — Provide the major educational activities at the Annual Meeting. Topics range from clinically oriented presentations reviewing common issues in epilepsy to more complex topics combining basic sciences and clinical neurology. While target audiences differ, all symposia include discussion of clinically relevant information.

Special Interest Groups (SIGs) — Offer education and networking for attendees with similar interests, in sessions organized by AES members. Although the sizes of SIG sessions vary, all lend themselves to active participation and dialogue.

General Lectures — Recognize the accomplishments of distinguished leaders in clinical epilepsy and research. The Hoyer Lecture is delivered by an AES President Emeritus. The Lennox and Lombroso Lecture is given by an invited member who has greatly advanced the collective understanding of epilepsy.

Annual Course — Encourage in-depth exploration of important topics related to epilepsy, focused on clinical care, including review of the science underlying the topics, reviews of clinical research and discussion of the associated clinical implications. The Annual Course includes a mixture of educational lectures, clinical vignettes and panel discussions.

Investigators Workshops — Highlight exciting developments in basic, translational and clinical epilepsy research in a format promoting interactive discussion. Speakers include established and junior epilepsy investigators, plus researchers from other fields.

Skills Workshops — Deliver hands-on and interactive learning opportunities in focused clinical areas or basic science research skills. Attendance at each workshop is limited to a small number of participants to allow optimal interaction. Advance registration and an additional fee are required.

Discover the Latest Research at Poster Sessions

Providing a forum for the latest research, poster sessions feature accepted abstracts, encouraging interaction between presenters and attendees. First authors will be present each day from noon – 2:00 p.m.

Find Valuable Resources in the Exhibit Hall

The Exhibit Hall is an integral part of the Annual Meeting experience, highlighting the latest in pharmaceuticals, publications, scientific equipment and technology relevant to the field of epilepsy.

To ensure safety and security, no children under 12 years of age, strollers, carriages, wheeled luggage or wheeled briefcases are allowed in the Exhibit Hall during exhibit hours.

Visit the Epilepsy Leadership Council Tables

A special feature of the AES Exhibit Hall is the Epilepsy Leadership Council (ELC) area. Network with non-profit organizations doing important work in advocacy, patient outreach, patient services and research funding.

Many ELC exhibitors will be offering mini-workshops and presentations in Booth 326 across from the AES Booth. A schedule will be posted near the entrance of that booth highlighting daily events.

Attend the Scientific Exhibits

AES has approved guidelines for industry-sponsored Scientific Exhibits at the Annual Meeting. Scientific Exhibits differ from traditional poster presentations in that a broad range of material can be presented as a collection of topics, such as results of various clinical trials or a thematic presentation of one aspect of drug development.

Network with Your Colleagues

Peer-to-peer networking is a longstanding tradition at the AES Annual Meeting. The meeting provides a unique forum for all professionals advancing research and patient care in epilepsy, a place to readily exchange ideas, practices and experiences. Attendees often cite interacting with other professionals as a key benefit of attending the Annual Meeting.

Connect at the Cyber Café

The Cyber Café, supported by Sunovion Pharmaceuticals Inc., is open during Exhibit Hall hours at the Convention Center. The Café provides email access, Internet connection and a printer for your convenience. Please note: complimentary Wi-Fi is also available in the concourses and public areas of the Convention Center.

EDUCATION CREDITS

Accreditation

The American Epilepsy Society is accredited by the Accreditation Council for Continuing Medical Education (ACCME) to provide continuing medical education for physicians.

AMA Credit Designation Statement

The American Epilepsy Society designates this live activity for a maximum of 29.50 *AMA PRA Category 1 Credits*™. Physicians should claim only the credit commensurate with the extent of their participation in the activity.

International Credits: The American Medical Association has determined that non-U.S. licensed physicians who participate in this CME activity are eligible for a maximum of 29.50 *AMA PRA Category 1 Credits*™.

Physician Assistants: AAPA accepts certificates of participation for educational activities certified for *AMA PRA Category 1 Credits*™ from organizations accredited by ACCME or a recognized state medical society. Physician assistants may receive a maximum of 29.50 hours of Category 1 credit for completing this program.

Continuing Education for Nurses and Pharmacists

Jointly provided by AKH, Inc., Advancing Knowledge in Healthcare, and the American Epilepsy Society.

Nurses: Advancing Knowledge in Healthcare is accredited as a provider of continuing nursing education by the American Nurses Credentialing Center's Commission on Accreditation. This activity is awarded 29.50 contact hours.

Pharmacists: Advancing Knowledge in Healthcare is accredited by the Accreditation Council for Pharmacy Education as a provider of continuing pharmacy education.

Select portions of this Annual Meeting are approved for pharmacy CE credit. Specific hours of credit for approved presentations and the Universal Activity Numbers assigned to those presentations are found elsewhere in the program materials. Criteria for success: credit is based on documented program attendance and online completion of a program evaluation/assessment.

If you have any questions about this CE activity relative to nursing and/or pharmacy CE, please contact AKH Inc at service@akhcme.com.

The American Board of Psychiatry and Neurology has reviewed the 70th Annual Meeting — American Epilepsy Society and has approved this program as part of a comprehensive epilpesy program, which is mandated by the ABMS as a necessary component of maintenance of certification.

Claiming CME Credit and CME Certificates

Attendees who registered in the following categories may claim CME or CE for the meeting: physician, health care provider, trainee, one-day and two-day. Meeting registration includes credit claiming: there is no separate fee to claim CME/CE.

Attendees will receive an emailed notification to access the online evaluation and credit claim system.

The evaluation and credit claim system will remain open through Tuesday, February 28, 2017. Evaluations and credit claims must be completed by this date in order to record and receive your CME/CE certificate.

Attendance Certificate/International Attendees

A meeting attendance certificate will be available at the registration desk for international meeting attendees on Tuesday, December 6.

Resolution of Conflicts of Interest

It is the policy of the American Epilepsy Society to ensure balance, independence, objectivity and scientific rigor. All persons involved in the selection, development and presentation of content are required to disclose any real or apparent conflicts of interest. In accordance with the ACCME Standards for Commercial Support of CME, AES implemented the mechanism of prospective peer review of this CME activity, to identify and resolve any conflicts. Additionally, the content of this activity is based on the best available evidence.

Unapproved Use Disclosure

AES requires CME authors to disclose to learners when products or procedures being discussed are off-label, unlabeled, experimental and/or investigational (not FDA approved); and any limitations on the information that is presented, such as data that are preliminary or that represent ongoing research, interim analyses and/or unsupported opinion. This information is intended solely for continuing medical education and is not intended to promote off-label use of these medications. If you have questions, contact the medical affairs department of the manufacturer for the most recent prescribing information. Information about pharmaceutical agents/devices that is outside of U.S. Food and Drug Administration approved labeling may be contained in this activity.

Disclaimer

This CME activity is for educational purposes only and does not constitute the opinion or endorsement of, or promotion by, the American Epilepsy Society. Reasonable efforts have been taken to present educational subject matter in a balanced, unbiased fashion and in compliance with regulatory requirements. However, each activity participant must always use his or her own personal and professional judgment when considering further application of this information, particularly as it may relate to patient diagnostic or treatment decisions including, without limitation, FDA-approved uses and any off-label, investigational and/or experimental uses.

The AES Annual Meeting is convening at:
George R. Brown Convention Center
1001 Avenida de las Americas
Houston, Texas 77010

Language
The official language of the Annual Meeting is English.

Meeting Attire
Dress for the Annual Meeting is business casual. Consider bringing a light jacket or sweater with you since meeting room temperatures and personal comfort levels vary.

No Smoking Policy
For the comfort and health of all attendees, smoking is not permitted at any AES function. This includes educational sessions, meetings and all food functions. The George R. Brown Convention Center and all the official meeting hotels are smoke-free facilities. Please note: smoking is also not permitted in public buildings, restaurants or bars.

Symposia Handouts
Handout materials for the educational symposia will be available via the Annual Meeting App.

Audience Response System
AES will use an Audience Response System (ARS) in several of the symposia. Faculty will have ARS questions throughout their presentations with multiple choice answers. To participate, you will use your mobile phone to text your answer. When a question appears in a presentation, simply text your answer (a 5 or 6-digit code) to "22333". Standard text rates will apply. The ARS allows the learner to participate in real time. Responding to these questions enhances the learning environment and provides feedback to the speaker to assure symposium learning objectives are met.

Program Changes
AES cannot assume liability for any changes in the program due to external or unforeseen circumstances.

Camera, Mobile Phone, and Video Recording Policies
AES strictly prohibits all photography (flash, digital, or otherwise), audio- and/or video- recording during all educational sessions at the Annual Meeting. **Violation of this policy will result in removal from the session and equipment will be confiscated.**

Material presented at the AES Annual Meeting may not be reproduced in any format without the express written consent of AES. Attendees acknowledge and agree that commercial or promotional distribution, publishing or exploitation of speaker sessions, content or materials from the AES Annual Meeting is strictly prohibited unless you have received the express prior written permission from AES or the otherwise applicable rights holder.

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AES uses photographs of meeting events in its promotional materials. Unless the permission is revoked in writing to AES, by virtue of their attendance, all meeting visitors agree to use of their likeness in such materials.

Insurance/Liabilities
AES cannot be held responsible for any personal injury, loss, damage, accident to private property or additional expenses incurred as a result of delays or changes in air, rail, sea, road or other services, strikes, sickness, weather, acts of terrorism and any other cause. All participants are encouraged to make their own arrangements for health and travel insurance.

Gratuities
Gratuities are not automatically added to the bill, except in some cases for large groups. Waiters and waitresses are usually given 15% to 20% of the bill. Taxi drivers typically receive 15% of the fare, doorman $2 per service and $2 per night for hotel housekeeping. Skycaps and porters are normally tipped $1 per bag.

Mother's Room
Convention Center, Room 380 A

Nursing mothers may utilize this private room at the Convention Center available during session times. Please note that parents or guardians are responsible to provide infant care supplies.

Quiet Room
Convention Center, Room 380 C

New in 2016 — A Quiet Room is available in the George R. Brown Convention Center, Room 380 C. The room is intended to provide a quiet, calm space where attendees can spend time away from noise, lights and other stimuli. The quiet room is not available for conversations or meetings.

Lost and Found
Convention Center, General Assembly Foyer

Please visit the Registration Desk to look for items lost or to turn in items found. The American Epilepsy Society is not responsible for missing items. Please do not leave unattended packages (i.e. briefcases, meeting totes, laptops, purses, etc.) in any area of the Convention Center or hotel. Rooms are cleaned between sessions and any items left behind will be discarded.

First Aid
Convention Center, Across from Room 342 A
Enter via hallway between rooms 340 A and 350 A.

Scooter/Wheelchair Rental
Scooter and wheelchair rentals are available by contacting Scootaround at 888-341-7575 or scootaround.com.

You must call to place an order and arrange delivery of equipment to the George R. Brown Convention Center.

ATM
There is one permanent automated teller machine (ATM) located near Starbucks on Level 2 of the Convention Center.

Faculty Ready Room

Convention Center, Room 322 A
Enter via the hallway in between rooms 320 A and 330 A, across from the registration area.

All faculty PowerPoint presentations have already been uploaded through the AES Faculty Management System. All faculty must stop by to reconfirm their presentation with an audiovisual technician.

Thursday, December 1 ..... 4:00 p.m. – 8:00 p.m.
Friday, December 2 ..... 8:00 a.m. – 6:00 p.m.
Saturday, December 3 ..... 8:00 a.m. – 6:00 p.m.
Sunday, December 4 ..... 8:00 a.m. – 6:00 p.m.
Monday, December 5 ..... 8:00 a.m. – 6:00 p.m.
Tuesday, December 6 ..... 8:00 a.m. – 11:00 a.m.

Press Room

Convention Center, Room 352 A
Enter via the hallway between rooms 340 A and 350 A.

AES hosts a press room to assist journalists to find resources and identify experts at the Annual Meeting. Exhibitors may leave press releases on designated tables in the press room.

Friday, December 2 ..... 11:00 a.m. - 6:30 p.m.
Saturday, December 3 ..... 7:30 a.m. - 6:30 p.m.
Sunday, December 4 ..... 7:30 a.m. - 6:30 p.m.
Monday, December 5 ..... 7:30 a.m. - 6:30 p.m.
Tuesday, December 6 ..... 7:30 a.m. - 1:00 p.m.

For more information about press at the Annual Meeting, please visit the AES website at **aesnet.org/annual_meeting/press**

Business Centers

Two full-service FedEx Office business centers are conveniently located for Annual Meeting attendees. Both offer a variety of services that include shipping, mailing, faxing and photocopying. Please contact the office directly for details.

FedEx Office
Level 2, above Hall D and next to Explore Houston Gift Shop
1001 Avenida de las Americas, Houston, TX 77010
Phone: 713. 658.1899 | Fax: 713.658.9617
Email: usa5000@fedex.com

FedEx Office
Located on Level 3 near the Skybridge to the Convention Center
1600 Lamar, Houston, TX 77010
Phone: 713.651.3013
Email: usa5721@fedex.com

Safety and Security Information

The following security measures have been designed to further enhance your personal and professional safety.

- Pick up any Convention Center house phone in the facility and dial the Security Command Station at extension 8087. Or call 713.853.8087 from a cell phone
- Uniformed Convention Center employees have radios and are ready to assist you. Advise the dispatcher of the exact location within the Convention Center. We respectfully request that you do NOT call 911 directly
- An EMT will be on duty in the Convention Center throughout the meeting
- A government-issued photo identification is required to receive a badge and to replace a lost badge
- Convention Center Security may randomly check packages and bags at the Convention Center entrances, meeting rooms and in the Exhibit Hall
- You will be asked to always clearly display your name badge and to use only approved Convention Center entrances and exits
- Appropriate badges will be required to enter all educational sessions, Poster Sessions, Exhibit Hall and meetings. Due to safety and fire regulations, doors will be closed to session rooms that fill to capacity
- Throughout the meeting, you will notice security staff presence to monitor the safety of all participants
- Do not leave unattended packages (i.e., briefcases, laptops, purses, etc.) in any area of the Convention Center or hotel
- Please report any suspicious activity to security staff or to the AES registration desk staff

General Safety Tips

- Take all items with you when leaving a session. Rooms are cleaned and items left behind are discarded
- Remove your badge once you leave the meeting facilities
- Carry important telephone numbers with you
- Do not display or carry large amounts of cash
- Walk in groups, especially at night
- Lock your hotel room door
- Always verify hotel room repair or service calls
- Do not disclose your room number to anyone
- Never give your personal information (credit card, room number, etc.) over the phone; instead, go to the front desk if the hotel calls with questions

Experience AES

To eradicate epilepsy and its consequences — that's the vision of the American Epilepsy Society. AES provides continuing education resources for members, is a champion of research, and serves as home to the brightest minds in the field of epilepsy.

Find out more about AES Membership:

Exhibit Hall Booth #327

During Exhibit Hall Hours

Online AESnet.org

Click on Membership Tab

American Epilepsy Society

AES member benefits include:

- *Epilepsy Currents*, a commentary and literature review journal from AES
- *Epilepsia*, research journal of the International League Against Epilepsy*
- Substantial discounts to meetings and programs, including the AES Annual Meeting
- Listing in Find-A-Doctor online directory*
- AES Connections email newsletter
- Research funding opportunities
- Scholarships and awards
- AES Connect, our online, members-only community for networking and exchange

**Benefit of select member categories*

AES Exhibit Booth

Open during Exhibit Hall hours

Booth #327
Convention Center - Exhibit Hall A3, B3

All Annual Meeting attendees are encouraged to visit the AES Booth #327. Come by to:

- Get assistance with the AES Annual Meeting App
- Take your official Annual Meeting selfie
- Show your professional connection with the purchase of an AES necktie, bowtie or scarf
- Learn more about joining our dynamic community 4,000 members strong—and counting
- Take a breather to catch up with colleagues: loitering is encouraged

5th Annual Wine Tasting and Silent Auction

Saturday, December 3
7:00 p.m. - 10:00 p.m.

Massa's South Coast Grill
1331 Lamar Street, Suite 114
Houston, TX 77010

The popular AES event returns with a Texas twist. Sample extraordinary wines from around the world paired with innovative hors d'oeuvres. All proceeds benefit the Lennox and Lombroso and Susan S. Spencer AES research funds.

New in 2016: Purchase tickets online through the registration system, while supplies last. Space is limited to 200 people. $175 per ticket

Questions? Please visit the registration desk at the Convention Center for up-to-date ticket availability.

Show You Care *Twice*

Give the Community of Caring Pin to someone special — a colleague, collaborator, mentor or friend. *The second benefit:* all proceeds support AES research and education programs.

Community of Caring Pins are available for purchase at the AES Booth #327.

AMERICAN EPILEPSY SOCIETY

Dedicated to eradicating epilepsy and its consequences.

The American Epilepsy Society is a medical and scientific society whose members are engaged in research and clinical care for people with epilepsy. For more than 75 years, AES has provided a dynamic global forum where professionals from academia, private practice, not-for-profit, government and industry can learn, share and grow.

AMERICAN EPILEPSY SOCIETY

AESnet.org

Executive Office
135 South LaSalle Street
Suite 2850
Chicago, IL 60603
Tel: 312-883-3800

#AES2016

Fresh ideas. New connections. Shared conversations.

Find them all at the Informal Learning Center.

Exhibit Booth #326

New for 2016! The Informal Learning Center is the place for active learning and impromptu conversations — with experts and colleagues alike!
Visit Booth #326 in the Exhibit Hall to hear industry speakers, interact with patient advocacy organizations, and participate in the new Speed Networking events.

SATURDAY, DECEMBER 3

12:30 p.m.	Citizens United for Research in Epilepsy (CURE): *New Sleep and Epilepsy Research Grant*
1:00 p.m.	"Campfire" Discussion: Sunovion Pharmaceuticals Inc.
1:30 p.m.	Presentation: My Epilepsy Story
2:00 p.m.	Presentation: Dup15q Alliance
2:30 p.m.	Hope for Hypothalamic Hamartoma: *Research Update — Highlights from 3rd International HH Symposium*
3:00 p.m.	Speed Networking, Supported by Eisai Inc. *This event requires advance sign-up. Visit the registration desk to see if spots are still available.*
4:30 p.m.	Presentation: Tuberous Sclerosis Alliance
5:00 p.m.	Presentation: Child Neurology Foundation

Informal Learning Center
Open during Exhibit Hall hours

Exhibit Hall, Convention Center, Hall A3, B3, Booth #326

An updated schedule will be posted daily at the Informal Learning Center. These events do not include CME credits.

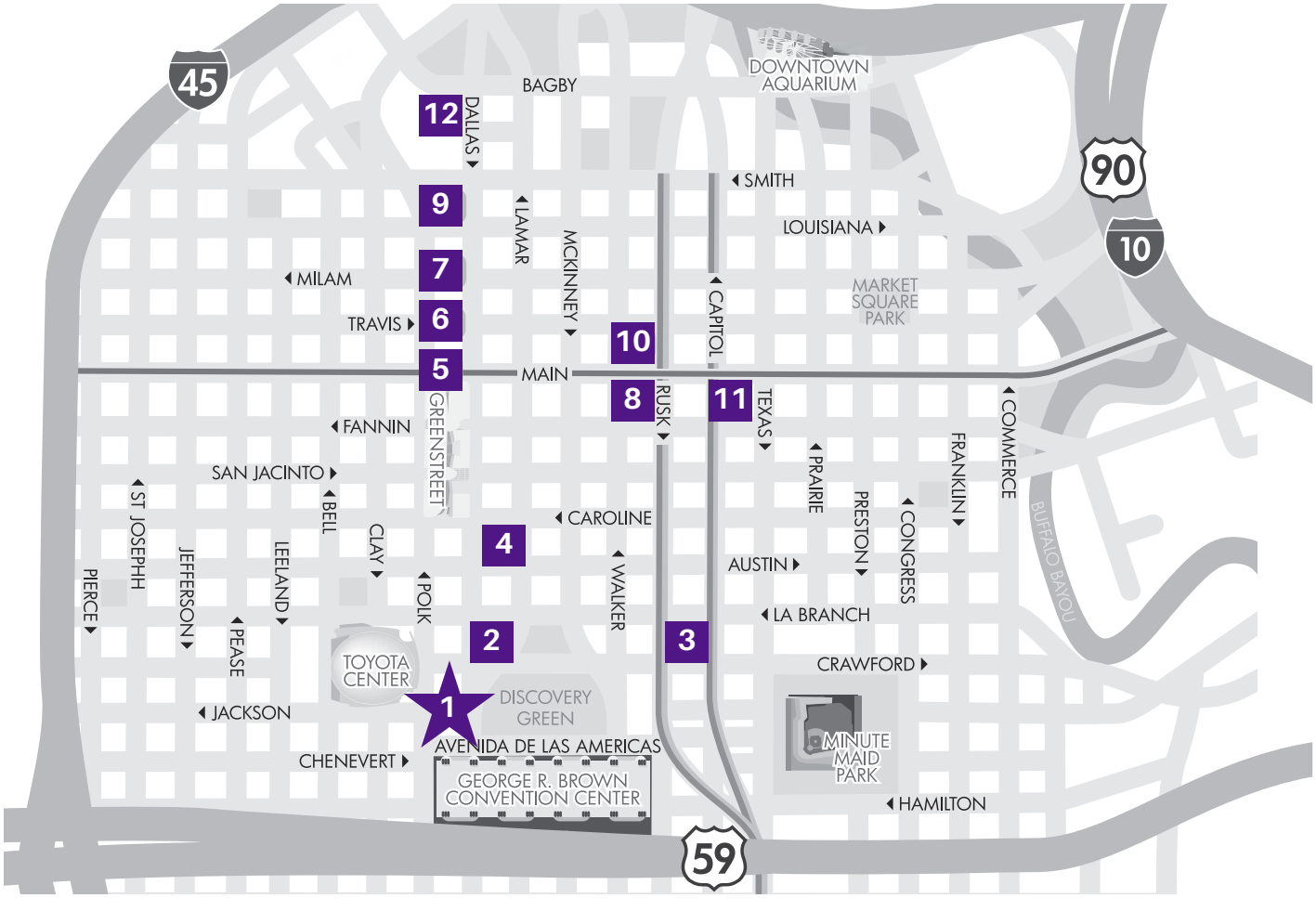
SUNDAY, DECEMBER 4

10:00 a.m.	Epilepsy Foundation: *Driving Innovation in Epilepsy Research*
10:30 a.m.	Veterans Health Administration (VHA) — Epilepsy Centers of Excellence (ECoE): *Epilepsy Basic Training*
11:00 a.m.	Speed Networking, Supported by Eisai Inc. *This event requires advance sign-up. Visit the registration desk to see if spots are still available.*
Noon	CDKL5: *What We Know and Where We Are Going*
12:30 p.m.	Citizens United for Research in Epilepsy (CURE): *Epilepsy Genetics Initiative — We're Making Great Progress!*
1:00 p.m.	"Campfire" Discussion: Sunovion Pharmaceuticals Inc.
1:30 p.m.	National Institute of Neurological Disorders and Stroke (NINDS): *Epilepsy Therapy Screening Program (ETSP)*
2:00 p.m.	Speed Networking, Supported by Eisai Inc. *This event requires advance sign-up. Visit the registration desk to see if spots are still available.*
3:00 p.m.	Presentation: Hope4Harper
3:30 p.m.	Epilepsy Foundation: *Patients as Partners in Research*

MONDAY, DECEMBER 5

11:00 a.m.	"Campfire" Discussion: Sunovion Pharmaceuticals Inc.

AREA MAP OF HOUSTON AND HOTELS



1 **Hilton Americas Houston - AES Headquarters Hotel**
1600 Lamar Street, Houston, Texas 77010
713-739-8000

2 **Embassy Suites Houston - Downtown**
1515 Dallas Street, Houston, Texas 77010
713-739-9100

3 **Hampton Inn / Homewood Suites Houston**
710 Crawford Street, Houston, Texas 77002
Hampton Inn: 713-224-0011
Homewood Suites: 713-224-0710

4 **Four Seasons Hotel Houston**
1300 Lamar Street, Houston, Texas 77010
713-650-1300

5 **Courtyard Houston Downtown**
916 Dallas Street, Houston, Texas 77002
832-366-1600

6 **Springhill Suites Houston Downtown**
914 Dallas Street, Houston, Texas 77002
713-655-0002

7 **Residence Inn Houston Downtown**
904 Dallas Street, Houston, Texas 77002
832-366-1000

8 **Aloft Houston Downtown**
820 Fannin Street, Houston, Texas 77002
713-225-0200

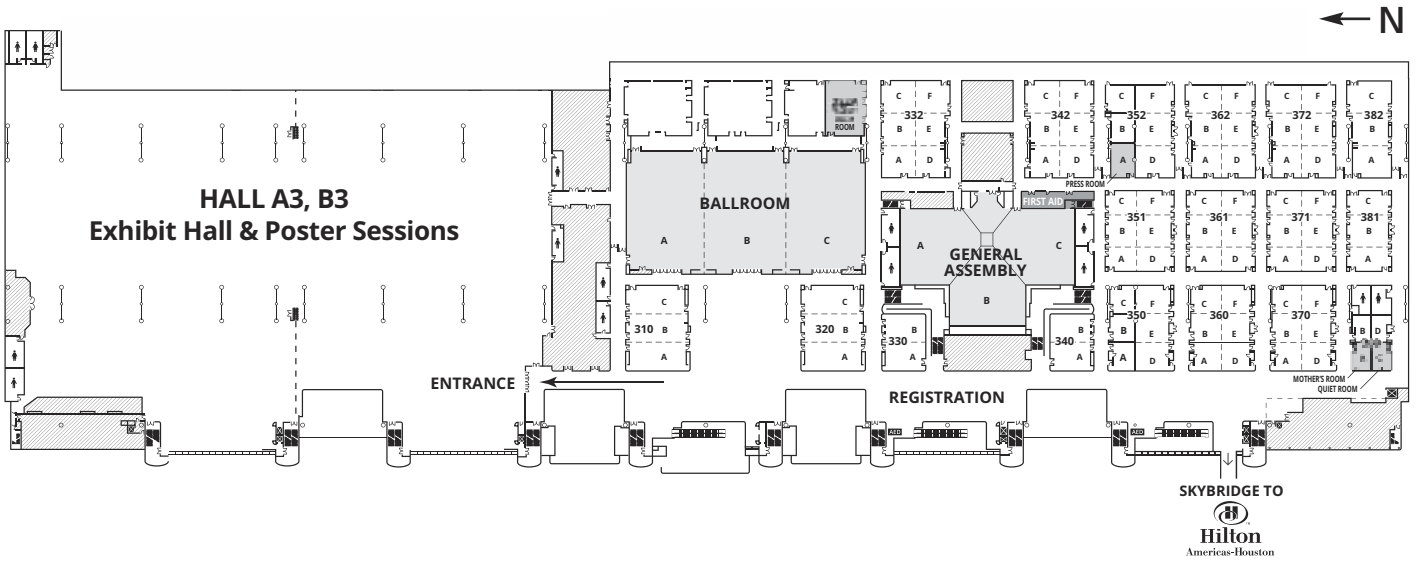
9 **Hyatt Regency Houston**
1200 Louisiana Street, Houston, Texas 77002
713-654-1234

10 **JW Marriott Houston Downtown**
806 Main Street, Houston, Texas 77002
713-237-1111

11 **Magnolia Hotel Houston**
1100 Texas Avenue, Houston, Texas 77002
713-221-0011

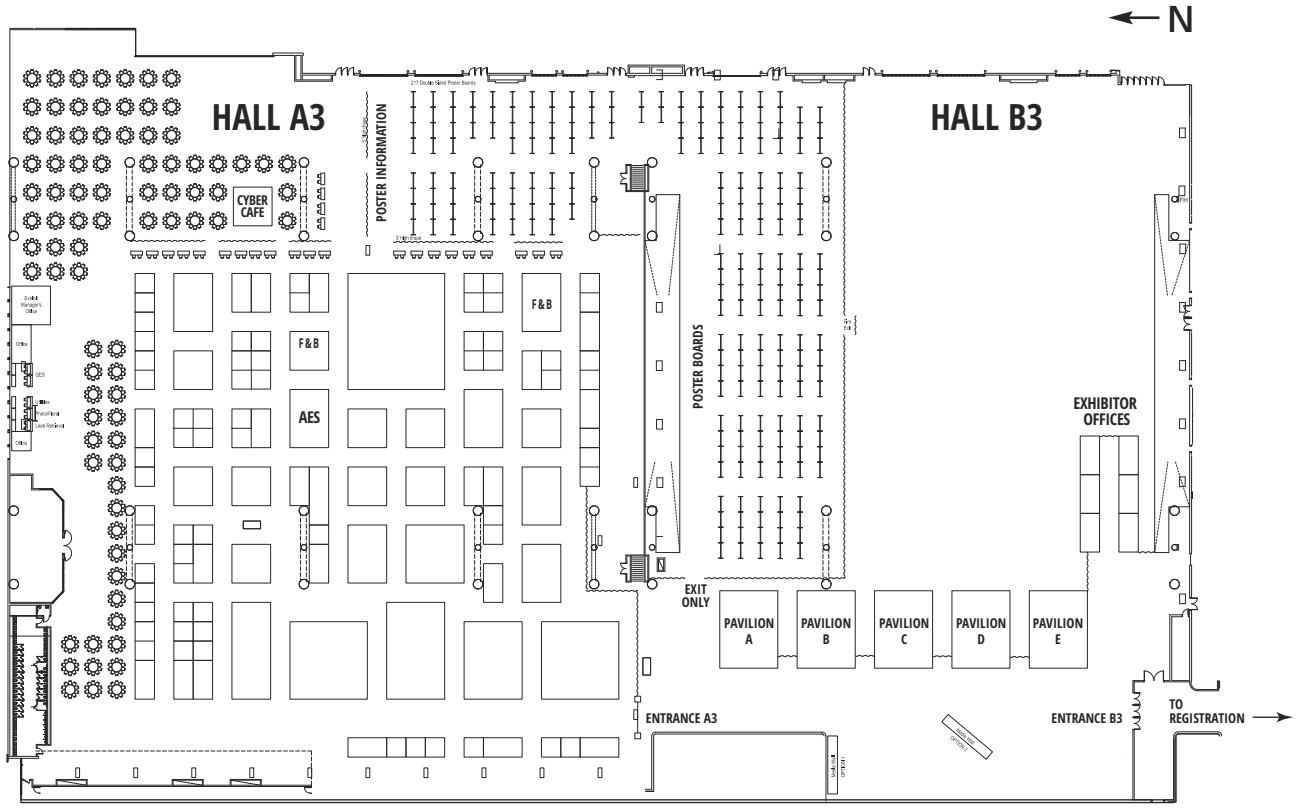
12 **DoubleTree by Hilton Hotel Houston Downtown**
400 Dallas Street, Houston, Texas 77002
713-759-0202

Third Floor Convention Center
Full View



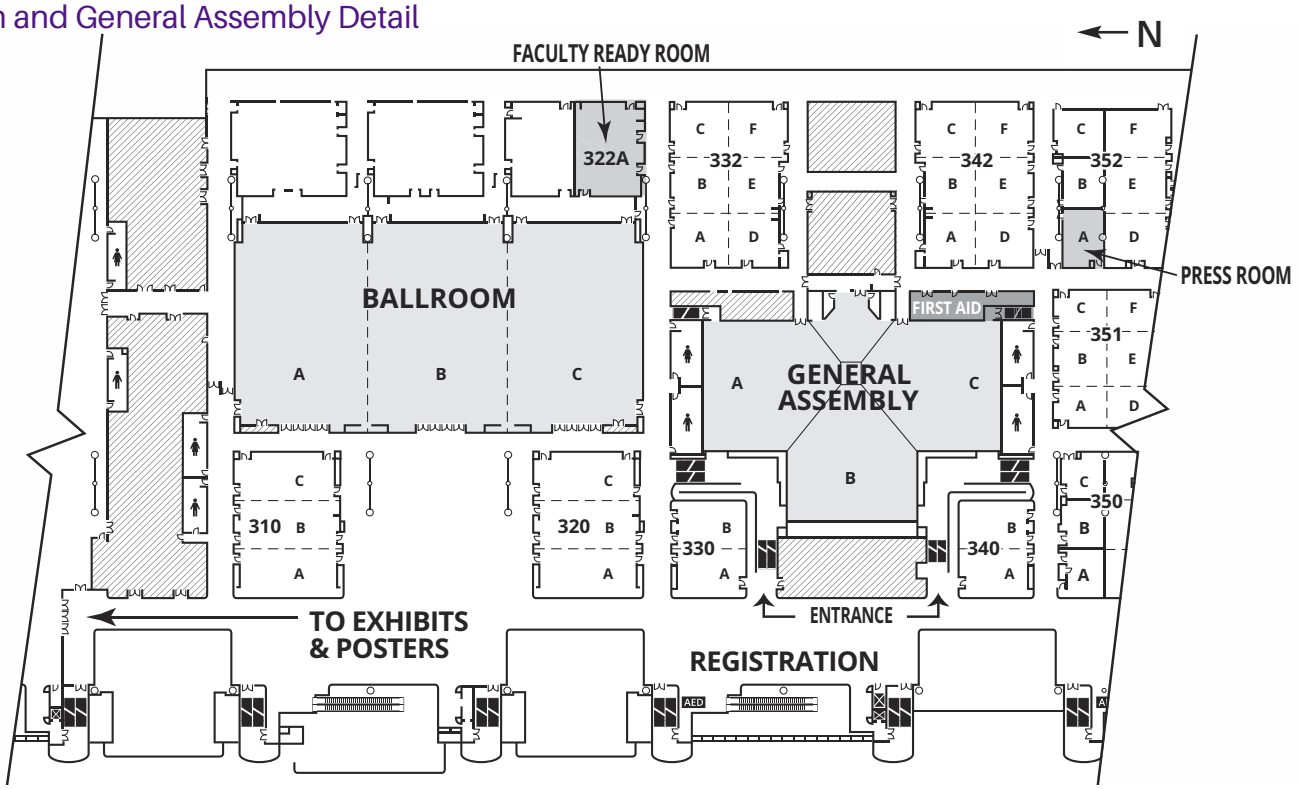
Third Floor Convention Center
Exhibit Hall A3, B3 Detail

See pages 120-121 for locations and map.

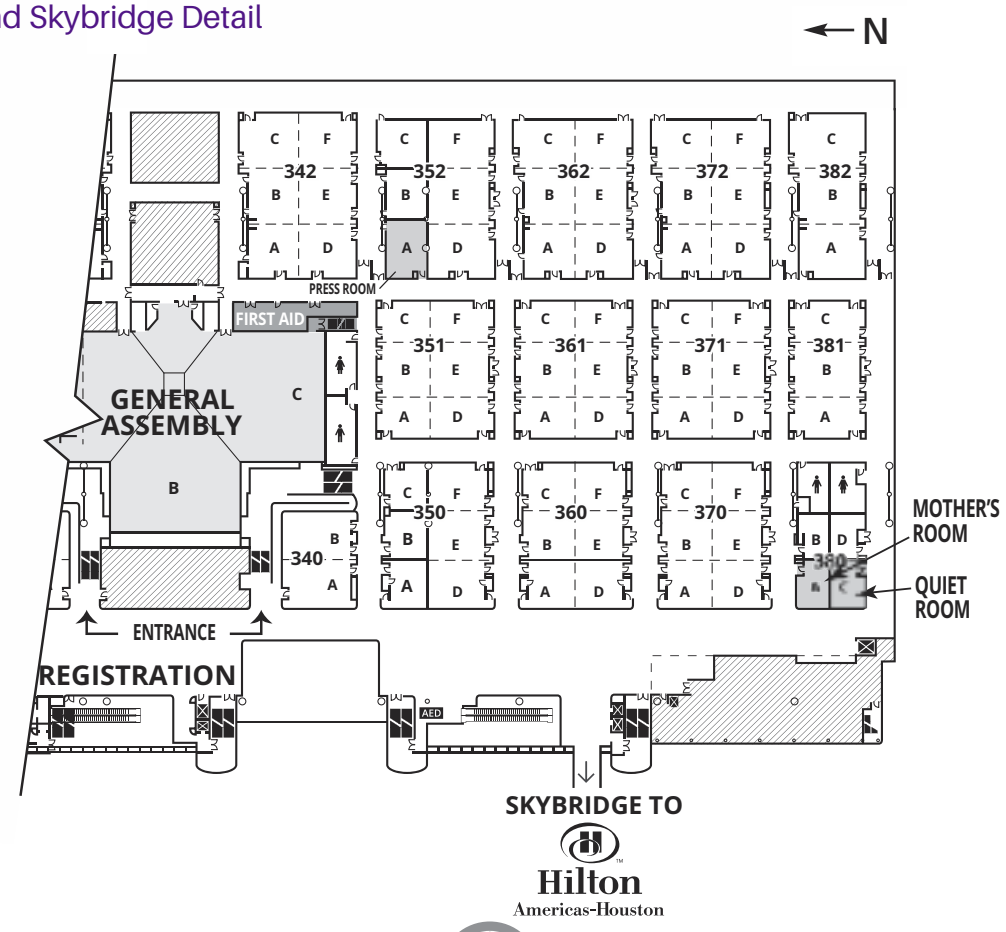


GEORGE R. BROWN CONVENTION CENTER

Third Floor Convention Center
Ballroom and General Assembly Detail

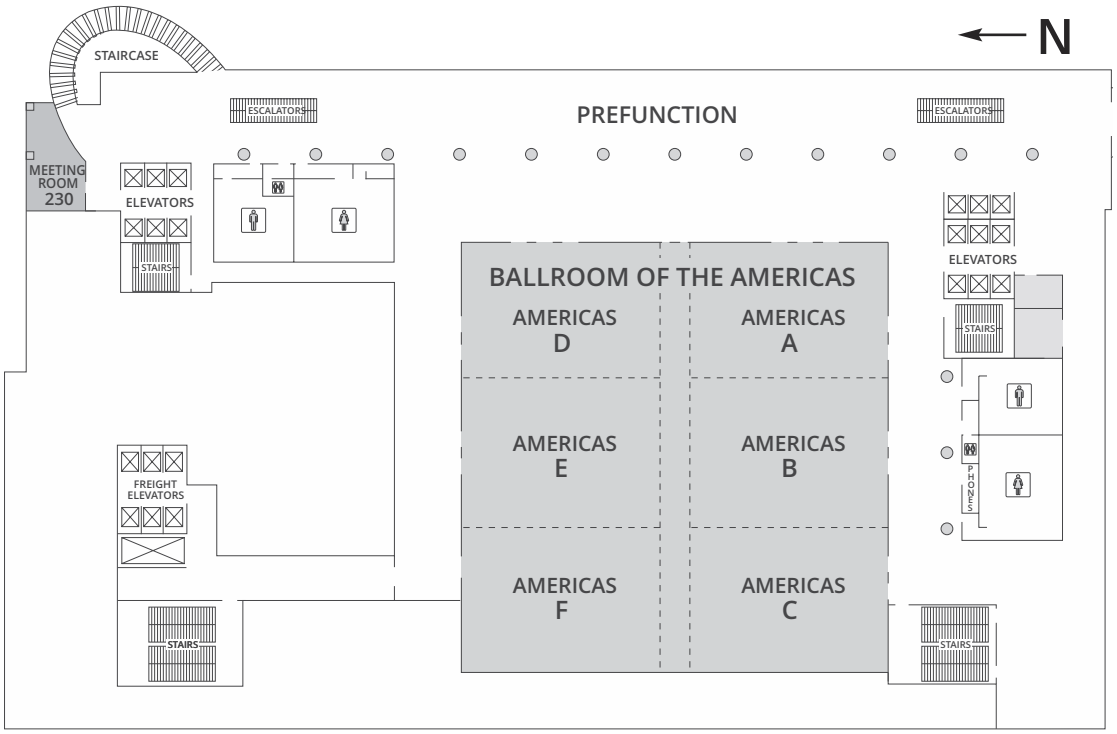


Third Floor Convention Center
Session Rooms and Skybridge Detail



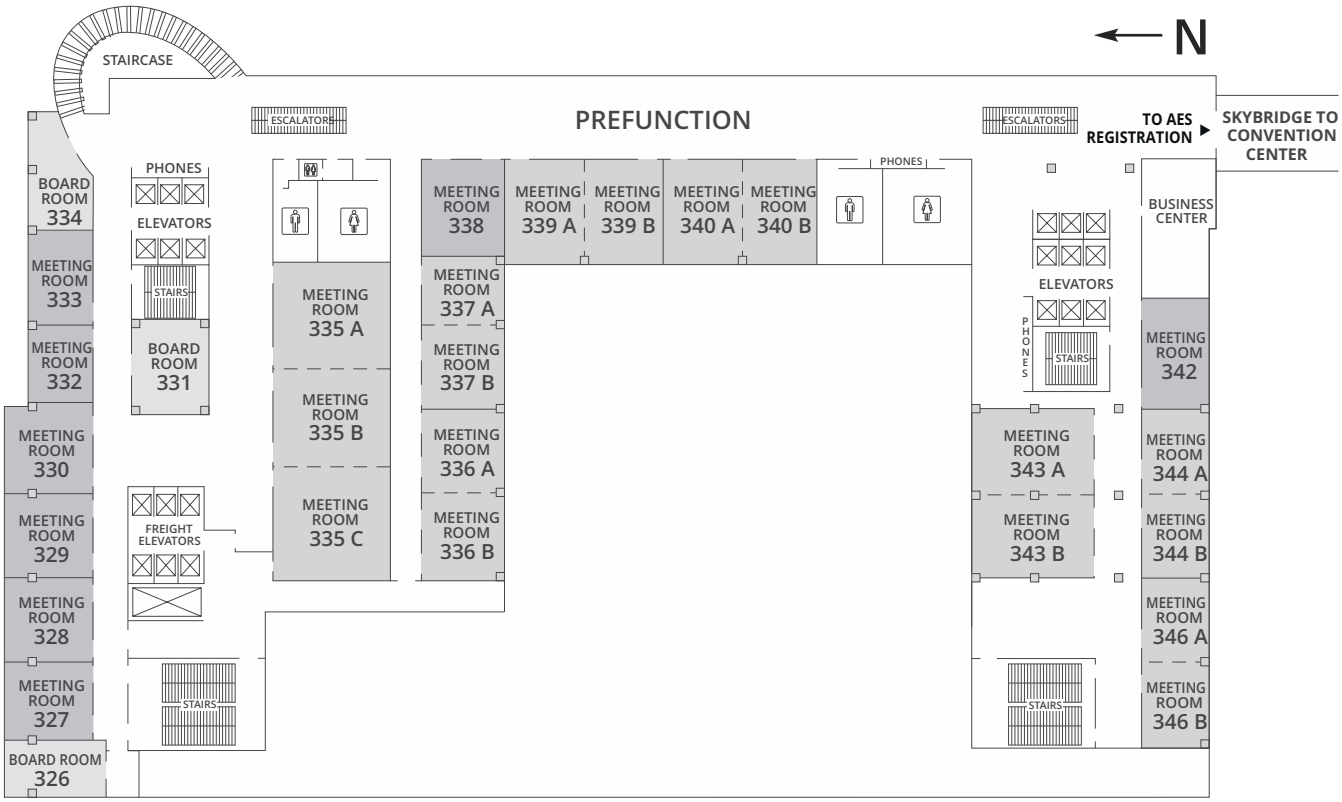
HILTON AMERICAS HOUSTON

Level Two

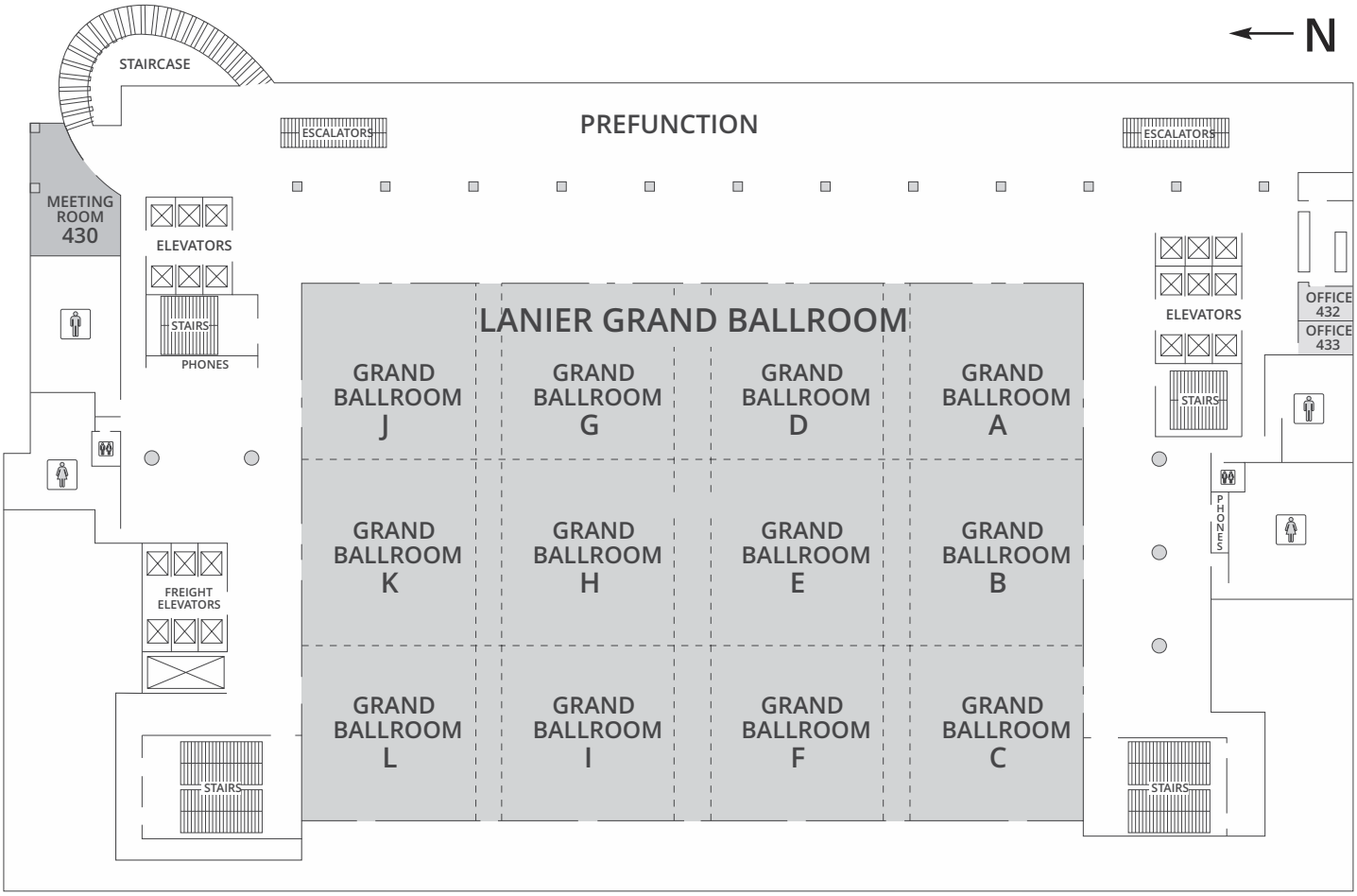


Level Three

Skybridge access to Convention Center



Level Four



Note: SkyBridge to Convention Center is on Level Three.

RESEARCH RECOGNITION AWARDS

The American Epilepsy Society Research Recognition Awards are given annually to active scientists and clinicians working in all aspects of epilepsy research. The awards recognize professional excellence reflected in a distinguished history of research of important promise for the improved understanding and treatment of epilepsy. These awards each include a $10,000 honorarium.

AWARD FOR BASIC SCIENCE

Saturday, December 3, 8:30 a.m.
Preceding the Presidential Symposium
Convention Center, General Assembly

Scott C. Baraban, Ph.D.

Dr. Scott C. Baraban received his bachelor's degree from Johns Hopkins University and doctorate from the University of Virginia. He conducted postdoctoral research with Phil Schwartzkroin at the University of Washington, and joined the faculty at University of California, San Francisco, in 1999. He was promoted to Professor and William K. Bowes Jr. Endowed Chair in Neuroscience in 2008.

His research has focused on translational questions in epilepsy, and currently includes work on models of pediatric epilepsy in zebrafish, drug discovery and interneuron-based cell therapies. The first zebrafish models for epilepsy were developed in his laboratory, and his group recently published the first high-throughput drug screens using a zebrafish model for Dravet syndrome. The initial demonstration that embryonic GABA progenitor cells transplanted into postnatal brain can functionally integrate and enhance inhibition emerged from his lab, and led to the first proof-of-principle cell therapy studies in epileptic mice. His research has also contributed to our understanding of epileptic networks in a malformed brain, NPY as an endogenous anticonvulsant peptide, and the therapeutic potential of diuretics.

He has authored more than 100 publications including papers in *Journal of Neuroscience, Proceedings of the National Academy of Sciences of the USA, Neuron, Nature Communications, Science,* and *Nature Neuroscience*. His research is funded by federal and private sources, and he has received several research honors, including the Basil O' Connor Scholar Research Award, a Klingenstein Foundation Fellowship in Neuroscience and a National Institutes of Health(NIH) EUREKA award. He was recently awarded a Javits Neuroscience Investigator Award from the NIH. He has served on various editorial, scientific advisory and grant review boards, and co-chaired a successful Gordon Research Conference on epilepsy with Jack Parent. He also served as AES Scientific Program Chair and several AES committees, and currently serves on the editorial board of *Experimental Neurology*, NIH Clinical Neuroplasticity and Neurotransmitter Study Section, and Scientific Advisory Board of the Dravet Syndrome Foundation.

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Convention Center, General Assembly

Graeme D. Jackson, B.Sc (Hons)., MB.BS., MD., FRACP

Dr. Graeme D. Jackson is a Professor and Neurologist at the Austin Hospital, Melbourne, Australia, and a clinician researcher in advanced brain imaging and epilepsy. His research is based on using innovations in MRI technology to understand the structure and function of the brain, and applying that to the understanding of epilepsy. His focus is on translating research insights to the treatment of patients.

He is active in supporting research excellence in his and related fields. He is deputy director of The Florey Institute of Neuroscience and Mental Health, Professorial Fellow of the University of Melbourne and Clinical Research Fellow of the National Health and Medical Research Council of Australia (NHMRC). He has personally received the NHMRC excellence award for highest ranked clinical fellow and, with other chief investigators, the NHMRC excellence award for highest ranked research program. He chairs the Science Council of Neuroscience Victoria and the Academic Board of the Institute for Social Neuroscience. He has served a term as associate editor of *Epilepsia* (Imaging) and is currently on the Diagnostic Methods commission of the International League Against Epilepsy.

Research highlights include the MRI definition of hippocampal sclerosis with criteria still in widespread use today (Neurology 1990, AJNR 1993); development of functional imaging including the first use of a clinical MRI system for fMRI (Radiology 1993), functional imaging of seizures (Neurology 1994) and classification of Malformations of Cortical Development (Barkovich 1996-2012, Palmini 2004). Recently; super resolution imaging (Calamente 2010) brain networks in absence epilepsy (Masterton 2013) and epileptic encephalopathy (Pilay 2013). He was a Consultant Paediatric Neurologist in London, applying MRI to childhood epilepsy and brain development.

Qualifications include: B.Sc. (Honours, Psychology); MB.BS. and M.D. (Monash University); FRACP (Fellow Royal Australasian College of Physicians). He has published more than 250 peer reviewed primary data papers with more than 44 citations per publication (ISI).

LENNOX AND LOMBROSO LECTURER

The Lennox and Lombroso Lecture is given each year by a clinician or scientist who is considered to be an outstanding investigator in the field of epilepsy research. The Lennox and Lombroso Lecturer is selected in collaboration with the AES President, Annual Meeting Chair, and Scientific Program Committee Chair. This year's Lennox and Lombroso Lecturer will mark the 50th lecture in this series.

Monday, December 5, 2:15 p.m. – 3:00 p.m.
Lennox and Lombroso Lecture: EEG, The New Frontier
Convention Center, General Assembly

Jean Gotman, Ph.D.

Dr. Jean Gotman obtained a degree in Electrical Engineering from the University of Paris, a master's degree in Computer Science from Dartmouth College, Hanover, N.H., and a Ph.D. in Neuroscience from McGill University, Montreal. He joined the faculty of the Montreal Neurological Institute of McGill in 1977 and became full professor in 1993.

He pioneered the automatic detection of spikes and seizures in the EEG and made his methods widely available through Stellate, a company he created in 1986, which developed and sold all over the world equipment and software for EEG, long-term epilepsy monitoring and polysomnography.

He has published 270 peer-reviewed papers and 40 book chapters. His research interests include analysis of the EEG, mechanisms of epileptogenesis, seizure generation and seizure spread in humans, High Frequency Oscillations (HFOs) and functional imaging in the diagnosis and study of epilepsy.

He received the Research Recognition Award from the American Epilepsy Society, the Pierre Gloor Award of the American Clinical Neurophysiology Society, the Penfield Award of the Canadian League Against Epilepsy, and was named Ambassador for Epilepsy by the International League Against Epilepsy.

ABOUT AES RESEARCH FUNDS

The American Epilepsy Society is a leader in educating epilepsy professionals, providing support and assistance in career development, and mentoring the next generation of epilepsy researchers. Part of fulfilling the AES mission is a strong commitment to fund basic and clinical projects that identify and deliver answers to treating and curing epilepsy.

AES has established several funds targeting specific research and programmatic needs in epilepsy research, including these opportunities:

- Susan S. Spencer Clinical Education and Research Fund
- Jack M. Pellock Pediatric Travel Fund — *All gifts matched up to $22,500!*
- Suzanne and Peter Berry International Travel Award
- Fritz Dreifuss Epilepsy Fund
- Rebecca Goldberg-Kaufman Ethical Neuropsychiatry Award Fund
- Lennox and Lombroso Trust for Research and Training
- J. Kiffin Penry Fund

To make a gift supporting research, visit the AES website at: **AESnet.org**
Click *About AES,* then *Contribute*.

AMERICAN EPILEPSY SOCIETY

DISTINGUISHED SERVICE AWARD

Established in 1993, this award recognizes outstanding service by an AES member in the field of epilepsy (including non-educational and non-scientific) with emphasis on exemplary contributions to advancing the mission of the American Epilepsy Society and service to its members. The award includes a $1,000 honorarium.

Friday, December 2, 4:00 p.m.
Preceding the Judith Hoyer Lecture
Convention Center, General Assembly

Manisha N. Patel, Ph.D.

Dr. Manisha Patel received her Ph.D. in Pharmacology and Toxicology at Purdue University and post-doctoral training in Neuroscience at Duke University. She is currently a tenured Professor in the Department of Pharmaceutical Sciences at the University of Colorado Anschutz Medical Campus.

The overarching theme of her laboratory's research is to understand the metabolic basis of epilepsy and develop metabolism-based therapeutic strategies for the epilepsies and their comorbidities. She has authored more than 80 scientific publications including articles in the journals *Neuron, Journal of Neuroscience, Journal of Biological Chemistry,* and *Proceedings of the National Academy of Sciences of the USA*. Dr. Patel serves on the editorial boards of *Epilepsia Open, Free Radical in Biology and Medicine,* and *Redox Biology*. She has been the recipient of numerous grants from the National Institutes of Health, Citizens United for Research in Epilepsy and the Epilepsy Foundation.

Dr. Patel's service to AES has been devoted to supporting the careers of early stage investigators. She began her leadership role in AES as the chair of the Junior Investigator Special Interest Group and Funding Success Taskforce. The work of the Funding Success Taskforce was critical in providing outcome data of trainees for the Development Committee. She has also served AES as chair of the Basic Science Committee, Scientific Program Committee and Research and Training Council. As chair of the Research and Training Council, she led efforts in transitioning to an autonomous early career research program while fostering partnerships with other organizations.

FRITZ E. DREIFUSS LECTURE (Inaugural)

The Fritz E. Dreifuss lecture honors the memory of Dr. Dreifuss, a leading clinical epilepsy specialist, clinical investigator and former President of the American Epilepsy Society and the International League Against Epilepsy. Founder of the Comprehensive Epilepsy Program at the University of Virginia, and mentor to a generation of epilepsy researchers, Dr. Dreifuss was devoted to mentoring those in clinical epilepsy research. Supported by the AES Fritz E. Dreifuss Fund, the lecture promotes clinical epilepsy research and outstanding patient care and includes a $1,000 honorarium.

Saturday, December 3, 8:30 a.m.
During the Presidential Symposium
Convention Center, General Assembly

Peter B. Crino, M.D., Ph.D.

Dr. Peter Crino received his M.D. degree from Yale University (Alpha Omega Alpha Honor Medical Society) and his Ph.D. degree in neuroscience from Boston University. He completed a neurology residency and a Howard Hughes Medical Institute physician post-doctoral fellowship at the University of Pennsylvania. He joined the faculty at the University of Pennsylvania Department of Neurology in 1997 and was promoted to Associate Professor in 2004. Dr. Crino directed the Penn Tuberous Sclerosis Complex Clinic from 1998-2012. He served as director of the Penn Epilepsy Program from 2007-2012. He joined the neurology faculty at Temple University School of Medicine in 2012 as professor where he served as vice chair for Research and director of the Comprehensive Epilepsy Center. In addition, he was an investigator at the Shriners Hospital Pediatric Research Center.

He has served on the board of directors of the Tuberous Sclerosis Alliance and was awarded a Lifetime Service Award in 2008. He is a member of the Developmental Brain Disorders Study Section at NINDS. Dr. Crino served as the scientific program chair for the 2016 American Epilepsy Society annual meeting and co-chair of the 2015 Society for Neuroscience "Neurobiology of Disease" workshop. Dr. Crino was the president of the Philadelphia Neurological Society in 2015-2016. Currently, Dr. Crino is professor and chair of the Department of Neurology at the University of Maryland School of Medicine.

Dr. Crino's laboratory has maintained a translational research program studying mechanisms of altered brain development associated with epilepsy, intellectual disability, and autism for the past 16 years. He has specific expertise in defining developmental disorders associated with intractable epilepsy including tuberous sclerosis complex (TSC), focal cortical dysplasia, and hemimegalencephaly. He has collaborated on identifying several new genes associated with neurodevelopmental disorders including NPRL3, STRADA, and WDR73. He has coauthored more than 140 peer reviewed manuscripts, chapters, and reviews, and has been an internationally invited speaker. He is principal or co-investigator on four current National Institutes of Health grants.

J. KIFFIN PENRY AWARD FOR EXCELLENCE IN EPILEPSY CARE

This award, originally funded by Abbott Laboratories and supported now by the AES J. Kiffin Penry Fund, was established in 1997 in honor of Dr. Penry's lifelong focus and genuine concern for the patient with epilepsy. It recognizes individuals whose work has had a major impact on patient care and improved the quality of life for persons with epilepsy. The award includes a $3,000 honorarium.

Saturday, December 3, 2:15 p.m.
Preceding the Epilepsy Therapies Symposium
Convention Center, General Assembly

Orrin Devinsky, M.D.

Dr. Orrin Devinsky is Professor of Neurology, Neurosurgery, and Psychiatry at the New York University School of Medicine and Director of the Comprehensive Epilepsy Center at NYU Langone Medical Center. He received his B.S. and M.S. from Yale University, M.D. from Harvard Medical School and interned at Boston's Beth Israel Hospital. He completed neurology training at the New York Hospital-Cornell Medical Center and his epilepsy fellowship at the National Institutes of Health.

His epilepsy research includes translational therapies, SUDEP, healthful behavioral changes, epilepsy genetics, autism, neural markers and imaging, therapeutic electrical stimulation, quality-of-life, cognitive and behavioral issues, and surgical therapy.

He is the Principal Investigator for the North American SUDEP Registry and the Sudden Unexpected Death in Children Registry and Research Collaborative. He is a Co-Principal Investigator in the NINDS Center for SUDEP Research, the NIMH Brain Rhythms Assisted Memory Enhancement, and the CDC Managing Epilepsy Well Network. He serves on the Executive Committee of the Epilepsy Foundation SUDEP Institute. He is the lead investigator for the Epidiolex Dravet and Lennox-Gastaut studies and the Ataluren Study in Dravet and CDKL5. He serves on the Scientific Advisory Boards of the Epilepsy Foundation, CDKL5 Program of Excellence, Dup15q Alliance, Tuberous Sclerosis Alliance, and Dravet syndrome. He served on the Board of the Epilepsy Foundation and the American Epilepsy Society. Dr. Devinsky founded Finding A Cure for Epilepsy and Seizures (FACES) and co-founded the Epilepsy Therapy Project and epilepsy.com.

Dr. Devinsky has been a guest on National Public Radio's *RadioLab* and has written pieces for *The New York Times* and *The New Yorker*. Outside interests include behavioral neurology, evolutionary biology and history of neuroscience.

WILLIAM G. LENNOX AWARD

Established in 1966, this award recognizes members of the society who have a record of lifetime contributions and accomplishments related to epilepsy. The award is funded by the Lennox and Lombroso Trust Fund, established in 1962 to advance and disseminate knowledge concerning epilepsy in all of its aspects—biological, clinical and social—and to promote better care and treatment for persons with epilepsy. The award includes a $10,000 honorarium.

Monday, December 5, 8:45 a.m.
Preceding the Merritt-Putnam Symposium
Convention Center, General Assembly

Gary W. Mathern, M.D.

Dr. Gary W. Mathern holds the Davies-Crandall Endowed Chair for Epilepsy Research at The University of California, Los Angeles (UCLA). He graduated sum cum laude from Morehead State University in Kentucky, with a dual major in Biology and Chemistry in 1978. He received his degree in medicine from Case Western Reserve University, Cleveland, in 1982. Dr. Mathern completed his Neurosurgery residency at UCLA in 1991, followed by a Clinical Neurophysiology/Epilepsy fellowship under Jerome Engel, Jr., in 1993. He has been on the Neurosurgery faculty at UCLA since 1992.

Dr. Mathern is known worldwide for his advancements in epilepsy research, which he began as a Neurosurgery resident in the UCLA laboratory of Thomas L. Babb, Ph.D. With the reward of a Milken Family Foundation Young Investigator Award in 1991, Mathern continued his passion for research, including clinical-pathologic studies of mesial temporal lobe epilepsy including hippocampal sclerosis and other etiologies in adults and children undergoing epilepsy neurosurgery. He was awarded the James A. Shannon Director's Award from the National Institutes of Health (NIH), in 1998, and a Clinical Investigator Development Award (K08) in 1993.

In 1997, he shifted his career and research as neurosurgical director of UCLA's Pediatric Epilepsy Surgery Program. Over the next 25 years, Dr. Mathern published more than 200 peer reviewed papers and book chapters. These addressed clinical and basic science aspects of pediatric epilepsy surgery including newer imaging techniques, improving surgical techniques, long-term outcomes, and rehabilitation using neuroplasticity. His laboratory research has characterized genomic abnormalities, neuroanatomy and cellular properties in cortical dysplasia, Rasmussen Encephalitis, and other etiologies related to epilepsy surgery in children.

Dr. Mathern continues to serve the epilepsy community with the NIH, nongovernmental organizations, the Epilepsy Foundation, and the American Epilepsy Society (including scientific program chair, board of directors, and the nominating committee) and the International League Against Epilepsy as one of the editors-in-chief of *Epilepsia* and *Epilepsia Open*.

AWARD RECIPIENTS

AES selects recipients for the following travel awards based on the scientific merit of submitted abstracts. Congratulations to the 2016 travel award winners.

Suzanne and Peter Berry International Travel Awardees

This award recognizes and honors two young investigators conducting clinical neuroscience research related to epilepsy in Asia, Africa, Oceania, the Middle East or Latin America. Awardees receive $1,000 travel stipend along with complimentary meeting registration.

Contact Author	Abstract Title	Poster/Platform
Felix Benninger, M.D.	Toll-like Receptor 3 Increases Epileptogensis and Exacerbates Neuroinflammation	3.138 |B.09
Aaron Warren, M.Sc., BPsych	Thalamocortical Functional Connectivity in Lennox-Gastaut Syndrome is Maximally Enhanced in Mediodorsal and Ventrolateral Nuclei	3.217

Grass Travel Awardees

This award recognizes and honors outstanding young investigators conducting research in basic or clinical neuroscience related to epilepsy. The Grass Foundation and AES combine resources to provide awardees with a $1,000 travel stipend and complimentary meeting registration.

Contact Author	Abstract Title	Poster/Platform
Milad Afrasiabi, Ph.D. (candidate)	Distinct Inhibitory Regulation of Dentate Granule Cells and Semilunar Granule Cells	1.145
Zane Lybrand, Ph.D.	Activity Dependent Regulation of Adult Born Neurons in Epilepsy	1.003
Wolfgang Muhlhofer, M.D.	Optimal Duration of Iatrogenic Coma in Treatment Refractory Status Epilepticus	1.195 | C.04
Alison Muir, Ph.D.	Hunting for the Genetic Cause in SCN1A Mutation Negative Dravet Syndrome Patients	3.327
Iren Orosz, M.D.	Diffusion Tensor Imaging: A Non-Invasive Surrogate Marker of Intracranial High Frequency Oscillations	3.034
Genevieve Rayner, Ph.D.	Mechanisms of Memory Impairment in Epilepsy Depend on Age at Disease Onset	1.320
Hannah Stamberger, M.D.	Recessive Variants in VARS Cause a Clinical Syndrome with Severe Developmental Delay, Epilepsy and Microcephaly and Might Cause Intra-Uterine Lethality at the Other End of the Spectrum	2.290
Yu Wang, M.D., Ph.D.	Models and Mechanisms of SPTAN1 Epileptic Encephalopathy	3.027 | A.07

Young Investigator Awardees

This award recognizes young investigators conducting basic, translational, or clinical epilepsy research. Awardees receive a $1,200 travel stipend. *The AES Young Investigator Awards are supported in part by a charitable grant from Medtronic.*

Contact Author	Abstract Title	Poster/Platform
Saad Abbasi, Ph.D.	Modeling Chd2-linked Epilepsy in Mice	3.133 | B.08
Sophie Adler	Whole-Brain Mapping of Gliosis in Temporal Lobe Epilepsy Using FLAIR	2.123
Jane Allendorfer, Ph.D.	Attention and Inhibitory Control During fMRI in Patients Taking Cannabidiol for Intractable Epilepsy	1.232
Boris Bernhardt, Ph.D.	Structure-Function Phenotypes in Temporal Lobe Epilepsy	1.216
Melanie Boly, M.D., Ph.D.	Global and Local Sleep Homeostasis in Patients with Focal Epilepsy: A High-density EEG Study	1.039 | A.08
Pablo Casillas-Espinosa, M.D., Ph.D.	Anti-Epileptogenic Effects of a Selective T-type Ca2+ Channel Antagonist, Z944, in the Post-Status Epilepticus Model of Temporal Lobe Epilepsy	2.183
Maria Centeno, M.D., Ph.D.	Better Together? EEG-fMRI and ESI Improve Localization Accuracy and Predict Surgical Outcome in Paediatric Focal Epilepsy	2.143
Nitish Chourasia, M.D.	Lateralization of Epilepsy and Response to Vagal Nerve Stimulation Therapy	2.098
Daniel Goldenholz, M.D., Ph.D.	Predicting the Variability of Seizure Frequency: The Pathway to Precision	1.072
Xiaosong He, Ph.D.	Machine Learning Prediction of Seizure Outcome with Presurgical Resting State fMRI Data	2.137

AWARD RECIPIENTS

Contact Author	Abstract Title	Poster/Platform
Anna Jeong, M.D.	Systemic Disease Manifestations and Epilepsy in Tuberous Sclerosis Complex: 1816 Patients Enrolled in the TSC Natural History Database Study	1.168
Hui Ming Khoo, M.D., Ph.D.	Interictal Epileptic Discharges-Related BOLD Responses Delineate the Seizure Onset Zone	1.231
Tara Klassen, Ph.D.	Genetic Risk Patterns in Sudden Death Are Linked with Age-Dependent Splice Variant Expression	3.331
Jochen Meyer, Ph.D.	Asynchronous Suppression of Superficial Cortex during Absence Seizures	3.132 |B.03
Laura Montier , MS., Ph.D. (candidate)	Novel Adenosine Therapy in Dravet Syndrome	3.228
Kenneth Myers, M.D., Ph.D.	Does Koolen-de Vries Syndrome Have a Distinctive Epilepsy Phenotype?	2.286
Jolien Roovers, M.Sc.	Search for De Novo Variants in MicroRNA Genes That Cause Epileptic Encephalopathies	2.289
Tristan Shuman, Ph.D.	In Vivo Interneuron Circuit Dysfunction in Chronically Epileptic Mice	1.146 | B.02
Benjamin Tolchin, M.D., M.S.	Non-Adherence with Psychiatric Care among Patients with Psychogenic Nonepileptic Seizures	1.247 | C.01
Britta Wandschneider, M.D.	Developmental Neuroimaging Markers Co-segregate in Juvenile Myoclonic Epilepsy Patients and Unaffected Siblings	2.122

Nurse Travel Awardees

This award recognizes and honors outstanding young investigators with nursing degrees who are conducting epilepsy research. Awardees receive a $1,000 travel stipend.

Contact Author	Abstract Title	Poster/Platform
Eliana Kovitch Thropp, MSN, APRN, CPNP	Infantile Spasms in Chromosomal 16p11.2 Abnormalities: Case Report and Review of Literature	2.349
Michael Mackow, BSN, RN	Clinical Management of Epilepsy Guided by Responsive Neurostimulation Device	3.128
Tara Myers, MSN, RN, CPNP	Synergestic Effect of Combining Vagus Nerve Stimulation with Ketogenic Diet Therapy in Patients with Drug Resistant Epilepsy	3.301
Althea Wasson, MSN, RN	Development of a Real Time Quality Indicator Assessment Program for Use in the Epilepsy Monitoring Unit	1.092

Additional Awards/Honors

JOHN (JACK) M. PELLOCK AWARD IN PEDIATRIC EXCELLENCE

The 2016 John (Jack) M. Pellock Award for Pediatric Excellence recognizes and honors one outstanding young investigator conducting clinical research in pediatrics related to epilepsy.

Contact Author	Abstract Title	Poster/Platform
Seok-Jun Hong	Multimodal Connectome Organization Across the Spectrum of Cortical Malformations	1.223

REBECCA GOLDBERG KAUFMAN HONOR

The Rebecca Goldberg Kaufman Ethical Neuropsychiatry Fund works to advance a better understanding of the psychiatric aspects of epilepsy care and treatment. The Kaufman Honor is awarded to the highest ranking abstract in the comorbidities topic category and is sessioned as Platform C.01.

Contact Author	Abstract Title	Poster/Platform
Benjamin Tolchin, M.D., M.S.	Non-Adherence with Psychiatric Care among Patients with Psychogenic Nonepileptic Seizures	1.247 | C.01

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Located in the Annual Meeting section.

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EEG Machine Becomes Research Dollars for American Epilepsy Society

For eight years, Nihon Kohden has generously supported a unique giving program it pioneered. Each year, it has auctioned off its EEG-1200 machine and donated 100% of the proceeds to the Lennox and Lombroso Trust for Research and Training and the Susan S. Spencer Fund for Clinical Education and Research.

The auction winner will be announced at the 2016 AES Annual Meeting.

Nihon Kohden executives will present a check to AES on Saturday, December 3, in the Exhibit Hall.

AES deeply appreciates the support of Nihon Kohden America, Inc., Booth #413. Thank you!

NIHON KOHDEN

SUPPORTER RECOGNITION SUMMARY

AES Recognizes the Following Companies for Supporting AES and the 2016 Annual Meeting*

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Eisai Inc.

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CHI St. Luke's Health
Children's Memorial Hermann Hospital/Mischer Neuroscience Institute
Electrical Geodesics, Inc.

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ANT North America
Ripple LLC
Empatica
Ad-Tech Medical Instrument Corp.
Elekta, Inc.
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**Status as of 10/26/2016. See supporter signs at the Annual Meeting for updated recognition levels.*

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WHO HAVE SHARED THEIR **TIME** AND **TALENTS**
TO **ADVANCE OUR MISSION** AND MADE
MEANINGFUL CONTRIBUTIONS
TO THE FIELD OF EPILEPSY.

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AES ANNUAL MEETING

DECEMBER 2 - 6, 2016 | HOUSTON TEXAS

EDUCATIONAL PROGRAM

8:30 a.m. – 11:30 a.m.

Epilepsy Specialist Symposium | Choosing from the Feast of Epilepsy Surgery Procedures: How Do We Decide the Best Course for Each Patient?

Convention Center, General Assembly

OVERVIEW

This symposium addresses the educational need for a contemporary survey of the constellation of invasive approaches to the medication-refractory epilepsy, and the ways in which different approaches might be selected or sequenced in order to provide optimal benefit for individual patients. The format will include an illustrative case presentation, a brief review of historical and recent surgical approaches, a mixed didactic and debate format including current understanding of neuropsychological outcomes and a panel discussion. Technical details of responsive neural stimulation and anterior thalmic stimulation will be minimal because of extensive attention paid to these techniques elsewhere at this meeting. Vagus-nerve stimulation will not be discussed.

LEARNING OBJECTIVES

Following participation in this session, learners should be able to:

- Delineate and describe the possible invasive choices for individual patients with medically refractory epilepsy so as to optimize the complex trade off among maximum benefit, minimum morbidity and minimum number of procedures.
- Educate patients that physicians must make trade-offs in recommending the best approach for their individual circumstances.
- Delineate their role in the new and more complex terrain of epilepsy surgery which makes said role more challenging and potentially more important.

TARGET AUDIENCE

Intermediate and Advanced

PROGRAM

Co-Chairs: Charles M. Epstein, M.D., and Robert E. Gross, M.D.

Introduction and Case Presentation
Charles Epstein, M.D.

Overview of Current Invasive Procedures
S. Kathleen Bandt, M.D.

Cognitive Morbidity
Natalie Voets, D.Phil, and Daniel Drane, Ph.D.

Mesial Temporal Lobe Epilepsy: Minimal
Robert Gross, M.D.

Mesial Temporal Lobe Epilepsy: Maximal
Michael Sperling, M.D.

Extra-Temporal Lobe Epilepsy: Minimal
Ryder Gwinn, M.D.

Extra-Temporal Lobe Epilepsy: Maximal
Steven Roper, M.D.

Hypothalamic Hamartomas: One Disorder Treated by Multiple Approaches
Daniel Curry, M.D.

Radiosurgery for Epilepsy: Multiple Disorders treated by One Approach
Jean Regis, M.D.

Case Outcome and Panel Discussion: Making the Best Decisions
All Faculty

EDUCATION CREDIT

3.0 CME Credits

Nurses may claim up to 3.0 contact hours for this session.

Pharmacists: AKH Inc., Advancing Knowledge in Healthcare approves this knowledge-based activity for 3.0 contact hours (0.3 CEUs). UAN 0077-9999-16-082-L01-P. Initial Release Date: 12/2/16.

KOaPOTTWeaXYWQYKU

The AES Fellows program encourages epilepsy fellows in training to attend the AES Annual Meeting, where they will learn about advances in epilepsy care and research and have opportunities to engage with expert mentors and peers. Program activities include a pre-meeting dinner with the AES Board of Directors, breakfast and lunch with mentors and career panel sessions highlighting a variety of career paths for clinicians and researchers. In addition, Fellows attend the Epilepsy Specialist Symposium, the Annual Fundamentals of Epilepsy Symposium and the Hoyer Lecture.

Approximately 85 fellows will participate in this year's program, including clinical fellows from approved epilepsy fellowship programs in the U.S. and Canada, as well as postdoctoral researchers and fellows in professional fields such as nursing, pharmacology, and psychology. Accepted participants receive a travel stipend, complimentary meeting registration, and a one-year AES membership.

The 2016 -2017 AES Fellows program is supported in part by educational grants from Eisai Inc., Lundbeck, Upsher-Smith Laboratories, Inc., Sunovion Pharmaceuticals Inc., Supernus Pharmaceuticals, Inc., GW Pharmaceuticals, and Acorda Therapeutics.

9:00 a.m. - 4:00 p.m.

30th Annual Advances in the Management of Epilepsy and the Epilepsy Clinic

Separate registration required.

Hilton, Lanier Grand Ballroom B, Level Four

This intensive one-day conference is designed for those professionals who participate in the care of persons with epilepsy. The overall purpose is to improve services to individuals and families affected by epilepsy. The conference is presented by the Department of Neurology of Wake Forest University School of Medicine, Winston-Salem, North Carolina, through an unrestricted grant committed to the education of health professionals, in an effort to promote the comprehensive care of those with epilepsy and their families.

Registration for this program is done separately from the AES Annual Meeting and begins on September 2, 2016. Register by calling Wake Forest School of Medicine at 800.642.0500.

This activity has been approved for *AMA PRA Category 1 Credit™*. Wake Forest is the accrediting entity.

12:30 p.m. - 3:00 p.m.

Annual Fundamentals Symposium | The New Definition and Classification of Epilepsy

Convention Center, General Assembly

OVERVIEW

This session will review the 2014 new definition of epilepsy, the new International League Against Epilepsy (ILAE) classification of seizures and epilepsy (these are official ILAE positions), the presentation and prognosis of acute symptomatic seizures and possible scientific underpinnings of the definition and classification of seizures. Participants will become familiar with application of the definition of epilepsy and new classification system for seizures. Familiarity with these areas will allow better determination in practice of who has epilepsy, who has outgrown it, what type of seizures they have and when seizures are "reactive."

LEARNING OBJECTIVES

Following participation in this symposium, learners should be able to:

- Review and discuss the ILAE definition of epilepsy and how it impacts clinical practice.
- Differentiate between epilepsy and an epilepsy syndrome.
- Recognize when a seizure is precipitated by an antecedent event rather than being an unprovoked seizure.
- Classify seizures according to the ILAE Operational Classification of Seizure Types system.

TARGET AUDIENCE

Intermediate

PROGRAM

Chair: Robert Fisher, M.D., Ph.D.

Introduction
Robert Fisher, M.D., Ph.D.

New Definition of Epilepsy
Jacqueline French, M.D.

New Classification of Seizure Types
Robert Fisher, MD, Ph.D.

New Roadmap for Epilepsy
Ingrid Scheffer, M.D., Ph.D.

New Insights about Acute Symptomatic Seizures
Dale Hesdorffer, Ph.D.

Towards a Scientific Basis for Definition and Classification of Epilepsy
Ivan Soltesz, Ph.D.

Conclusions
Robert Fisher, M.D., Ph.D.

EDUCATION CREDIT

2.5 CME credits

Nurses may claim up to 2.5 contact hours for this session.

Pharmacists: AKH Inc., Advancing Knowledge in Healthcare approves this knowledge-based activity for 2.5 contact hours (0.25 CEUs). UAN 0077-9999-16-083-L01-P. Initial Release Date: 12/2/16.

COMMERCIAL SUPPORT ACKNOWLEDGEMENT

Supported in part by educational grants from Eisai Inc., Lundbeck, and Supernus Pharmaceuticals, Inc.

1:00 p.m. - 3:00 p.m.

Epilepsy Fellowship Program Directors Meeting

Convention Center, Room 320 B

Chair: David Ficker, M.D.

This third annual session is intended for current Epilepsy Fellowship Directors, Clinical Neurophysiology Program Directors, and those interested in starting an ACGME-accredited Epilepsy Fellowship and will meet ACGME program requirement II.A.4, that advises program directors to attend one program director meeting per year. The goal of this session is to provide a forum for program directors to address challenges encountered in running a program and in meeting accreditation requirements. This year the session will provide an update on the AES Fellowship Core Curriculum Project, that will serve as a resource for program directors of Epilepsy Fellowship Programs. Also to be discussed is board certification for faculty and graduating fellows and strategies for success in this regard. Additionally, the fellowship match process will be discussed in which two match options (NRMP and SF match) will be presented and discussed. A formal vote on these two match options (thumbs up/thumbs down) will take place post-meeting.

EDUCATION CREDIT

2.0 CME credits

1:30 p.m. - 3:00 p.m.

Professional Development: Grow Your Network

Hilton, Lanier Grand Ballroom J, Level Four

Interested in enlarging your circle of professional connections? Make plans to attend this special session by AES. Learn how to take full advantage of the networking opportunities available at the meeting to grow your network.

1:30 p.m. - 3:00 p.m.

Special Interest Groups

Epidemiology | Access and Utilization of Health Care for Patients with Epilepsy

Convention Center, Room 360 A, D

Coordinators: Christine Baca, M.D., M.S.H.S., and Zachary Grinspan, M.D.

Speakers: Christine Baca, M.D., Nicholas Schiltz Ph.D., Charles Begley Ph.D., Jana Jones, Ph.D., Mary Jo Pugh, Ph.D., and Zachary Grinspan, M.D.

Using different data sources and methods (big administrative, prospective observational, qualitative and implementation based), this session will address how different non-disease related factors (i.e., race/ethnicity, socioeconomic status, residence, language, health insurance, health beliefs, and preferences) can be associated with access to and utilization of different types of health care and outcomes for people with epilepsy.

Global Health in Epilepsy | Get Involved!

Convention Center, Room 330 A

Coordinators: Sheryl Haut, M.D., and Dave Clarke, M.B.B.S.

Speakers: Dave Clarke, M.B.B.S., Jorge Burneo, M.D., M.S.P.H., and Sheryl Haut, M.D.

Participation in epilepsy-related global health projects is exciting, productive and important! Many people are still unaware of the possible avenues to pursue global health. This SIG will focus on opportunities for international collaborations, professorships and partnerships around the world, both in person and via telemedicine. The speakers will discuss challenges across different regions, successes and lessons learned. Areas to be highlighted include ILAE supported activities such as the Partnering Epilepsy Centers in the Americas (PECA) program of the North American Commission of the ILAE, epilepsy care in the Caribbean, epilepsy surgery project in Peru, and visiting professorship programs in Africa.

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Convention Center, Room 361 A, D

Coordinators: Catherine Schevon, M.D., Ph.D., and William Stacey, M.D., Ph.D.

Speakers: Richard Staba, Ph.D., Lisa Bateman, M.D., Sydney Cash,M.D., Ph.D., and John Wittig, Ph.D.

An increasing number of centers are conducting high-density or microelectrode studies in epilepsy patients undergoing invasive EEG recordings. These recordings are valuable for a broad range of epilepsy and cognitive neuroscience investigations. The session will provide a review of technical and clinical issues that are likely to be encountered. These range from environmental and equipment issues that can impact recording quality, interactions between research and clinical recording systems, patient safety concerns and handling of the resulting large data sets. Format will be short talks followed by a panel discussion with audience participation encouraged.

Surgery | Battle Royale II: StereoEEG vs Subdural Electrodes

Convention Center, Room 371 A, D

Coordinators: Saadi Ghatan, M.D., and Gerald A. Grant, M.D.

Building on the success of last year's session, the discussion continues on difficult surgical cases that could be undertaken with either approach. StereoEEG is gaining popularity in the United States, and both European and American experts in the field are invited to participate in the debate over the efficacy and utility of each approach.

Translational Research | Conducting Translational Research: Challenges, Realization and Recognition

Convention Center, Room 370 A, D

Coordinators: Andrew J. Cole, M.D., and Martha Morrell, M.D.

Speakers: Alica Goldman, M.D., Ph.D., Tom Sutula, M.D., Ph.D., and Merit Cudkowicz

Taking a promising therapy from bench to bedside requires a network of collaborators, a timeline of years, a variety of funding sources and a willingness to accept the risk that such significant effort might not lead to a realized treatment. The risk is mitigated when the investigator selects collaborators with the right skill sets and experience, when consortium organization and management are well thought and when a long-term regulatory strategy is included in the earliest clinical trial considerations. Finally, academicians who wish to pursue translational research must be recognized and rewarded for efforts that are quite

different from lab based and single center clinical research. Specific learning objectives: 1) provide concrete advice about how to establish a translational research consortium, including identifying the investigators and funding and managing start-up efforts; 2) stress the importance of considering the potential regulatory pathway when designing the earliest clinical trials; and 3) discuss how investigators engaged in translational research can receive career recognition for their collaborative efforts.

3:15 p.m. – 4:00 p.m.

Career Pathways in Epilepsy Care and Research

This session will highlight a variety of career paths, with two concurrent panels focused on clinical epilepsy and basic science research. Participants will self-select and may go between sessions in adjacent rooms. Each panel will represent career options in different sectors, such as private practice, academia, industry, and non-profit organizations. Panelists will describe their work, provide advice to those interested in similar careers and answer questions from the audience. This session is open to all meeting attendees, but may be of particular interest to residents, fellows and others at an early career stage or considering a transition.

Career Pathways in Epilepsy Care and Research – Clinical Epilepsy

Convention Center, Room 332 B

Panelists: Alison Pack, M.D., M.P.H., Kelly Knupp, M.D., Lynn Kramer, M.D., F.A.A.N., and Robert T. Wechsler, M.D., Ph.D., F.A.A.N.

Career Pathways in Epilepsy Care and Research – Basic and Translational Research

Convention Center, Room 332 A

Panelists: Lori L. Isom, Ph.D., Eric Marsh, M.D., Ph.D., Brandy Fureman, Ph.D., and Andrew Carrel, Ph.D.

3:30 p.m. – 6:00 p.m.

Spanish Symposium | How to Evaluate and Ameliorate the Treatment Gap in Epilepsy: Important Considerations for Spanish-speaking Countries

Presented in Spanish.

Convention Center, Room 351 A, D

OVERVIEW

Epilepsy is estimated to affect over 60 million people worldwide, the majority of whom live in low and middle-income countries where access to medical treatment is limited. Cost-effective epilepsy treatments are available and an accurate diagnosis can be made without much technological equipment. Nonetheless, a vast majority of individuals with epilepsy in many resource-poor regions do not receive treatment. Anecdotal and descriptive estimates suggest a treatment gap of more than 80% in many low-income countries. In recent years, many countries have undertaken initiatives to decrease the epilepsy treatment gap, notably the demonstration projects such as the Global Campaign Against Epilepsy, conducted jointly by the International League against Epilepsy, the International Bureau for Epilepsy and the World Health Organization. However, measuring the gap, defined in simple terms as the proportion of people with epilepsy who require treatment but do not receive it, is challenging. There are numerous methodological issues to be considered and many cultural, demographic, economical and logistical factors that evidently influence this treatment gap and are themselves difficult to assess. This symposium will address the gaps in primary care, pharmacological and surgical care as well as in social acceptance and development in epilepsy as related to specific social, economic and political conditions in Spanish-speaking countries.

LEARNING OBJECTIVES

Following participation in this session, learners should be able to:

- Define the term "treatment gap" and describe the relative impact regarding current practices in pharmacological treatment, neurophysiologic resources and surgical treatment of epilepsy in Spanish-speaking countries that contribute to this gap.
- Describe standardized methods for measuring the aforementioned treatment gap.
- Delineate the specific cultural, economic and social factors identified in Spanish-speaking countries that contribute to this treatment gap.
- Review and discuss strategies for reducing this treatment gap in Spanish-speaking countries.
- Differentiate between strategies at the biological, social and economic levels that should be adapted or even copied from higher income countries and actions that must necessarily take specific circumstances into account and must be developed locally.

TARGET AUDIENCE

Basic

PROGRAM

Co-Chairs: Mario A. Alonso-Vanegas, M.D., and David King-Stephens, M.D.

Introduction

Mario A. Alonso-Vanegas, M.D.

Treatment Gap, How Can It Be Qualified and Quantified? How Do We Stand in Spanish-speaking Countries?
Jorge Burneo, M.D., M.S.PH.

Pharmacological Treatment Gap in Spanish-speaking Countries
Juan Jesús Rodríguez Uranga, M.D.

Neurophysiology Resources Gap in Spanish-speaking Countries
Silvia Kochen, M.D.

Surgical Treatment Gap in Spanish-speaking Countries
Jorge Álvaro González-Martínez , M.D., Ph.D.

Conclusions
David King-Stephens, M.D.

EDUCATION CREDIT
2.5 CME credits

Nurses may claim up to 2.5 contact hours for this session.

Pharmacists: AKH Inc., Advancing Knowledge in Healthcare approves this knowledge-based activity for 2.5 contact hours (0.25 CEUs). UAN 0077-9999-16-084-L01-P. Initial Release Date: 12/2/16.

4:00 p.m. - 5:30 p.m.

14th Judith Hoyer Lecture in Epilepsy | The SUDEP Movement: From Inception to the Goal

Convention Center, General Assembly

Lecturer: Elson So, M.D.

Award Presentation: AES Distinguished Service Award

NINDS Update: Walter J. Koroshetz, M.D., *National Institute of Neurological Disorders and Stroke*

SUDEP appeared to be an epilepsy epiphenomenon mostly noted in case reports until researchers in the 1980s highlighted its importance with basic science research and clinical series of postmortem cases. Over the next decade, medical interest in SUDEP increased steadily, but it was the advocacy of dedicated family and friends of SUDEP persons that placed SUDEP in the consciousness of the medical communities and the public. It has been 10 years since the start of an ongoing and growing collaboration between epilepsy organizations and government agencies in the effort to stop SUDEP. This presentation will summarize the results of that unique collaborative effort, and it will also attempt to project the promise of SUDEP research, education and advocacy.

SUPPORT ACKNOWLEDGEMENT
Supported in part by educational grants from Upsher-Smith Laboratories, Inc. and the National Institute of Neurological Disorders and Stroke (NINDS).

6:00 p.m. - 7:30 p.m.

Special Interest Groups

Epilepsy and Aging | Hot Topics in Aging, Alzheimer's Disease and Epilepsy

Convention Center, Room 310 B

Coordinators: Bruce Hermann, Ph.D., and Helen Scharfman, Ph.D.

Speakers: Erik Roberson, M.D., Ph.D., Daniel Friedman, M.D., and Cynthia Harden, M.D.

Three speakers will address hot topics. The first is Erik Roberson, M.D., Ph.D., a clinician-scientist who will address the surprising and robust anticonvulsant effects of tau, a molecule critical to Alzheimer's disease. The second is Daniel Friedman, M.D., who will discuss the issues raised by basic science and clinical research in Alzheimer's disease and epilepsy. The third speaker, Cynthia Harden, M.D., will address the current view of how seizures change at menopause and andropause.

Ictal Semiology | Detailed Examination of Seizure Semiology to Determine Onset and Spread of Ictal Discharges

Convention Center, Room 361 A, D

Coordinators: Andrew Bleasel, M.B.B.S., Ph.D., and Philippe Kahane, M.D., Ph.D.

Speakers: Andrew Bleasel, M.B.B.S., Ph.D., Hans Luders, M.D., Ph.D., Philippe Kahane, M.D., Ph.D., and Felix Rosenow, M.D.

This popular SIG presents clinical cases to illustrate how seizure semiology can be used in the localization of seizure onset and routes of ictal propagation. The panel and the audience are challenged in the detailed examination of seizure semiology with four to six cases of typical or unusual seizures. Format: one short slide presentation, one video, discussion, one slide showing the explanation. After showing each video members of the audience will be invited to describe and analyze seizure semiology and to form hypotheses of seizure onset and spread. The faculty will comment on the material with brief discussion of particular clinical features. The presenter will give the final explanation based upon neuroimaging, intracranial EEG and the surgical outcome. Brief didactic material is delivered to end each case. The format of the session is interactive with the main aim to show the audience how to use subjective and observable clinical elements to localize the seizure onset and reconstruct the propagation pattern of the ictal discharge.

Neuroendocrinology | Neuroendocrine Aspects of Sleep in Epilepsy

Convention Center, Room 330 A

Coordinator: D. Samba Reddy, Ph.D., R.Ph.

Speakers: Mathew Jones, Ph.D., Rama Maganti, M.D., and Amy Z. Crepeau, M.D.

Sleep plays an intricate role in epilepsy. Sleep can affect the frequency and occurrence of seizures. Nearly 35% of U.S. adults are not getting the recommended seven hours of sleep every night. Sleep deprivation is a trigger of seizures in many persons with epilepsy. It is well-known that seizures are very sensitive to sleep patterns. Some patients have their first seizure or repeated seizures after an "all-nighter" at college or after not sleeping well for long periods. Sleep can affect seizures in many different ways. During normal sleep-wake cycles, changes in the brain's electrical and hormonal activity occur. These changes can be linked to patterns of sleep and seizures. Some forms of epilepsy are especially prone to sleep problems. On the contrary, sleep may not be a common trigger, or the association is less clear in some patients. Despite the complexity of the sleep-seizure relationship, the prognosis is a favorable one for patients with sleep disorders and epilepsy. Yet improving sleep and optimizing seizure control can have significant positive effects on the quality of life of these patients. There are many outstanding questions on sleep and epilepsy. How can sleep deprivation trigger an epileptic seizure? How do circadian and hormonal changes influence sleep pattern and seizure occurrence? Can hormones or sleeping pills help with sleep in epilepsy? The 2016 Neuroendocrinology SIG session will discuss these and many other questions through expert presentations and panel discussion, with an emphasis on sleep clock, hormone changes, risk factors and possible prevention strategies.

Neuroimaging | The "Normal-appearing" White Matter in Focal Epilepsy: From Pathology to Imaging

Convention Center, Room 371 A, D

Coordinators: Neda Bernasconi, M.D., Ph.D., and Boris Bernhardt, Ph.D.

Speakers: Maria Thom, Ph.D., and Neda Bernasconi, M.D., Ph.D.

Speakers will discuss white matter anomalies in temporal lobe epilepsy and focal cortical dysplasia. They will present the most recent findings derived from histology, ex vivo and in vivo MRI and connectomics.

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Convention Center, Room 330 B

Coordinators: Tara Myers, CPNP, and Wendy Miller, Ph.D., RN, CCRN

Speaker: Wendy Miller, Ph.D., RN, CCRN

Last year's SIG focused on literature review, evidence-based practice skills and the nursing research process. Building on that discussion, this year's session will delve further into the details of conducting research. Attendees will learn about research methods, implementation and evaluation.

Seizure and Cerebrovascular Disease | Acute Cerebrovascular Disease and Acute Seizures

Convention Center, Room 310 A

Coordinators: Naim I. Haddad, M.D., and David Chuang, M.D.

Speakers: Peter Forgacs, M.D., Matthew Maas, M.D., and Jong Woo Lee, M.D., Ph.D.

There is great clinical and conceptual interest in the interface of epileptic and cerebrovascular illnesses. This session will center on seizures in the setting of acute intracranial hemorrhages. Presentations and speakers:

- EEG and Seizures after Subarachnoid Hemorrhage with Aneurysm Coiling - Peter Forgacs, M.D.
- From Ubiquitous Antiseizure Medication Prophylaxis to Targeted Seizure Treatment in Acute Subarachnoid Hemorrhage - Matthew Maas, M.D., and Elizabeth Gerard, M.D.
- Choice of Antiepileptic Drugs in Acute Subdural and Intraparenchymal Hemorrhages - Jong Woo Lee, M.D., Ph.D.

6:00 p.m. – 7:30 p.m.

Basic Science Skills Workshop

Pre-registration and tickets are required for this session. An additional $50 registration fee applies; maximum of 30 people per session.

Convention Center, Room 340 A

EEG Analysis and Seizure Detection in Experimental Models

Moderators: Brian Litt, M.D., and Edward H. Bertram, M.D.

This interactive workshop for basic scientists will focus on the analysis of EEG data obtained from animal models of epilepsy, with an emphasis on approaches to seizure detection.

7:00 a.m. - 8:30 a.m.

Junior Investigators Roundtable Discussions

Convention Center, Room 332 B

This session will provide an opportunity for junior investigators to connect with each other, senior investigators and other experts in an informal setting to share ideas, strategies, and challenges. Expert facilitators will lead small group discussions on a range of topics, and participants may rotate among topics of interest. Space will be available for general networking as well.

Table Topics and Expert Facilitators

Publishing Your Research

Helen E. Scharfman, Ph.D., Christophe Bernard, Ph.D., Nathalie Jette, M.D., M.Sc., F.R.C.P.C., and Gregory D. Cascino, M.D.

Funding Your Research

Vicky Whittemore, Ph.D., Adam Hartman, M.D., Julie B. Milder, Ph.D., Manisha Patel, Ph.D., and Cara Long, Ph.D.

Navigating the Job Market

Robert Fisher, M.D., Mackenzie C. Cervenka, M.D., Edward Glasscock, Ph.D., and Amy Brooks-Kayal, M.D.

Balancing Research and Clinical Duties

Ethan M. Goldberg, M.D., Ph.D., and Page B. Pennell, M.D.

Starting/Managing a Lab

Tara Klassen, Ph.D., and Jamie L. Maguire, Ph.D.

7:00 a.m. - 8:30 a.m.

Putting the "Public" Back in Health: Resources and Opportunities Available to People with Epilepsy

Convention Center, Room 371 A, D

Moderators: Rosemarie Kobau, M.PH., MAPP, and David Labiner, M.D.

The 2012 seminal report on epilepsy by the National Academy of Medicine (formerly the Institute of Medicine), galvanized epilepsy stakeholders to advance public health action on epilepsy. This report stimulated projects to expand epilepsy surveillance, refocus attention on comorbidities, explore new care models, emphasize improved access to care and quality of care and facilitate research collaboration. The report also stressed the need for more impactful public and professional education about epilepsy and highlighted opportunities for public health action for epilepsy stakeholders. In response, AES and CDC coordinated this new educational session on utilizing existing public health resources to expand epilepsy programs, services and outcomes to benefit people with epilepsy. Note: Participants will be able to enroll onsite for ongoing technical assistance with a CDC Epilepsy Program Community of Practice.

LEARNING OBJECTIVES:

Following participation in this session, learners should be able to:

- Identify federal, state, and local public health systems relevant for epilepsy stakeholders.
- Delineate public health partners that epilepsy stakeholders can connect with to advance epilepsy surveillance, research and quality of care.
- Describe other chronic disease prevention models supported by federal, state or local public health systems.
- Discuss reimbursement mechanisms that can be employed to provide preventive care, patient education and self-management for other chronic diseases.

7:00 a.m. - 8:30 a.m.

Special Interest Groups

Basic Mechanisms and Neuroscience | From Channels to Vesicles to Synapses

Convention Center, Room 360 A, D

Coordinators: Katty (Jing-Qiong) Kang, M.D., Ph.D., and Joaquin Lugo, Ph.D.

Speakers: Wayne Frankel, Ph.D., Martin Gallagher, M.D., Ph.D., and Mingshan Xue, Ph.D.

Mutations in multiple genes have been associated with epilepsy in humans. These genes have diverse biological functions, which have changed our traditional view of epilepsy as a "channelopathy." This session will provide an update of discoveries in human patients and related animal models that give insights into how disruptions in different cellular events lead to epilepsy.

Critical Care Epilepsy | What's New in Clinical Research

Convention Center, Room 330 A

Coordinators: Cecil Hahn, M.D., and Howard Goodkin, M.D., Ph.D.

Speakers: Markus Leitinger, M.D., Wolfgang Muhlhofer, M.D., Anna Rosati, M.D., Ph.D., Eric Rosenthal, M.D., and Robert Silbergleit, M.D.

This year's SIG will highlight important new clinical research advances in critical care epilepsy. Markus Leitinger will present a study of the Diagnostic Accuracy of the Salzburg Criteria for Nonconvulsive Status Epilepticus. Wolfgang Mulhofer will present a study of the Optimal Duration of Iatrogenic Coma in the Treatment of Refractory Status Epilepticus. Anna Rosati will present an update on an ongoing trial of Ketamine in Refractory Convulsive Status Epilepticus, Eric Rosenthal will present an

update on the ongoing SAGE-547 Treatment as Adjunctive Therapy Utilized in Status Epilepticus (STATUS) Trial and Robert Silbergleit will present an update on the ongoing Established Status Epilepticus Treatment Trial (ESETT) trial.

Dietary Therapies for Epilepsy | Is The Ketogenic Diet Really Worth It? Clinical Challenges of the Ketogenic Diet and an Update on Mechanistic Research Providing Potential Therapeutic Targets

Convention Center, Room 370 A, D

Coordinators: Timothy Simeone, Ph.D., and Amy Kao, M.D.

Speakers: Tanya McDonald, Ph.D. (candidate), Robin SB Williams, Ph.D., Mary Scott Ramnitz, M.D., and Christopher W. Beatty, M.D., Timothy A. Simeone, Ph.D., and Jeffrey Buchhalter, M.D., Ph.D.

As the population of patients who continue on dietary therapies long-term (such as glucose transporter deficiency syndrome, pyruvate dehydrogenase deficiency) increases, the potential for long-term side effects becomes a more critical issue. This session will discuss these long-term risk, and the benefit of collaborating with other subspecialties to develop rational clinical protocols. In a medical climate that is increasingly challenged financially, there are also significant challenges to program development; strategies for overcoming these issues will be discussed and solicited. Clinical difficulties with dietary therapies contribute to the motivation for research into the mechanisms of dietary therapies. Therefore, we will also hear about the latest mechanistic research with emphasis on identification of viable targets with greatest therapeutic potential. Clinical and basic science segments will each be followed by a short panel discussion.

Frontal Lobe | Frontal Lobe Structure and Function in Focal and Generalized Epilepsies

Convention Center, Room 310 B

Coordinators: Andrea Bernasconi, M.D., and Neda Bernasconi, M.D., Ph.D.

Speakers: Fernando Cendes , M.D., Ph.D., Carrie McDonald, Ph.D., and Britta Wandschneider, M.D., Ph.D.

This session will cover new research aimed at understanding the structure and function of the frontal lobe across various epilepsy syndromes.

Genetics | Variant Fight Club 2: The Dark Side of Genetics

Convention Center, Room 361 A, D

Coordinators: Tara L Klassen, Ph.D., and Eric Marsh, M.D., Ph.D.

In this session, selected speakers will provide an overview of the genetic complexity observed within patient (and control) genomes which confound interpretation in molecular diagnostics. A focus on the concerns, considerations and approaches used to translate this information into clinical risk prediction within the genetics clinic will be explored. In addition, the audience will be invited to submit cases for consideration and discussion building on the framework from last year's SIG on Pathogenic Variant Interpretation and Utility. A full 60 minutes of the SIG will be designated for discussion and conversation among the attendees.

Neonatal Seizures | Neonatal Epileptic Encephalopathies

Convention Center, Room 320 B

Coordinators: Tim Benke, M.D., Ph.D., and Courtney Wusthoff, M.D.

Speakers: Tim Benke, M.D., Ph.D., Edward Cooper, M.D., Ph.D. Scott Demarest, M.D., and Dennis Dlugos, M.D.

This SIG will have a roundtable discussion of emerging knowledge of neonatal onset epileptic encephalopathies, including genotype/phenotype correlations, treatment options and clinical testing considerations. The panel will include three – five speakers, each with expertise in a different specific disease (CDKL5, KCNQ2, etc.)

8:30 a.m. - 11:45 a.m.

Presidential Symposium | Epilepsy Care: A Futurist View

Convention Center, General Assembly

Award Presentation: AES Research Recognition Awards in Basic Science and Clinical Science

OVERVIEW

This session will begin by outlining the current state of epilepsy diagnosis and treatment, then identify existing roadblocks and speculate on future trends. Key topics for the current and future management of epilepsy will include: 1) existing, new and future approaches to epilepsy surgery and devices; 2) development of new antiepileptic and antiepileptogenic medications; 3) how understanding molecular mechanisms in signaling pathways like mTOR, and new and future gene discoveries will influence diagnosis and treatment; 4) how the expanding field of bioinformatics will influence decision making now and in the future; and 5) how current and future brain imaging methods will be applied to epilepsy.

LEARNING OBJECTIVES

Following participation in this session, learners should be able to:

- List several molecular pathways that may be altered in people with epilepsy and identify existing treatment(s) that can be applied.
- Describe the process for the development of new antiepileptic drugs.

- Delineate the risks and benefits of the currently available surgical approaches to treating people with epilepsy.
- Employ bioinformatic methods to create performance improvement projects with existing clinical data.
- Select the appropriate currently available bioimaging technique(s) to optimize diagnosis and treatment for people with epilepsy.
- Describe how current and future brain imaging methods can supplement and enhance neuropsychological evaluation and outcomes.

TARGET AUDIENCE
Intermediate and Advanced

PROGRAM
Chair: Michael Privitera, M.D.

Introduction
Michael Privitera, M.D.

Current and Future Approaches to Surgery and Sevices for Epilepsy
Dennis Spencer, M.D.

Harnessing the Power of Bioinformatics in Epilepsy
Tracy Glauser, M.D.

Brain Imaging in Epilepsy Now and in the Future
Jerzy Szaflarski, M.D., Ph.D.

Current and Future Trends in Development of Antiepileptic Drugs
Henrik Klitgaard, Ph.D.

Genes and Signaling Pathways: Future Therapeutic Strategies
The Fritz R. Dreifuss Lecture
Peter Crino, M.D., Ph.D.

Conclusions
Michael Privitera, M.D.

EDUCATION CREDIT
3.0 CME credits

Nurses may claim up to 3.0 contact hours for this session.

Pharmacists: AKH Inc., Advancing Knowledge in Healthcare approves this knowledge-based activity for 3.0 contact hours (0.3 CEUs). UAN 0077-9999-16-085-L01-P. Initial Release Date: 12/3/16.

COMMERCIAL SUPPORT ACKNOWLEDGEMENT
Supported in part by educational grants from Eisai Inc., Lundbeck, UCB, Inc., and Sunovion Pharmaceuticals Inc.

Noon - 6:00 p.m.
Exhibit Hall
Grand opening at noon

Convention Center, Hall A3, B3

Noon - 6:00 p.m.
Poster Session One

Convention Center, Hall A3, B3
Enter in back of Hall A3, behind the 400 aisle.
See page 56-65.

2:15 p.m. - 5:00 p.m.
Epilepsy Therapies Symposium | Risky Business: From Repetitive Seizures to Status

Convention Center, General Assembly

Award Presentation: J. Kiffin Penry Award for Excellence in Epilepsy Care

OVERVIEW
The treatment spectrum of "risky" seizure scenarios from seizure clustering to status epilepticus encompasses a wide variation in recognition of these clinical entities as well as treatment approaches. This symposium will discuss the newest definitions of these entities and the evidence-based guidelines for treating convulsive status epilepticus, as well as treatments for seizure clusters, periodic patterns and subclinical seizures in critically ill patients, and outlying conditions that present as status epilepticus, including neuroinflammatory states. The pathophysiologic underpinnings for the spectrum from seizure clustering to epileptiform patterns in critically ill patients to status epilepticus will be discussed. Treatment algorithms will be presented for each of these "risky" scenarios, with the educational goals of early recognition, identification of the risks of progression, and rapid institution of appropriate treatments.

LEARNING OBJECTIVES
Following participation in this session, learners should be able to:

- Recognize seizure emergencies, impending seizure emergencies (cluster seizures) and complex potentially epileptic clinical scenarios and have a treatment approach in mind so that patients are treated as quickly and appropriately as possible.
- Assist in the rapid identification of seizure emergencies, impending seizure emergencies (cluster seizures) and complex potentially epileptic clinical scenarios; select appropriate treatment approaches so patients are treated as quickly as possible.
- Delineate the uses of interventions for seizure emergencies and clusters and enable their availability in the appropriate clinical settings.

- Describe the perception of seizure clustering by patients and by providers and assist in overcoming the barriers to increased use of seizure rescue strategies outside the hospital.

TARGET AUDIENCE
Basic, Intermediate and Advanced

PROGRAM
Co-Chairs: Cynthia Harden, M.D. and Jerry Shih, M.D.

Introduction
Cynthia Harden, M.D.

New Outlook on Treatment for Status Epilepticus: Evidence-based Guidelines and Beyond
Thomas P. Bleck M.D., MCCM, FNCS

Seizure Clustering: What Is It and How Do We Keep it from Escalating?
Lara Marcuse, M.D.

Subclinical Seizures and Periodic Patterns in the Critically Ill: Experts Debate on When to Treat and When to Not Treat
Lawrence Hirsch, M.D., and Suzette M. LaRoche, M.D.

Status Epilepticus "Outliers": Early Recognition and Prompt Management
Jeffrey W. Britton, M.D.

Conclusions
Jerry Shih, M.D.

EDUCATION CREDIT
2.5 CME credits

Nurses may claim up to 2.5 contact hours for this session.

Pharmacists: AKH Inc., Advancing Knowledge in Healthcare approves this knowledge-based activity for 2.5 contact hours (0.25 CEUs). UAN 0077-9999-16-086-L01-P. Initial Release Date: 12/3/16.

COMMERCIAL SUPPORT ACKNOWLEDGEMENT
Supported in part by an educational grant from Eisai Inc.

2:30 p.m. - 4:00 p.m.

Investigators Workshop

Convention Center, Room 310 B

Whole Exome Sequencing in the Epilepsies: Making Sense of the Sequence Data

Moderators: Sam Berkovic, M.D., and Ingo Helbig, M.D.

Speakers: Sarah Weckhuysen, M.D., Slave Petrovski, Ph.D., and Roland Krause, Ph.D.

5:30 p.m. - 7:30 p.m.

North American Commission Symposium: Controversies in the Management of Epilepsy During Pregnancy

Convention Center, General Assembly

OVERVIEW
Management of women with epilepsy during child-bearing years requires complex decision-making, with the goal of optimal maternal seizure control balanced against potential fetal risks of in utero anti-epileptic drug exposure. An abundance of data is now available to differentiate risks between some AEDs with regard to both structural teratogenecity and neurodevelopmental consequences. However, evidence is still lacking regarding many of the AEDs overall, and details of how best to dose AEDs during critical time windows to optimize maternal and fetal outcomes is an area of active research. Peripartum is a particularly vulnerable time for seizure worsening; data is now available to support the benefits of breastfeeding in women with epilepsy on AEDs but sleep disruption can worsen seizures in many. This session will provide the latest data available regarding these issues, as well as delve into some of the more contemporary controversies including when use of valproate is justifiable in this special patient population, dosing strategies, and when therapeutic drug monitoring should be used and alternative strategies when it is not available.

LEARNING OBJECTIVES
Following participation in this session, learners should be able to:

- List the antiepileptic drugs (AED) that carry relatively higher teratogenic risk (structural and neurodevelopmental).
- Review and discuss how each AED does or does not increase the teratogenic risk.
- Describe gestational pharmacokinetic principles and restate how to adjust medications during pregnancy to maintain seizure control, as well as in the postpartum period.

- Delineate approaches for counseling women regarding strategies to lower seizure risk during the peripartum period.
- Recognize and describe the neurodevelopmental risks of children born to women with epilepsy, with specific considerations of contributory factors that include family history, vitamin use, AED type and dose, and maternal seizure control as well as other obstetric and neonatal complications.
- Identify the neurocognitive profiles to monitor during early child development.

TARGET AUDIENCE

Basic and Intermediate

PROGRAM

Co-Chairs: Sheryl Haut, M.D., and Page B. Pennell, M.D.

Introduction
Sheryl Haut, M.D.

The Valproate Controversy: The Worldwide Perspective
Torbjörn Tomson, M.D.

Pre-pregnancy Planning: AED Choice and Pregnancy Outcomes
Kimford J. Meador, M.D.

Management of Epilepsy During Pregnancy and Postpartum: AED Dosing Strategies
Page B. Pennell, M.D.

Postpartum Management: Risk of Seizures and Safety, Newborn Care and Nursing
Sanjeev Thomas, M.D.

Panel Discussion
All Faculty

EDUCATION CREDIT

2.0 CME credits

Nurses may claim up to 2.0 contact hours for this session.

Pharmacists: AKH Inc., Advancing Knowledge in Healthcare approves this knowledge-based activity for 2.0 contact hours (0.2 CEUs). UAN 0077-9999-16-087-L01-P. Initial Release Date: 12/3/16.

5:30 p.m. - 8:00 p.m.

Interprofessional Care Symposium (formerly Professionals in Epilepsy Care): Interprofessional Assessment and Intervention of the Psychosocial Comorbidities of Epilepsy

Convention Center, Ballroom A

OVERVIEW

This symposium will present recent research and practice evidence regarding how assessing, evaluating and treating the psychosocial comorbidities of epilepsy is part of comprehensive lifespan care for persons with epilepsy and their families. As per the IOM Epilepsy Across the Spectrum (2012) recommendations, treating psychosocial comorbidities is a priority and supports improved health outcomes and quality of life. Attendees will be prepared to apply this information to meet the comprehensive care needs of patients with epilepsy and their families. In addition, possible interventions to accommodate lack of resources and funding regarding psychosocial comorbidities of epilepsy will also be addressed.

LEARNING OBJECTIVES

Following participation in this session, learners should be able to:

- Delineate and implement strategies to assess and treat psychosocial comorbidities care into practice, to include: networking with other health care providers regarding comprehensive patient care needs, including the primary care provider, other epilepsy specialists and community organizations.
- Recognize and actively participate in the assessment and treatment of psychosocial comorbidities as part of the comprehensive epilepsy team including providing education, resources and counseling to persons with epilepsy and their families.
- Recognize and engage in the interprofessional contribution of pharmacists as part of the comprehensive epilepsy team assessing the patient's medication therapy adherence and treatment needs.
- Recognize the interprofessional contribution of psychologists / neuropsychologists as part of the comprehensive epilepsy team in assessing the psychosocial comorbidities of epilepsy including referrals for and evidence-based therapeutic interventions for patients with epilepsy and their family members.

TARGET AUDIENCE
Basic, Intermediate and Advanced

PROGRAM
Co-Chairs: Gigi Smith, Ph.D., MSN, CPNP, and Janelle Wagner, Ph.D.

Introduction
Janelle Wagner, Ph.D.

Cognitive and Behavioral Comorbidities of Epilepsy Throughout the Lifespan
Jana Jones, Ph.D.

Screening Practices for Psychosocial Comorbidities in Epilepsy: Preliminary Survey Results
Mary Lou Smith, Ph.D., and Rochelle Caplan, M.D.

Pediatric Epilepsy Side Effects Questionnaire and Clinically Meaningful Change
Diego Morita, M.D.

Assessing Psychosocial Comorbidities in Adults with Epilepsy in the Epilepsy Monitoring Unit and Outpatient Clinic
Madona Plueger, MSN, CNRN, ACNS-BC

Conclusion and Faculty Panel
Gigi Smith, Ph.D., MSN, CPNP

EDUCATION CREDIT
2.5 CME credits

Nurses may claim up to 2.5 contact hours for this session.

Pharmacists: AKH Inc., Advancing Knowledge in Healthcare approves this knowledge-based activity for 2.5 contact hours (0.25 CEUs). UAN 0077-9999-16-088-L01-P. Initial Release Date: 12/3/16.

COMMERCIAL SUPPORT ACKNOWLEDGMENT
Supported in part by educational grants from Eisai Inc., UCB, Inc., and Supernus Pharmaceuticals, Inc.

7:00 p.m. - 10:00 p.m.

5th Annual AES Wine Tasting and Silent Auction

Additional fee and ticket required.

Join friends and colleagues for a fun, casual evening that also raises funds for the AES Lennox and Lombroso and Susan S. Spencer Funds. Sample exceptional wines from all over the world paired with choice cheeses and innovative hors d'oeuvres. Plus your participation helps fund early career fellowships and training in basic science and clinical research.

Massa's South Coast Grill
1331 Larmar Street, Suite 114
Houston, TX 77010

New in 2016: Purchase tickets online through the registration system, while supplies last. Space is limited to 200 people. $175 per ticket

Questions? Please visit the registration desk at the Convention Center for up-to-date ticket availability.

Noon – 6:00 p.m.
Poster Session One

Convention Center, Hall A3, B3

Enter in back of Hall A3, behind the 400 aisle.

TRANSLATIONAL RESEARCH

Mechanisms

1.001 Nitro-Oxidative Stress and Neuroinflammation Markers Are Suppressed by 1400W, a Highly Selective Inducible Nitric Oxide Synthase Inhibitor, during Epileptogenesis in the Rat Kainate Model of Temporal Lobe Epilepsy | S. Puttachary, S. Sharma, M. Putra, A. Thippeswamy, T. Thippeswamy

1.002 Long-Term Changes of Gamma Event Functional Connectivity after KA Induced Status Epilepticus | L. Li, J. Almajano, J. Engel, A. Bragin

1.003 Activity Dependent Regulation of Adult Born Neurons in Epilepsy | Z. Lybrand, M. Aktar, S. Ge, J. Hsieh

1.004 Continuous Spike-Waves during Slow-Wave Sleep (CSWS) in a Mouse Model of Focal Cortical Dysplasia (FCD) | Q. Sun, C. Zhou, W. Yang, D. Petrus, C. Zhang

1.005 Neural Progenitor Cells Rotor Ablation Impairs Development but Benefits to Postepileptic Behaviors | L. Chen, M. Wu, J. Kai, L. Zeng

1.006 Optogenetic Induction and Interference of Epileptogenesis | J. Slevin, J. Boychuk, F. Pomerleau, R. Alcala, Y. Ai, G. Greg, S. Bret

1.007 Hippocampal Cytokine Release in Experimental Epileptogenesis - A Longitudinal In Vivo Microdialysis Study | S. Bauer, F. Rosenow, H. Hamer, B. Norwood, L. Costard, K. Siebenbrodt, V. Neubert

1.008 Risk Factors to Develop Post Stroke Epilepsy: Epidemiology and Lesion Mapping | B. Diehl, B. Wandschneider, A. Leff, U. Chaudhary, L. Lemieux, C. Price

1.009 Growth Associated Protein 43 (GAP-43) a Novel Target for the Diagnosis, Treatment and Prevention of Epiletogenesis | A. Nemes, K. Ayasoufi, Z. Ying, I. Najm

1.010 Whole Brain Slice Functional Connectivity Analysis of Interictal Activity | K. Lillis, T. Jacob, K. Staley

1.011 AMPA Receptor Plasticity Initiates Status Epilepticus | S. Joshi, K. Rajasekaran, H. Sun, M. Penmetsa, J. Williamson, J. Kapur

1.012 Fibronectin Modulates Neuroplasticity in Hippocampal Neurons via Alpha5Beta1-Integrins in Epilepsy | X. Wu, M. Muthuchamy, S. Reddy

1.013 Re-Activation of Primed Innate Immune System by Peripheral Infection Augments Post-Traumatic Co-Morbidogenesis and Seizure Susceptibility | Y. Wang, D. Miszczuk, A. Pitkänen

1.014 Identification of a Transcription Factor Controlled Neuronal Transcript Signature Activated Early after Status Epilepticus | A. Becker, N. Surano, J. Xue, K. van Loo, S. Schoch

1.015 Extracellular Ionic Environment Modulates the Spatial Distribution and the Time Course of Ictal-Like Discharges in Rat Adult Neocortical Slices | R. Serafini

1.016 The Impact of MIF Binding and CD74 on the Activation and Expansion of Pro Inflammatory B Cells and Gamma Delta T Cells in a Model of Post-Traumatic Epilepsy | L. Shapiro, S. Rogers, D. Nizamutdinov, R. Bucala, M. Newell-Rogers

1.017 Conversion of Slow-Gated to Fast-Gated BK Potassium Channels Following Seizures | L. Whitmire, V. Bugay, L. Ling, D. Jaffe, J. Cavazos, R. Brenner

1.018 Genome-Wide Long Non-Coding Rna Analysis in Mouse Models of Temporal Lobe Epilepsy | B. Park, J. Moon, J. Lim, J. Jun, T. Yang, K. Kim, S. Lee, K. Park, K. Jung, K. Jung, K. Chu, S. Lee

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1.021 A GRIN2D Mutation in Transmembrane Domain M3 Associated with Severe Epileptic Encephalopathy | H. Yuan, G. Kosobucki, W. Chen, A. Schulien, A. Tankovic, C. Hu, H. Kusumoto, D. Li, X. Ortiz-Gonzalez, E. Marsh, M. Falk, E. Aizeman, S. Traynelis

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1.035 Ripples on Spikes Show Increased Phase-Amplitude Coupling in Mesial Temporal Lobe Epilepsy Seizure Onset Zones | S. Weiss, I. Orosz, S. Moy, W. Linqing, M. Van 't Klooster, R. Knight, R. Harper, A. Bragin, I. Fried, J. Engel, R. Staba

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1.273 Misdiagnosis of Lamotrigine Toxicity as Posterior Circulation TIA or Stroke | P. Ramey, M. Osborn, B. Abou-Khalil

1.274 Cross-Sectional Head-to-Head Comparison of Cognitive Effects of Common Antiepileptic Drugs in Mono- And Polytherapies | C. Helmstaedter, J. Witt

1.275 Prevalence of Fractures in Pediatric Patients on an Antiepileptic Drug | S. DiCarlo, T. Haworth, M. Messinger, B. Moffett, A. Schwabe, A. Wilfong

1.276 Perampanel and Forced EEG Normalization: A Possible Association | A. Russo, E. Spezia, E. Fiumana, A. Boni, T. Messana, M. Filippini, P. Bergonzini, M. Santucci, R. Fagioli, A. Guerra, G. Gobbi

1.277 Predictors and Inhibitors of Weight Loss with Topiramate-Treated Epilepsy | H. Iwaki, M. Fujikawa, S. Kaneko, N. Nakasato

1.278 HLA-B*40:02 and HLA-DRB1*04:03 Alleles Are Genetic Risk Factors for Oxcarbazepine-Induced Maculopapular Eruption | J. Jun, J. Moon, J. Lim, B. Park, T. Yang, K. Kim, K. Park, S. Lee, K. Jung, K. Jung, K. Chu, S. Lee

1.279 Analysis of Immunoglobulin Levels in Epileptic Children Treated with Anti-Epileptic Drugs | G. Yamanaka, N. Morishita, M. Takeshita, S. Morichi, Y. Ishida, T. Mlyajima, H. Kawashima

1.280 Cases of Probable Psychiatric Symptoms Associated with Levetiracetam | K. Hara, T. Maehara, M. Inaji, C. Nagamiri, Y. Sumi, M. Akaza, M. Hara

Other

1.281 Intact Levetiracetam Extended-Release Tablets after Dissolution | D. Sun, H. Wen, A. Externbrink, Z. Gao, D. Keire, G. Krauss, W. Jiang

1.282 Current State of the Union of Epilepsy Care in the United States: Antiepileptic Drugs | J. Sirven, P. Shafer, L. Kalilani, I. Wild, J. Fishman

1.283 Medication Reconciliation Prior to Epilepsy Surgery | M. Messinger, M. Bragdon

1.284 Lacosamide Plasma Concentration and Tolerability during Add-On Compared to Monotherapy by CYP Class of the Background AED: Post-Hoc Analysis of a Conversion to Lacosamide Monotherapy Trial | S. Dimova, Y. Zhang, D. Chellun, M. De Backer, W. Cawello

1.285 Tolerability and Effectiveness of Lacosamide Monotherapy in Patients with Newly Diagnosed Epilepsy and Psychiatric Comorbidities: Post-Hoc Analysis of a Prospective Randomized Double-Blind Trial | B. Schmitz, M. Newton, S. Dimova, Y. Zhang, D. Chellun, M. De Backer, T. Gasalla

1.286 Comparing Long-Term Healthcare Costs Associated with the Use of Enzyme Inducing Antiepileptic Drugs (EIAEDs) and Non-Enzyme Active Antiepileptic Drugs (nEAAEDs) in Elderly Patients | S. Thieffry, S. Borghs, J. Chan, M. Noack-Rink, P. Dedeken, L. Byram, V. Kiri

1.287 Adherence to Antiepileptic Drugs in Patients from José De San Martín Hospital | L. Orellana, M. Rivira, M. Pacha, G. Ernst, I. Lagger, O. Martínez

1.288 Usefulness of Intravenous Levetiracetam for the Treatment of Seizures in Neurological Emergency and Perioperative Periods | T. Yamamoto

1.289 Perampanel in Patients with Refractory and Super-Refractory Status Epilepticus in a Neurological Intensive Care Unit- An Update | A. Rohracher, J. Höfler, G. Kalss, M. Leitunger, G. Kuchukhidze, C. Neuray, J. Dobesberger, E. Trinka

1.290 Antiepileptic Drug Exposure in Infants of Breastfeeding Mothers with Epilepsy | A. Birnbaum, K. Meador, S. Praneeth Bathena, M. Roslawski, R. May, E. Gerard, P. Penovich, L. Kalayjian, N. Velez-Ruiz, J. Cavitt, P. Pennell

SURGERY

Adult

1.291 Infection and Erosion Rates in Trials of a Cranially Implanted Neurostimulator Do Not Increase with Subsequent Neurostimulator Placements | P. Weber, R. Kapur, R. Gwinn, D. Roberts, R. Zimmerman, M. Morrell

1.292 Post-Epilepsy Surgery De Novo Psychogenic Nonepileptic Seizures | A. Asadi-Pooya, M. Asadollahi, J. Tinker, M. Nei, M. Sperling

1.293 Seizure Control and Drug Load Determine Cognitive Development and Recovery 5-22 Years after Epilepsy Surgery | C. Helmstaedter, C. Elger, V. Vogt

1.294 SUDEP Rate in Patients with Medically Intractable Partial Onset Seizures Treated with Brain Responsive Neurostimulation | O. Devinsky, R. Kapur, R. Duckrow, N. Fountain, R. Gwinn, J. Leiphart, A. Murro, P. Van Ness, M. Morrell

1.295 Withdrawn

1.296 Novel Multimodal Surgical Interventions Achieve Engel Class II Outcome in a Highly Refractory Patient with Non-Lesional Frontal Lobe Epilepsy | H. Henninger, V. Thadani, K. Bujarski, D. Roberts, K. Secore, L. Schommer, B. Jobst

1.297 MR-Guided Laser Interstitial Thermal Therapy for Drug-Resistant Mesial Temporal Lobe Epilepsy | J. Tao, S. Wu, N. Issa, S. Rose, A. Ali, J. Jacobsen, V. Tolwe, C. Young, P. Warnke

1.298 Long Term Outcomes of the SANTE Trial: 7-Year Follow-up | E. Sandok, M. Sperling, R. Gross, R. Fisher

Pediatrics

1.299 Ischemia and Inflammation are Involved in the Onset of Epilepsy in Sturge-Weber Syndrome | M. Nakajima, H. Sugano, H. Suzuki, T. Higo, H. Arai

1.300 Endoscopic Minimally Invasive Hemispherotomy and Corpus Callosotomy | S. Sood, E. Asano, A. Luat

1.301 Corpus Callosotomy for Intractable Epilepsy: The Children's Hospital of Michigan Series | A. Luat, E. Asano, H. Chugani, S. Sood

1.302 Corpus Callosotomy: Successful Surgical Treatment in Two Pediatric Patients with Super-Refractory Status Epilepticus | Y. Khan, M. Morrissey, M. Smyth, M. Bertrand

1.303 Trans-Falcine and Sub-Frontal Insertion of Contralateral Subdural and Depth Electrodes in Pediatric Epilepsy Surgery | C. Rozzelle, J. Pindrik, B. Rocque, S. Tubbs

1.304 Adverse Events after VNS Implantation in Children under the Age of 8 Years with Lennox and Lennox-Like Syndrome | C. Cukiert, A. Cukiert, J. Burattini, P. Mariani

1.305 Reoperation after Failed Resective Epilepsy Surgery in Children | O. Muthaffar, C. Chan, L. Rubinger, C. Go, C. Snead III, J. Rutka, E. Widjaja

1.306 Motor Function Mapping with Stereo-Electroencephalography in Young Children | H. Kim, J. Chern

1.307 Can We Prevent Motor Deficits after Resective Epilepsy Surgery in Children? A Novel Paradigm of Electrical Stimulation Mapping | P. Kršek, J. Rybář, P. Ježdík, B. Beňová, A. Jahodová, M. Kudr, V. Komárek, M. Tichý

All Ages

1.308 Long Term Outcome from Stereotactic MRI-Guided Laser Ablation Surgery for Epilepsy: Temporal and Extratemporal Experiece | M. Chez, S. Ciricillo, A. Ghassemi, A. Sekhon, E. Nagy-Wilde, N. Seminario-Lopez, J. Quan

1.309 Therapeutic Outcome of 101 Patients with Sturge-Weber Syndrome and Effective Diagnostic Modalities for Identifying Seizure Severity and Epileptic Zone | H. Sugano, M. Nakajima, H. Suzuki, T. Mitsubishi, H. Arai

1.310 Targeting Technique in Patients Submitted to Hippocampal Deep Brain Stimulation | A. Cukiert, C. Cukiert, J. Burattini, P. Mariani

1.311 Breaking the Age Barrier: First Comprehensive Look at Respective Epilepsy Surgery in Patients 60 Years and Older | V. Punia, A. Abdelkader, R. Busch, J. Gonzalez-Martinez, A. Stojic

1.312 Favorable Outcomes of VNS Therapy after Failed Epilepsy Surgery | F. Arruda, P. Ragazzo, H. van der Linden Jr, S. Melo Souza, J. Arruda, V. Costa

1.313 Cortical Stimulation for Drug-Resistant Focal Epilepsy of Various Etiologies | C. Chang, S. Lim, C. Lee, S. Lee, W. Tseng, H. Li, M. Cheng, H. Hsieh, H. Chiang, B. Chang, C. Lee, T. Wu

1.314 Outcome of Epilepsy Surgery in a Developing Country in a Heterogenous Group of Adults and Children | S. Iyer, J. Mani, P. Gadgil

BEHAVIOR/NEUROPSYCHOLOGY/ LANGUAGE

Adult

1.315 Naming Decline Following Left Temporal Lobectomy: Patient Subjective Report | R. Busch, D. Floden, L. Ferguson

1.316 Evaluation of Nursing Perceptions in Psychogenic Nonepileptic Spells | A. Cramer, J. Bisping

1.317 Factors Associated with Subjective Cognitive Impairment in Adult Epilepsy Clinic Patients | L. Gotterer, Y. Fan, R. Busch, J. Bautista

1.318 Baseline Performance Effects on Memory and Naming Outcome Following Brain Responsive Neurostimulation | D. Loring, R. Kapur, K. Meador, M. Morrell

1.319 Irritability in Korean Epilepsy Patients and Its Predictors | O. Kwon, Y. Kim, S. Park

1.320 Mechanisms of Memory Impairment in Epilepsy Depend on Age at Disease Onset | G. Rayner, G. Jackson, S. Wilson

1.321 Provocative Induction of Psychogenic Nonepileptic Seizure: Effectiveness of Placebo vs. Non-Placebo Techniques | H. Dave, A. Alobaidy, D. Chen

1.322 Factors Contributing to the Development of Anxiety and Depression One Year after Diagnosis in People with Newly Diagnosed Epilepsy | J. Jeon, S. Lee

1.323 How2tell- The Collaborative Development of an Evidence-Based Educational Resource for Self-Disclosure Strategies for People with Epilepsy | N. Elliott, S. Pembroke, M. White, N. Pender, D. Colin, C. Begley, A. Higgins

Pediatrics

1.324 The Moderating Effect of Family Environment on IQ in Pediatric Medically Refractory Epilepsy | K. Puka, M. Smith, E. Widjaja

1.325 Validating the Shortened Quality of Life in Childhood Epilepsy Questionnaire (QOLCE-55) in Children with Drug Resistant Epilepsy | L. Conway, E. Widjaja, M. Ferro, K. Speechley, M. Smith

1.326 Text and Application-Based Adherence Interventions in Adolescents with Epilepsy | A. Modi, K. Mann, L. Urso, B. Hater, J. Peugh

1.327 Postoperative Changes in Auditory and Visual Naming in Children with Lateralized Epilepsy | M. Hamberger, W. MacAllister, W. Seidel, A. Williams, M. Smith

1.328 Long-Term Effects of Maternal Depression on Health-Related Quality of Life in Individuals Diagnosed with Epilepsy in Childhood | M. Ferro, C. Camfield, S. Levin, M. Smith, S. Wiebe, G. Zou, K. Speechley

1.329 Mapping Visuospatial Memory Using a Tablet Computer Program | A. Nishi, T. Kambara, Y. Nakai, H. Matsuura, E. Asano

1.330 Can We Map Cognitive Flexibility Using a Tablet Computer Program? | H. Matsuura, Y. Nakai, T. Kambara, A. Nishi, E. Asano

1.331 Cognitive Functioning among Children with Psychogenic Non-Epileptic Seizures | R. Trobliger, L. Myers, K. Lebeau, M. Lancaman, M. Lancman

1.332 Executive Dysfunction in Children with Rolandic and Temporal Lobe Epilepsy: Distinct Neurocognitive Phenotypes | E. Lima, P. Rzezak, M. Montenegro, M. Guerreiro, K. Valente

1.333 Cognitive Change in Children after Frontal Lobe Resection | L. Ferguson, J. Haut, D. Floden, P. Klaas, J. Gonzalez-Martinez, R. Busch

GENETICS

Human Studies

1.334 Novel Genetic Variants in PNKP Gene in a Small Cohort of Pediatric Epilepsy Patients | C. Trandafir, K. Bowers, I. Butler, G. VonAllmen

1.335 Frequency of Mosaicism in Parents of Children with Epileptic Encephalopathies | C. Myers, Z. Thuesmunn, A. Muir, G. Hollingsworth, A. Schneider, G. Carvill, L. Sadleir, I. Scheffer, H. Mefford

1.336 Expanding the Phenotype of CACNA1H Mutations | H. Osso-Rivera, N. Chourasia, M. Koenig, G. Von Allmen

1.337 Clinical Utility of Genetic Testing in Adults with Epilepsy: Pilot Experience of the Baylor Genetic Epilepsy Clinic | D. Marafie, J. Rosenfeld, P. Van Ness, P. Zohrevand, Z. Haneef, D. Chen, V. Pacheco, H. Robinson, J. Drabek, A. Goldman

1.338 Expanding the Etiologies for Epilepsy, Autism and Intellectual Disabilities- The Role of mTOR Mutations | E. Bebin, K. Bowling, S. Hiatt, M. Amaral, M. Thompson, C. Finnila, D. Gray, J. Whittle, W. Kelley, K. East, K. Brothers, N. Lamb, S. Simmons, G. Barsh, R. Myers, E. Lose, G. Cooper

1.339 Frequency of CNKSR2 Mutation in X-Linked Epilepsy-Aphasia Syndrome | M. Hildebrand, J. Damiano, R. Burgess, S. Kivity, T. Lerman-Sagie, I. Scheffer, S. Berkovic

1.340 The Role of Monoamine Oxidase a Genetic Polymorphisms in Temporal Lobe Epilepsy Caused by Hippocampal Sclerosis | S. Vincentiis, J. Alcantara, P. Rzezak, D. Kerr, W. Gattaz, H. van der Linden Jr, B. dos Santos, S. Melo-Souza, F. Arruda, P. Ragazzo, T. Chaim, M. Serpa, F. Fernandes, R. Moreno, G. Busatto Filho, R. Alessi, R. Demarque, K. Valente

1.341 Two Definite Sudden Unexpected Deaths in Epilepsy in a Family with a DEPDC5 Mutation | F. Nascimento, F. Borlot, P. Cossette, B. Minassian, D. Andrade

1.342 The Role of Serotonin Transporter Genetic Polymorphisms in Temporal Lobe Epilepsy Caused by Hippocampal Sclerosis | J. Alcantara, S. Vincentiis, P. Rzezak, D. Kerr, W. Gattaz, H. van der Linden Jr, B. dos Santos, S. Melo-Souza, F. Arruda, P. Ragazzo, T. Chaim, M. Serpa, F. Fernandes, R. Moreno, G. Busatto Filho, R. Alessi, R. Demarque, K. Valente

HEALTH SERVICES

Delivery of Care

1.343 Epilepsy Nurse Practitioners Deliver High Quality Care to Patients with Epilepsy | C. Hill, B. Frasch, K. Sansalone, K. Davis, B. Litt, N. Dahodwala

1.344 Improving Delay in Presurgical Evaluation of Epilepsy Patients | C. Hill, J. Raab, L. Ferraro, V. Klinov, S. Krish, D. Roberts, K. Davis, J. Pollard

1.345 Adherence to Recommended 2014 Epilepsy Quality Measures in Epilepsy and Neurology Clinics | S. Ahadi, G. Chari, K. Arya

1.346 Assessment and Management of Epilepsy in Children and Young People with Neurodisabilities Attending Special Schools: Preliminary Results from a Retrospective Audit | K. Kallambella, K. Martin, W. Whitehouse

1.347 Seizure Action Plans for Pediatric Epilepsy Patients: A Randomized Controlled Trial | D. Albert, B. Haridas, J. Cole, P. Glynn, M. Fults, J. Moreland, P. Moreland, A. Patel

CASE STUDIES

1.348 Reflex Seizures Triggered by Diaper Change in Dravet Syndrome | A. Subki, A. Alasmari, F. Jan, F. Moria, M. Jan

1.349 A Case Study of the Emotional Sequelae of Pediatric Drug-Rash-with-Eosinophilia-and-Systemic-Symptoms (DRESS) Syndrome on the Family | J. McGinley, E. Yozawitz, S. Escalante, K. Ballaban-Gil, S. Moshé

1.350 Felbamate-Induced Dyskinesias | M. Molina, M. Holmes, D. Anbarasan

1.351 Seven Cases of New Onset Refractory Status Epilepticus NORSE in Qatar Improved Outcome with Early Immunotherapy | F. Ibrahim, G. Melikyan, B. Mesraoua, H. AL hail, N. Azar, N. Haddad, D. Deleu

1.352 Possible Association between Heavy Consumption of Energy Drinks and Status Epilepticus: A Case Report | N. Haddad, H. Alhussein, H. AL hail, B. Uthman

1.353 Multifocal Epilepsia Partialis Continua, Type 1 Diabetes and Alopecia Universalis: An Atypical Autoimmune Syndrome | D. Carvalho, J. Britton, A. McKeon

1.354 Automated Epileptic Seizure Detection Using Accelerometry, Heart Rate and Electromyogram | T. De Cooman, A. Van de Vel, B. Ceulemans, L. Lagae, S. Van Huffel

1.355 Retinal Structure and Function during Adjunctive Vigabatrin Treatment: Case Reports of Potentially Significant Changes during a Phase IV Study | K. Laxer, R. Ramsay, J. Slater, E. Kutluay, C. Johnson, R. Sergott

1.356 Epilepsy of Infancy with Migrating Focal Seizures: Case Report of a Novel SCN2A Mutation and Effective Therapy with Lacosamide | E. Carter, J. Brown, T. Koch, M. Moustafa

1.357 Localization of Epileptinogenic Zone Facilitated by Stimulation of Intracranial Leads | G. Lai, W. Gao

1.358 Epileptic Encephalopathy Associated with Chronic Granulomatous Herpes Simplex Encephalitis | B. Taskin, K. Tanji, N. Feldstein, C. Akman

1.359 Frequent Sleep-Related Bitemporal Focal Seizures in Transient Epileptic Amnesia Syndrome: Evidence from Ictal Video-EEG | A. Jones, D. Burkholder, D. Jones, R. Fabris, J. Britton, T. Lagerlund, E. So, G. Cascino, G. Worrell, C. Shin, E. St. Louis

8:00 a.m. – 8:30 a.m.

AES Business Meeting

Open to all AES members.

Hilton, Ballroom of the Americas A, Level Two

Hometown hospitality supported by Texas Children's Hospital

8:00 a.m. – 5:00 p.m.

Scientific Exhibits

Convention Center

See page 21

8:45 a.m. – 4:45 p.m.

Investigators Workshops

These workshops highlight exciting developments in basic, translational and clinical epilepsy research and are designed to encourage interactive discussion about challenges and opportunities for future advances. Speakers include established and junior epilepsy investigators, as well as researchers from other fields with expertise that may be applied to epilepsy. In addition, one workshop will feature presentations by junior investigators in cutting-edge areas of research.

Most Investigators Workshops will run as concurrent sessions on Sunday, with one workshop on Saturday afternoon and two workshops on Monday afternoon. A poster session will accompany the workshops on Sunday, with posters selected from among highly-ranked Annual Meeting abstracts.

TARGET AUDIENCE

Neurologists, neuroscientists, pharmacologists, neuropsychologists, neurosurgeons and other scientists, professionals and trainees who are performing research in epilepsy.

PROGRAM

Investigators Workshop Committee Chair: Viji Santhakumar, Ph.D.

Clinical Investigators Workshop Committee Chair: Edward J. Novotny Jr., M.D.

Morning Session I: 8:45 a.m. – 10:15 a.m.

1. Epilepsy - A Tauopathy?
   Convention Center, Room 310 B
   Moderator: Matthias Koepp, M.D., Ph.D.
   Speakers: Xin-You Tai, M.D., Jeffrey Noebels, M.D., Ph.D., and Christophe Bernard, Ph.D.
2. Role of Aberrant Neurogenesis in Epileptogenesis
   Convention Center, Room 320 B
   Moderator: Jenny Hsieh, Ph.D
   Speakers: Helen Scharfman, Ph.D., Kyung-Ok Cho, M.D., Ph.D., and Steve Danzer, Ph.D.
3. Autistic Traits in Epilepsy Models: Why, When and How?
   Convention Center, Ballroom A
   Moderator: Jana Veliskova, M.D., Ph.D.
   Speakers: Melissa Benson, Ph.D., Jill L. Silverman, Ph.D., and Pierre-Pascal Lenck-Santini, Ph.D.

Morning Session II: 10:30 a.m. – Noon

4. Electrocorticographic Language Mapping in Epilepsy Surgery: From Cortical Stimulation to Real-time Dynamic Networks
   Convention Center, Room 310 B
   Moderator: Ravindra Arya, M.D., D.M.
   Speakers: Ravindra Arya, M.D., D.M., Nathan Crone, M.D., and Prasanna Jayakar, M.D., Ph.D.
5. Peripheral and Imaging Biomarkers in Epilepsy
   Convention Center, Room 320 B
   Moderators: Manisha Patel, Ph.D., and Stefanie Dedeurwaerdere, Ph.D.
   Speakers: Stefanie Dedeurwaerdere, Ph.D., Svenja Heischmann, Ph.D., and William Theodore, M.D.
6. Glial Mechanisms of Epilepsy
   Convention Center, Ballroom A
   Moderator: Long-Jun Wu, Ph.D.
   Speakers: Ukpong Eyo, Ph.D., Devin Binder, M.D., Ph.D., and Karen Wilcox, Ph.D.

Noon – 1:30 p.m.

Investigators Workshops Poster Session

Includes Lunch

Convention Center, Ballroom Prefunction, Between Rooms 310 and 320

Note: Number below refers to poster assignment

1.002 Long-Term Changes of Gamma Event Functional Connectivity after KA Induced Status Epilepticus | Lin Li

1.003 Activity Dependent Regulation of Adult Born Neurons in Epilepsy | Zane Lybrand

1.008 Risk Factors to Develop Post Stroke Epilepsy: Epidemiology and Lesion Mapping | Beate Diehl

1.009 Growth Associated Protein 43 (GAP-43) a Novel Target for the Diagnosis, Treatment and Prevention of Epiletogenesis | Ashley Nemes

1.011 AMPA Receptor Plasticity Initiates Status Epilepticus | Suchitra Joshi

1.017 Conversion of Slow-Gated to Fast-Gated BK Potassium Channels Following Seizures | Luke Whitmire

1.028 Development of a Rapid Functional Assay That Predicts GLUT1 Diesease Severity | Sasha Maria Zaman

1.031 Developmentally Regulated Alternative Splicing Potentiates Dysfunction of Ohtahara Syndrome-Associated SCN2A Variants | Christopher Thompson

1.033 Malfunction of β-Catenin Pathways Leads to Infantile Spasms and Seizures | Antonella Pirone

1.035 Ripples on Spikes Show Increased Phase-Amplitude Coupling in Mesial Temporal Lobe Epilepsy Seizure Onset Zones | Shennan Weiss

1.036 Circadian Regulation of High Frequency Oscillations (HFOs): Divergent Behavior of Physiological Versus Pathological HFOs | Jean Gotman

1.037 Gamma Activity within Human Epileptic and Non-Epileptic Brain during Cognitive Stimulation | Fatemeh Khadjevand

1.056 Ndel1 Conditional Knockout Mice Exhibit Morphofunctional Hippocampal Alterations and Spontaneous Recurrent Seizures | Cezar Gavrilovici

1.057 Cortical Interneuron Differentiation from hPSCs for Modeling of Epileptic Encephalopathies | Kesavan Meganathan

1.058 Development and Pharmacologic Characterization of the Rat 6 Hz Model | Cameron Metcalf

1.059 Spontaneous Seizures and Behavioral Abnormalities in a Novel Open-Access Inducible Mouse Model of Dravet Syndrome | Ana Mingorance

1.065 Effects of Genetic Elimination of Serotonin Neurons on Seizure Susceptibility and the Cardio-Respiratory Consequences of Seizures in Two Mouse Models of Epilepsy Are Vigilance State Dependent | Gordon Buchanan

1.072 Predicting the Variability of Seizure Frequency: The Pathway to Precision | Daniel Goldenholz

1.078 Induction of Epileptiform Activity and Effects of Anti-Epilepsy Drugs in Cultured Human Induced Pluripotent Stem Cell-Derived Cortical Neuronal Networks | Aoi Odawara

1.080 Chemogenetic Silencing of Excitatory Hippocampal Neurons Prevents Spontaneous Seizures in a Mouse Model for Temporal Lobe Epilepsy | Robrecht Raedt

1.139 Silencing of Nigrotectal Projections Is Sufficient to Recapitulate the Anti-Seizure Effects of Substantia Nigra Inactivation in Diverse Experimental Models of Seizures | Evan Wicker

1.145 Distinct Inhibitory Regulation of Dentate Granule Cells and Semilunar Granule Cells | Milad Afrasiabi

1.146 In Vivo Interneuron Circuit Dysfunction in Chronically Epileptic Mice | Tristan Shuman

1.223 Multimodal Connectome Organization across the Spectrum of Cortical Malformations | Seok-Jun Hong

1.262 Tackling Epileptogenesis via the mGlu7 Glutamate Receptor | Benoit Girard

2.288 The Role of Non-Coding Variation in the Pathogenesis of Epileptic Encephalopathy | Gemma Carvill

3.006 Loss of Function of the Circadian Molecular Clock Underlies Hyper-Excitability in Focal Epilepsy | Judy Liu

3.018 Glycolytic Inhibition with 2-Deoxyglucose Attenuates Epileptiform Activity following Traumatic Brain Injury | Jenny Koenig

3.024 GATOR 1 Subunit Knockdown Produces mTOR-Dependent Changes in Cellular Morphology and Function | Philip Iffland

3.027 Models and Mechanisms of SPTAN1 Epileptic Encephalopathy | Yu Wang

3.029 Novel Immunological Mechanisms of Dravet Syndrome Identified in a SCN1A Knock-Out Mouse Model | Dan Xu

3.034 Diffusion Tensor Imaging: A Non-Invasive Surrogate Marker of Intracranial High Frequency Oscillations | Iren Orosz

3.038 Stabilized Step Function Opsins Switch Firing Mode of Ventral Basal Thalamic Neurons to Abort Non-Convulsive Seizures at Their Onset in Dravet Syndrome | Stefanie Makinson

3.057 Computational Models of Ictogenesis: Synaptic Depression, Recovery, and Connectivity | Theju Jacob

3.058 Crowdsourcing Reproducible Seizure Detection | Steven Baldassano

3.062 Spike-Wave-Discharges (SWDs) Do Not Reflect Absence Epilepsy in Healthy Awake Behaving Rats: Awareness and Voluntary Control of Epileptiform SWDs | Daniel Barth

3.063 Changes in Cardiac K+ Currents in a Mouse Model of Scn1B-Linked Dravet Syndrome | Chad Frasier

3.067 A Human Neuronal Model for Tuberous Sclerosis | John Blair

3.080 Modeling Epilepsy Syndromes Caused by SCN1B Mutations Using Human Induced Pluripotent Stem Cells | Helen Zhang

3.132 Asynchronous Suppression of Superficial Cortex during Absence Seizures | Jochen Meyer

Afternoon Session I: 1:30 p.m. – 3:00 p.m.

7. **Natural Fluctuations of Epilepsy: Lessons from a Million Seizures**
   Convention Center, Room 310 B
   Moderator: William Theodore, M.D.
   Speakers: Daniel Goldenholz, M.D.,Ph.D., Tobias Loddenkemper, M.D., and Victor Ferastraoaru, M.D.

8. **Novel Immunomodulatory Therapies in Epilepsy**
   Convention Center, Room 320 B
   Moderators: Dan Xu, Ph.D., and Sooky Koh, M.D., Ph.D.
   Speakers: Dan Xu, Ph.D., Eleonora Aronica, M.D., Ph.D., and Teresa Ravizza, Ph.D.

9. **Hot Topics from Young Investigators in the Epilepsy Community**
Convention Center, Ballroom A
Moderators: Viji Santhakumar, Ph.D., and Sydney Cash, M.D., Ph.D.

   Activity Dependent Regulation of Adult Born Neurons in Epilepsy
   Speaker: Zane R. Lybrand, Ph.D.

   In Vivo Interneuron Circuit Dysfunction in Chronically Epileptic Mice
   Speaker: Tristan Shuman, Ph.D.

   Multimodal Connectome Organization across the Spectrum of Cortical Malformations
   Speaker: Seok-Jun Hong, Ph.D.

   Hunting for the Genetic Cause in SCN1A Mutation Negative Dravet Syndrome Patients
   Speaker: Alison M. Muir, Ph.D.

Afternoon Session II: 3:15 p.m. – 4:45 p.m.

10. **Neurovascular Unit in Seizures and Epilepsy**
Convention Center, Room 310 B
Moderators: Devin Binder, M.D., Ph.D., and Viji Santhakumar, Ph.D.
Speakers: Nicola Marchi, Ph.D., Todd Fiacco, Ph.D., and G. Campbell Teskey, Ph.D.

11. **Emerging Strategies Using Stem Cells to Prevent Epilepsy**
Convention Center, Room 320 B
Moderator: Helen Scharfman, Ph.D.
Speakers: Jenny Hsieh, Ph.D., Jack Parent, M.D., and Robert Hunt, Ph.D.

12. **Seizure Termination: Multiple Mechanisms**
Convention Center, Ballroom A
Moderators: Peter Carlen, M.D., and Marco de Curtis, M.D.
Speakers: Marco de Curtis, M.D., Peter Carlen, M.D., and Paolo Bazzigaluppi, Ph.D.

8:45 a.m. – 5:15 p.m.

Annual Course | When All Else Fails: Intractable Epilepsy — Pathophysiology to Treatment

Convention Center, General Assembly

OVERVIEW

This year's Annual Course will focus on the evaluation and management of the "worst of the worst," super-refractory patients with epilepsy. Four cases will be presented throughout the day which span the age spectrum from infancy to old age. In addition to case-based scenarios, lectures, debates and counterpoints, plus questions and answers will be employed regarding the recommended approaches to diagnosing and treating these challenging cases. The morning session will start with the case of an infant with epileptic encephalopathy, followed by lectures on neuroimaging beyond MRI, genetic etiologies for children and the value of palliative surgeries. A case of a teenager with seizures from autoimmune disease will then be used to discuss autoimmune etiologies, how to treat, dietary management for adolescents and adults and then a counterpoint on the transition of care from pediatric to adult providers. After the lunch break, there will be a case of an adult with progressive myoclonic epilepsy, followed by lectures on genetic conditions for adults, neuropsychiatric strategies to improve quality of life, new drugs in the pipeline and a debate regarding the true value of the newest anticonvulsant drugs. A case of an elderly patient who has failed surgeries, followed by lectures on the causes of surgical failure, use of herbs and botanicals and a closing debate on when in the course of treatment care should be more palliative.

LEARNING OBJECTIVES

Following participation in this session, learners should be able to:

- Delineate the appropriate role of genetic testing and advanced neuroimaging in diagnosing refractory epilepsy.
- Discuss the impact of severe epilepsy on the patient's family.
- Recognize when immunotherapy and dietary management would be appropriate treatments for refractory epilepsy.
- Describe "best practices" for transitioning pediatric patients to adulthood.
- Select the appropriate anti-epileptic medication(s) and herbs for the treatment of refractory epilepsy.
- Delineate the causes of surgical failure.
- Compare and contrast the newest anti-epileptic drugs (AEDs) available and in the pipeline with standard AEDs in terms of efficacy, side effects and cost.
- Restate whether or not these newer AEDs are truly changing the percent of patients in the refractory category.
- Discuss strategies neuropsychologists can use to improve the quality of life for patients with refractory epilepsy.

TARGET AUDIENCE

Intermediate and Advanced

PROGRAM

Chair: Eric Kossoff, M.D.

8:45 a.m. **Introduction**
Eric Kossoff, M.D.

8:55 a.m. **Case One: Infant with Epileptic Encephalopathy (Ohtahara) Due to Multiple Cortical Malformations**
Jack Lin, M.D.

9:00 a.m. **Lecture: Diagnostic Neuroimaging Beyond MRI**
Fernando Cendes, M.D., Ph.D.

9:25 a.m. **Lecture: Genetic Causes of Severe Refractory Epilepsy**
Annapurna Poduri, M.D.

9:50 a.m. **Lecture: The Role of Palliative Surgery in 2016: Callostomy, MST**
Howard Weiner, M.D.

10:05 a.m. **Lecture: Impact on the Family: What Topics Should Be Discussed and When (Prognosis, SUDEP, Siblings, etc)?**
Katherine Junger, Ph.D.

10:20 a.m. **Break**

10:35 a.m. **Case Two: Seventeen-Year-Old with Refractory Seizures from Autoimmune Encephalitis**
Robert Bollo, M.D.

10:40 a.m. **Lecture: Algorithm for Evaluating Patients for Presumed Autoimmune Disease**
Nicolas Gaspard, M.D., Ph.D.

11:00 a.m. **Lecture: Immunotherapy for the Refractory Patient**
Eric Lancaster, M.D.

11:20 a.m. **Lecture: Diets for Older Adolescents and Adults with Highly Refractory Seizures**
Mackenzie Cervenka, M.D.

11:40 a.m. **Counterpoint: Transition of the Adolescent with Severe Epilepsy to Adult Providers — How Best to Accomplish This**
Sarah Kelley, M.D. (Pediatrics), and Elizabeth Felton, M.D., Ph.D. (Adult)

Noon–2:00 p.m. **Lunch**

2:00 p.m. **Case Three: Adult with Progressive Myoclonic Epilepsy**
David Ficker, M.D.

2:05 p.m. **Lecture: Important Genetic Conditions to Consider in Adults**
David Goldstein, Ph.D.

2:25 p.m. **Lecture: Beyond Epilepsy — Neuropsychiatric Strategies to Improve Quality of Life**
Jay Salpekar, M.D.

2:45 p.m. **Lecture: Exciting New Drugs in the Pipeline**
Steve White, Ph.D.

3:05 p.m. **Debate: The Newer Drugs Are Changing the Landscape and Helping These Patients**
Daniel Friedman, M.D.

These New Drugs Have NOT Improved the Outcomes for Refractory Patients
Michael Rogawski, M.D., Ph.D.

3:25 p.m. **Break**

3:40 p.m. **Case Four: Elderly Adult Who Has Failed Multiple Surgeries: Johnny Returns**
Michael Gelfand, M.D., Ph.D.

3:45 p.m. **Lecture: Anticipating Surgical Failures**
Elaine Wyllie, M.D.

4:05 p.m. **Lecture: Herbs and Botanicals — Can They Help?**
Dana Ekstein, M.D.

4:25 p.m. **Debate: There Is Always Hope! Keep Trying!**
Frank Gilliam, M.D., M.P.H.

There Are Times to Be Helpful, but Times to Stop
Lara Jehi, M.D.

4:55 p.m. **Course Wrap-up**
Eric Kossoff, M.D.

EDUCATION CREDIT

6.0 CME Credits

Nurses may claim up to 6.0 contact hours for this session.

Pharmacists: AKH Inc., Advancing Knowledge in Healthcare approves this knowledge-based activity for 6.0 contact hours (0.6 CEUs). UAN 0077-9999-16-089-L01-P. Initial Release Date: 12/4/16.

COMMERCIAL SUPPORT ACKNOWLEDGEMENT

Supported in part by educational grants from Eisai Inc. and GW Pharmaceuticals.

10:00 a.m. - 4:00 p.m.
Exhibit Hall
Convention Center, Hall A3, B3

10:00 a.m. - 4:00 p.m.
Poster Session Two
Convention Center, Hall A3, B3
Enter in back of Hall A3, behind the 400 aisle.
See Pages 72-82.

Convention Center, Room 361 A, D

Coordinators: Rima Nabbout, M.D., Ph.D., and Elaine Wirrell, M.D.

Speakers: Michael Duchowny, M.D., Prakash Kotagal, M.D., Prasanna Jayakar, M.D., Ph.D., Mary Connolly, M.D., William Bingaman, M.D., Nathalie Baddaent, Mai-Lin Ho, Kees Braun, M.D., Ph.D., and William Gaillard, M.D.

This SIG will focus on three – four challenging cases of children with intractable epilepsy due to MCD. Each presenter will present relevant history and physical findings, along with relevant imaging. An expert panel consisting of Pediatric Neurosurgery, Pediatric Neuroradiology and Pediatric Epileptology will discuss each case, focusing on imaging, neurophysiologic and/or surgical techniques that would assist with each case, along with input from the audience. Proposed cases are: 1) intractable epilepsy with overlapping or very near to eloquent cortex; 2) infantile spasms, with evidence of focal features clinically and/or on EEG but negative MRI; focus will be on investigation of young child less than two years old; 3) tuberous sclerosis; and 4) frontal lobe epilepsy with negative structural MRI with focus on investigation of older child with negative structural MRI.

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Convention Center, Room 310 B

Coordinators: Gaston Baslet, M.D., and Jana E. Jones, Ph.D.

Speakers: Madison Berl, Ph.D., Alan Ettinger, M.D., Tatiana Falcone, M.D., Sarah Wilson, Ph.D.

Identification and management of psychosocial comorbidities in epilepsy can be challenging due to several factors. These factors include, but are not limited to, understanding psychiatric symptoms in the context of seizure occurrence, potential impact of psychiatric treatment on seizure control, implications of comorbidities on seizure treatment outcome and on overall functioning. This year's panel of experts will discuss cases that highlight these challenges and how to navigate complex scenarios to achieve the best possible outcome. The discussion will involve adult and pediatric cases and comorbidities will include psychiatric/behavioral and cognitive presentations.

Quality, Value and Safety in Epilepsy | Quality Improvement Projects to Improve Epilepsy Care: Why, When and How

Convention Center, Room 310 A

Coordinators: Katherine Noe, M.D., Ph.D., and Gabriel Martz, M.D.

Speakers: Ashan Moosa Naduvil, M.D., Richard Zimmerman, M.D., and Jennifer Disabato, DNP, CPNP-PC, AC

The focus of this year's SIG will be to provide a framework on how quality improvement projects are conceived, designed and executed. The target audience will be both the novice who may be considering undertaking a QIP in their epilepsy practice as well as the more experienced researcher. The session will start with a brief overview/primer of the QIP from Richard Zimmerman, M.D., epilepsy neurosurgeon and leader of quality management services at Mayo Clinic Arizona. This will be followed by two invited speakers who will discuss their own QIP from conception to publication. Ahsan Moosa Naduvil, M.D., pediatric epileptologist and quality improvement officer at the Cleveland Clinic will present his experience with improving the seizure interview process in the pediatric epilepsy monitoring unit via a guided team based approach. Jennifer Disabato, DNP, CPNP-PC, AC, from Children's Hospital Colorado will discuss a QI project to address the transition from pediatric to adult care for adolescents and young adults with refractory epilepsy. There will be time for audience members to ask questions and discuss their own QIPs.

Sleep and Epilepsy | Sleep Disorders and Their Relation to Epilepsy: Updates on Insomnia, Sleep Apnea and Movement Disorders

Convention Center, Room 320 A

Coordinators: Milena Pavlova, M.D., and Erik St. Louis, M.D.

Speakers: Milena Pavlova, M.D., Erik St Louis, M.D., and Veronique Latreille, Ph.D.

Within the last five years, there have been some major developments in understanding the relationship between sleep disorders and epilepsy. In addition to new evidence on how impaired sleep may exacerbate epilepsy, there have been several new methods of treatment for these disorders. For example, there have been several publications addressing the frequency of insomnia complaints among epilepsy patients, a new medication available for treatment of insomnia, more thorough understanding of the risk of worsening epilepsy among patients with untreated sleep apnea, new methods of treatment of obstructive sleep apnea and a better understanding of the relationships of nocturnal movements disorders and epilepsy. Based on these developments, the SIG will focus on the relationship of major sleep disorders and epilepsy. The SIG will start with a 10 minute introduction by the chair. Subsequently, there will be the following presentations: 1) Insomnia and Epilepsy – Milena Pavlova, M.D.; 2) Obstructive Sleep Apnea and Epilepsy – Milena Pavlova, M.D., and Veronique Latreille, Ph.D.; and 3) Movement Disorders and Parasomnias – Erik St Louis, M.D.

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Convention Center, Room 371 A, D

Coordinators: Jerome Engel, Jr., M.D., Ph.D., and Jean Gotman, Ph.D.

Speakers: Eishi Asano, M.D., Ph.D., M.S., Maeike Zijlmans, M.D., Ph.D., and Shennan Weiss, M.D.

Increasing evidence indicates that specific aspects of high frequency oscillations during long term invasive monitoring and intraoperative ECoG can be used to determine the boundaries of the epileptogenic region. Noninvasive approaches to recording high frequency oscillations are also being developed. What is now needed to apply these advances to standard approaches to resective surgery for epilepsy?

10:00 a.m. - 4:00 p.m.
Poster Session Two

Convention Center, Hall A3, B3
Enter in back of Hall A3, behind the 400 aisle.

NEUROPHYSIOLOGY

Video EEG Monitoring

2.001 Intracranial Markers of Loss of Consciousness in Frontal Lobe Seizures | R. Gebre, M. Dhakar, E. Grover, I. Quraishi, E. Sternberg, I. George, A. Sivaraju, J. Bonito, H. Zaveri, L. Gober, S. Ahammad, S. Ghoshal, P. Farooque, L. Hirsch, D. Spencer, J. Gerrard, H. Blumenfeld

2.002 Prospective Serial EEG Study in Infants with Tuberous Sclerosis Complex (TSC) | G. de Bruyn, B. Verhelle, J. Vervisch, D. Domanska-Pakiela, E. Aronica, P. Curatolo, A. Jansen, F. Jansen, S. Jozwiak, K. Kotulska-Jóźwiak, D. Kwiatkowski, L. Lagae

2.003 Utility of Long-Term Video EEG in Evaluating Pediatric Patients with Autoimmune Epilepsy | S. Suwannachote, W. Gaillard, E. Wells, I. Kahn, J. Schreiber

2.004 Mirror Image Video Artifact: An Under-Reported Video-EEG Artifact | A. Bhatt, M. Babcock, W. Levis

2.005 Triphasic Ictal Scalp EEG Pattern in Interhemispheric Onset Seizures | M. Nishimura, T. Okanishi, H. Motoi, T. Yokota, K. Sato, T. Yamazoe, A. Fujimoto, H. Enoki, T. Yamamoto, H. Otsubo

2.006 The EEG Patch: An Update on Feasibility as a Discrete, Wearable, EEG Device for Counting Seizures | M. Lehmkuhle, M. Elwood, J. Wheeler, J. Fisher, F. Dudek, K. Lervik, L. Frey, A. Shrestha, C. Drees, M. Brown, P. Korb, M. Spitz

2.007 The Diagnostic Yield of Epilepsy Monitoring Unit Evaluation for Veterans with Apparent Syncope of Unknown Origin | C. Saipetch, D. Chen

2.008 Hormonally Inactive Paraganglioma Presenting as Spells | D. Kenney-Jung, F. Anteneh, C. Shin, E. So

2.009 Intravenous Midazolam as First Line Therapy for Status Epilepticus in a Pediatric Epilepsy Monitoring Unit | L. Whittaker, M. Messinger, T. Baierlipp, K. Frost, M. Fernandez, J. Frontiero, W. Morton, S. Marcion, R. Coorg, A. Wilfong, A. Anderson

2.010 Video-Electroencephalography (vEEG) and Patient Safety: A Systems Change Addressing Missed Events | W. Meghan, K. Ashraf, B. Bush, C. Loik, R. Lesanu, C. Hammontree, D. Giss, S. Glynn

2.011 Predictive Factors of Postictal Generalized EEG Suppression in Patients with Generalized Tonic-Clonic Seizures | K. Jin, Y. Kakisaka, Y. Kitazawa, M. Fujikawa, N. Nakasato

2.012 The Golden Standard Is Only Gilded - Failure of Perioperative Electrical Stimulation Mapping Is Explained by Edge-of-Grid, or Bent-Grid Phenomenon | K. Riley, T. Gaston, H. Barkan

2.013 Left Frontal Seizures Causing Asystole in a Patient with a Left Frontal Meningioma Resection - A Case Report | A. Khawaia, A. Venkatraman, S. Pati, T. Gaston, H. Barkan

2.014 Slow Is the Way to Go - ISA (Infra-Slow Activity) in Scalp EEG Is Possibly Lateralizing and Localizing for Frontal Lobe Epilepsy | H. Barkan

2.015 Increased R Wave Visibility with Lead II ECG during Generalised Tonic Clonic Seizures (GTCS) | L. Allen, S. Toescu, C. Scott, C. McLaughlin, H. Millward, L. Lemieux, B. Diehl

ICU EEG

2.016 Survey of American Clinical Neurophysiology Society (ACNS) Standardized Critical Care Electroencephalography (EEG) Terminology in the Neurocritical Care Unit | C. Atallah, N. Badjatia, J. Pritchard

2.017 Nonconvulsive Status Epilepticus (NCSE) in the Pediatric ICU, At a Tertiary Care Center in Saudi Arabia | S. Siddiqui, D. Al Sowat, B. Stigsby, T. Abalkhail, O. Dabbagh, S. AlYamani, H. Al Dhalaan, A. Chedrawi, M. AlMuhaizea

2.018 Seizure Detection and Time to Treatment in the Neonatal Intensive Care Unit | E. Buraniqi, A. Sansevere, K. Kapur, P. Pearl, T. Loddenkemper

2.019 EEG Reporting in the Pediatric Intensive Care Unit | A. Sansevere, N. Abend, R. Arya, J. Brenton, J. Carpenter, K. Chapman, W. Gaillard, M. Gaínza Lein, T. Glauser, J. Goldstein, H. Goodkin, M. Jackson, K. Kapur, M. Mikati, E. Payne, K. Pearlso, J. Riviello, I. Sánchez Fernández, R. Tasker, D. Tchapyjnikov, A. Topjian, M. Wainwright, A. Wilfong, K. Williams, T. Loddenkemper

2.020 Electroencephalography as a Predictor of Mortality and Short-Term Functional Outcome in Children in Intensive Care Unit | A. Ko, I. Sol, K. Kim, H. Kang, J. Lee, H. Kim, S. Kim

2.021 EEG Abnormalities During Positional Changes in Brain Sagging Syndrome | N. Sotudeh, B. Bensam, S. Heustein, P. Bhalla, D. Ledoux, S. Hwang

2.022 Pediatric Continuous EEG Monitoring-The Predictive Value of EEG Background on Outcome | R. Guerriero, A. Sansevere, I. Sanchez, T. Loddenkemper

2.023 Continuous EEG for Seizures in Pediatric Critical Care: Yield and Efficiency of Identification | A. Sansevere, E. Duncan, M. Libenson, T. Loddenkemper, P. Pearl, R. Tasker

Other Clinical EEG

2.024 An Interaction between Warfarin and Epidiolex, A Case Report | B. Vines, L. Grayson, K. Nichol, E. Bebin, J. Szaflarski

2.025 Paroxysm Duration Helps Differentiate Juvenile Absence Epilepsy from Childhood Absence Epilepsy | U. Seneviratne, M. Cook, W. D'Souza

2.026 Head vs. Whole Body Cooling in Hypoxic Ischemic Encephalopathy: Comparing EEG and MRI Brain Changes | A. Goenka, E. Yozawitz

2.027 Identification of Threshold Concepts in Electroencephalography to Support Learning and Curriculum Development | J. Moeller, T. Fawns

2.028 Unusual Electrographic Findings of Lithium Toxicity | D. Desai, S. Bhalla, A. Palade

2.029 Epileptic Network Characterization for Resective Surgery | H. Keijzer, W. Zweiphenning, E. van Diessen, M. van 't Klooster, M. van Putten, M. Zijlmans

2.030 EEG and Seizures in Ischaemic Stroke Treated with IV Thrombolysis | C. Bentes, H. Martins, A. Peralta, C. Morgado, C. Casimiro, C. Fonseca, R. Geraldes, P. Canhão, T. Pinho e Melo, J. Ferro

2.031 Electroclinical Aspects and Prevalence of Eyelid Myoclonia in Pediatric Generalized Epilepsy | A. Polavarapu, K. Carvalho, D. Hasbani

2.032 Effects of Cannabidiol on the EEG | L. Grayson, R. Singh, Y. Liu, C. Gary, E. Bebin, J. Szaflarski

2.033 Subclinical Electrographic Seizures Revealed by High Resolution Dense Array Scalp EEG Recordings | H. Hasegawa

2.034 Electroencephalography Findings to Differentiate Acute Encephalopathy with Biphasic Seizures and Late Reduced Diffusion (AESD) from Prolonged Febrile Seizures | A. Ohno, A. Okumura, T. Fukasawa, T. Suzuki, Y. Nakamura, M. Miyake, T. Kubota, T. Tsuji, N. Ando, S. Saitoh, J. Natsume

MEG

2.035 Benign Epileptiform Variants in MEG | J. Ebersole, S. Ebersole, J. Camerone

2.036 Cortical Current Density Source Analysis Resolves Ambiguities in EEG/MEG Dipole Modeling | M. Wagner, J. Ebersole

2.037 Comparison of MEG SAMepi and Dipole Interictal Spike Localization with Presurgical Evaluation Results and Area of Surgical Resection | R. Joshua, J. Scott, K. Zaghloul, J. Heiss, A. Zachery, S. Susumu, W. Theodore, S. Inati

2.038 The Evaluation of Perceptive Language Area in Child Cases with Language Impairment Using Magnetoencephalography | H. Yamamoto, H. Shiraishi

2.039 Brain Dynamics during Associative Multimodal Memory: Implications for the Assessment of Memory in Epilepsy | E. Martinez Castillo, T. Kleineschay, M. Korostenskaja, B. James, J. Seo, H. Skinner, P. Chen, M. Westervelt, K. Lee

2.040 Combination of Magnetoencephalography and Voxel-Based Morphometry for the Detection of Focal Cortical Dysplasia Type 2 | S. Rampp, H. Hamer, H. Stefan, M. Buchfelder, K. Rössler, B. Kasper

Brain Stimulation

2.041 Ezio Sciammana, Early Progenitor of Cortical Stimulation Mapping | A. Ritaccio, S. Casciato, G. Schalk, P. Brunner

2.042 Predictive Modelling the Effect of Neurostimulation on Memory Biomarkers in Epileptic Patients | J. Malcolm, M. Connolly, R. Gross, M. Kahana, B. Mahmoudi

2.043 Responsive Neurostimulation (RNS) Artifact- A Novel EEG Finding | S. Mathias, K. Haas, M. Gallagher, A. Arain

2.044 Dual-Site Pontine and Thalamic Neurostimulation to Improve Ictal and Postictal Arousal: Assessment of Its Effect with a Behavioral Task | M. Galardi, J. Xu, E. Musonza, J. Pok, J. Osteen, T. Liao, A. Kundishora, A. Gummadavelli, L. Feng, C. McCafferty, J. Gerrard, M. Laubach, H. Blumenfeld

2.045 Structure-Specific Dynamic Oscillation Profiles during Afterdischarges | J. Kleen, E. Chang

2.046 A Real-Time Sense-and-Stimulate Intracranial System Detects and Slows Impending Movements | B. Moore, A. Aron, N. Tandon

2.047 Hemispheric Differences in Cortical Excitability in Patients with Epilepsy | K. Schiller, B. Gorman, B. Mudigoudar, J. Wheless, F. Boop, A. Papanicolaou, S. Narayana

Animal Studies

2.048 Neuronal and Hemodynamic Determinants of Absence Seizure Severity | C. McCafferty, J. Sampognaro, E. Johnson, W. Islam, P. Vitkovskiy, P. Antwi, N. Smith, A. Morawo, A. Kundishora, J. Osteen, L. Makusha, J. Xu, M. Galardi, H. Blumenfeld

2.049 Investigating Seizure Spread in an Acute Penicillin Rat Model | D. Harris, Y. Liu, L. Bateman, Q. Wang, C. Schevon

2.050 Ganaxolone and Diazepam Administered IV Elicit a Synergistic Anti-Epileptic Response in a Rodent Model of Status Epilepticus | M. Saporito, J. Gruner, J. Tsai, A. Patroneva

2.051 Toll-Like Receptor 4 Signaling Contributes to Excitotoxic Injury of Dentate Somatostatin Interneurons after Brain Injury | Y. Li, A. Korgaonkar, A. Kasnetz, V. Santhakumar

2.052 Autonomic Dysfunction and Increased Arrhythmogenic Potential in Mice Following Status Epilepticus | A. Levine, A. Dao, Y. Lai, H. Born, A. Anderson

2.053 Reduction of Neurodegeneration Process after Changes of Glucose Availability in Hippocampus in Model of Temporal Lobe Epilepsy | I. Melo, Y. dos Santos, A. Pacheco, M. Costa, V. Silva, C. Arroxelas, C. Cavalcante, R. Sabino-Silva, O. Castro

2.054 Interictal Spikes: Hallmark of the Ictal Onset Zone or the Epileptic Network? | H. Ma, E. Baird-Daniel, M. Zhao, J. Liou, T. Schwartz

2.055 Characterization of Seizures and EEG in an Animal Model of Cardiac Arrest and Cardiopulmonary Resuscitation Induced Global Ischemia | Y. Roal, L. Weitzel, D. Sampath, K. Shimizu, A. White, P. Herson

2.056 Cannabidiolic Acid Controls Neural Excitability and Seizure-Like Activity | M. Hosseini Zare, A. Abdulla, K. Akula, J. Ziburkus

Computational Analysis & Modeling of EEG

2.057 Inference on Synaptic Connections in the Generation and Propagation of Seizure Activity Based on Intracranial EEG Recordings | C. Park, Y. Choi, A. Jung, E. Kwon, J. Lee, H. Lee

2.058 Masters and Slaves: A New Perspective on the Epileptogenic Network Dynamics | A. Principe, A. Tauste, G. Sisó, M. Ley, R. Rocamora

2.059 Seizure Detection Algorithm for Seizure Suppression by Closed-Loop Electrical Stimulation at Early Stages of Seizure Formation | F. Manzouri, M. Dümpelmann, A. Brandt, S. Heller, C. Donos, A. Schulze-Bonhage

2.060 Occipito - Frontopolar Connectivity in Panayiotopoulos Syndrome - A Case Report | A. Al-Otaibi, S. Tabassum, T. Al-Otaibi, L. Jad

2.061 Pre-Ictal Regulatory Rhythms Predict Patterns of Ictal Spread | D. Groppe, T. Valiante, C. Honey

2.062 Multilobar Epileptogenic Network Skipping the Primary Motor Cortex in a Subset of Children with Drug-Resistant Epilepsy | Y. Iimura, A. Noda, K. Hoashi, K. Jones, S. Baba, M. Nakajima, K. Kagawa, A. Ochi, Y. Nonoda, E. Asano, T. Akiyama, H. Otsubo

2.063 A Computational Model for EEG Recovery in Postanoxic Encephalopathy | B. Ruijter, J. Hofmeijer, H. Meijer, M. van Putten

2.064 Computational Intracranial EEG Analysis Techniques for Localization of Epileptogenic Zones in Secondary Generalized Epilepsy | A. Ko, S. Youn, H. Kim, J. Lee, S. Kim, C. Im, H. Kang

CLINICAL EPILEPSY

Classification and Syndromes

2.065 Phenotypic Analysis of 303 Multiplex Families with Common Epilepsies | S. Bellows, E. Consortium

2.066 Clinical Core Symptoms Are Rather Homogeneous in Nationwide Survey of Benign Adult Familial Myoclonus Epilepsy in Japan | K. Kobayashi, T. Hitomi, R. Matsumoto, M. Watanabe, R. Takahashi, A. Ikeda

2.067 Seizure Characteristics in Adult Tuberous Sclerosis | D. Bauer, N. Fountain

2.068 Assessment of Prodromes during Video-EEG Monitoring in Patients with Drug-Resistant Epilepsy: A Cohort Study | H. Akçay, E. Oguz Akarsu, N. Bebek, H. Dede, C. Gurses, B. Baykal Baykan, A. Gokyigit

2.069 Infantile Spasms Associated with a Heterozygous R297C Variant in the KCNJ10 Gene | J. Baquero, M. Pietryga, J. Vidaurre

2.070 Assessment of Short Term Inpatient Seizure Diary Accuracy Using the DISCOVER Form | J. Louik, D. Friedman, K. Farrell, S. Meade, J. French

2.071 Heide's Syndrome: Infantile Spasms, Epidermal Nevus, PIK3CA-Related Overgrowth Spectrum | L. Flores-Sarnat

2.072 Status Epilepticus and Histopathological Findings in a Case of Stroke-Like Migraine Attacks after Radiation Therapy (SMART) Syndrome | P. Voinescu, J. Helgager, C. Parres, M. Graham, H. Vaitkevicius, U. De Girolami, R. Cosgrove, R. Sarkis

Clinical Diagnosis

2.073 Pyridoxal 5'-Phosphate Levels in the Cerebrospinal Fluid in Neonatal and Infantile Epilepsy and Its Diagnostic Utility for Antiquitin Deficiency | T. Akiyama, M. Akiyama, H. Yoshinaga, K. Kobayashi

2.074 Seizures Frequency and EEG Findings in Posterior Reversible Encephalopathy Syndrome | H. Elnour, A. Abubakr, H. Uschmann, S. Abdelhafiz

2.075 Biomechanical Analysis of Fencing Response in Cases Sports-Related Traumatic Brain Injury | O. Martinez, I. Lagger, M. Pacha, L. Orellana, C. Calandra, A. Roel, D. Vazquez, G. Ernst

2.076 Misdiagnosis Perseveration Facilitated by the EMR: Nonepileptic Spells " Reverting" to Seizures | J. Boggs, C. O'Donovan, M. Sam, H. Munger-clary, M. Wong, V. Woodard

2.077 Seizures with a Presumed Autoimmune Etiology | M. Tom, S. Pawate

2.078 Diagnostic Testing in Children with Epileptic Encephalopathy Using Targeted Massively Parallel Sequencing | K. Kothur, M. Lorentzos, H. Katherine, G. Sachin, T. Christopher, R. Dale, G. Ho, E. Farnsworth, B. Bennetts, J. Christadoulou, D. Gill

2.079 Epileptic Spasms in Pediatric Post-Traumatic Epilepsy at a Tertiary Referral Center: A 2-Year Follow Up Study | J. Park, H. Chugani

2.080 Diagnostic Yield of Cardiac Tilt Table Test during Epilepsy Monitoring Unit Admission | A. Crepeau, K. Noe, E. Aniles Renova, J. Sirven

2.081 Jevons Syndrome Features in Patients with Juvenile Myoclonic Epilepsy and Photo Paroxysmal Response on EEG | D. Benech, A. Gupta, A. Moosa, R. Burgess, A. Alexopoulos, L. Jehi, E. Pestana Knight

2.082 Under Recognized, Atypical Presentation of Cerebral Folate Deficiency in an Older Child | J. Vidaurre, S. Nunley

2.083 A Novel Gene Mutation Causing Refractory Infantile Spasms: A Case Report | J. Fox, B. Appavu, L. Kristin, R. Jarrar

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2.349 Infantile Spasms in Chromosomal 16p11.2 Abnormalities: Case Report and Review of Literature | E. Kovitch, H. Li, K. Velayudam

2.350 Electroencephalogram and Intrathecal β-Cyclodextrin-Hydroxypropyl in Niemann-Pick Type C: A Biomarker? | S. Seinfeld, K. O'Hara, J. Pellock

2.351 First Case of Refractory Uncinate Seizures Secondary to Post Embolization Perianeurysmal Edema | V. Shah, S. Izadyar

2.352 BRAT1 Mutation Causes Lethal Neonatal Rigidity and Multifocal Seizure Syndrome | K. Fitzgerald, I. Butler

2.353 Paraneoplastic Seizures and Auditory Hallucinations Due to Anti-Ta (anti-Ma2) Negative Testicular Germinoma: A Case Report | K. Hagen, M. McCaskill

2.354 Late Neuroimaging Findings after Cryptogenic New Onset Refractory Status Epilepticus: A Case Report | C. Calandra, M. Pacha, I. Lagger, G. Ernst, L. Orellana, M. Verdaguer, S. Morganti, O. Martinez

2.355 Leptomeningeal Inflammation Masquerading as Complex Partial Status Epilepticus in a Patient with Sturge Weber Syndrome | S. Zahoor, D. Miller, A. Mahajan

2.356 Musical Hallucinations Associated with Left Temporal Hemangioblastoma in Von Hippel-Lindau | J. Montes-Rivera, S. Benchaya, M. Eugene

2.357 Are These Postictal Changes or a Glioma in a Patient Presenting with Focal Status Epilepticus? | H. Hasan, X. Li

2.358 The Effect of Levetiracetam on Inter-Ictal Epileptiform Discharges in a Patient with Genetic Generalized Epilepsy | S. Maturu, B. Assaad

2.359 Electro-Clinical Features in Early Onset Infantile Myoclonic Epilepsy Associated with TBC1D24 Gene Mutations | M. Balestri, M. Trivisano, D. Claps Sepulveda, D. Longo, M. Valeriani, F. Vigevano

2.360 Use of Cannabidiol (CBD) in Refractory Epilepsy at a Level 4 Epilepsy Center: An Open Label Study | S. Maciver, P. Patel, C. Robles

Convention Center, Room 320 A

Coordinators: Vicky Whittemore, Ph.D., Tracy Dixon-Salazar, Ph.D., Mackenzie C. Cervenka, M.D., and Catherine Chu, M.D.

Speakers: Manisha Patel, Ph.D., Cara Long, Ph.D., Julie Milder, Ph.D., Vicky Whittemore, Ph.D., Catherine Chu, M.D., Brandy Fureman, Ph.D., Greg Krauss, M.D., Melanie Huntley, Ph.D., Chris Dulla, Ph.D., Farah Lubin, Ph.D., Susan Masino, Ph.D., Suzan Nadi, Ph.D., Avtar Roopra, Ph.D., Helen Scharfman, Ph.D., and Ivan Soltesz, Ph.D.

This session will highlight funding opportunities for epilepsy research and provide an overview of the peer review process with a mock study section presentation. Funding opportunities for junior investigators will be emphasized, but basic and clinical researchers at all stages are encouraged to attend. Panelists will include representatives from organizations that support epilepsy research nationwide or worldwide and researchers who have served as reviewers for NIH and non-profit organizations. Tips will be shared for investigators submitting applications and for those serving as reviewers, and participants will also receive a handout listing funding opportunities for junior investigators along with eligibility criteria and important deadlines.

Practice Management

Convention Center, Room 310 A

Coordinator: Gregory Barkley, M.D.

The 2016 Practice Management SIG will seek to answer questions on how to run epilepsy practices in 2017 by focusing upon pertinent changes in CMS payment policies which will be announced in the summer and fall. Many of these policies, such as the 2017 CMS Physician Fee Schedule, are slated to be released on November 1, 2016. Others, such as any changes to the 2017 ICD-10-CM codes, will go into effect on October 1, 2016. The proposed rule for inpatient hospital billing, IPPS, was released on April 18, 2016, and will be finalized on August 1, 2016, and go into effect on October 1, 2016. Proposed IPPS CMS changes continue the mandated move from payment for volume to payment for value and include new notification rules on observation patients, decreased payments to Disproportionate Share Hospitals (DSH) for uncompensated care and many changes to hospital Inpatient Quality Reporting Programs. If time permits, new payment models, such as the Specialty-Based Global Payment programs based upon the Oncology Care Model, will be discussed along with how such programs might affect care of patients with epilepsy and seizures.

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Convention Center, Room 370 A, D

Coordinators: Marcelo Lancman, M.D., and Pavel Klein. M.D.

Speakers: Ro Elgavish, M.D., Ph.D., Eric Segal, M.D., Pavel Klein, M.D., and Marcelo Lancman, M.D.

The session will cover the many challenges that epilepsy practitioners in private practice will be facing in 2017. It will also expand on the potential of current collaboration in research among private epilepsy practices and centers. Existing achievements that have resulted from collaborative efforts among private epilepsy centers will be presented and further expansion of these plans will be discussed.

SUDEP | Hot Topics in SUDEP Research

Convention Center, Room 361 A, D

Coordinators: Lisa Bateman, M.D., Gordon Buchanan, M.D., Ph.D., and Daniel Friedman, M.D.

In the past five years, there has been a significant increase in the number of investigators pursuing research into the understanding of SUDEP. As a result, the rate of new discoveries has increased to the point where it may be difficult for many to keep up with the latest research. In this session, we will discuss the state of the art in clinical and basic science SUDEP-related research. The session will begin with an update on the activities of the NINDS sponsored Center for SUDEP Research, including its major research initiatives and early results. Discussion follows on the latest cutting-edge SUDEP-related research with presentations by junior investigators. Speakers and topics will be selected based on research presented at this AES meeting and at the recent Partners Against Mortality in Epilepsy (PAME) Conference. A panel of experts will provide commentary on the presentations and time will be allowed for discussion with audience members.

Tumor-related Epilepsy | Scientific Developments and Their Impact on the Treatment

Convention Center, Room 310 B

Coordinators: Jeffrey Politsky, Ph.D., FRCP(C), and Sandeep Mittal, M.D.

Speakers: Mark Cunningham, Ph.D., Kris Smith, M.D., FACS, Aashit Shah, M.D., Sandeep Mittal, M.D., FRCS(C), and Jeffrey Politsky, M.D., FRCP(C)

In 2016, we will discuss the hottest topics relating to the underlying mechanisms of how tumors cause epilepsy and the current investigational and newly approved treatments target certain pathologic markers. In addition, we will discuss the most recent advances in the diagnostic and therapeutic approach to patients with brain tumors and epilepsy, focusing specifically on the peri-operative stage.

Women with Epilepsy: Catamenial Epilepsy and Menopausal Transition: Blessing or Blight?

Convention Center, Room 320 B

Coordinators: Mona Sazgar, M.D., and Danielle M. Andrade, M.D.

Speakers: Cynthia L. Harden, M.D., Alison M. Pack, M.D., and Mohamad A. Mikati, M.D.

Women with catamenial epilepsy often wonder whether their seizure exacerbation with hormonal fluctuations will resolve with the arrival of menopause. In fact a question often posed to the treating epileptologist is whether pharmacologically or surgically induced menopause will help reduce their seizure severity and improve their quality of life. They consider drastic measures such as hysterectomy and oophorectomy in the hopes of trade off for better seizure control. There are also issues regarding treating the menopausal symptoms with hormonal replacement therapy, as well as osteoporosis and bone loss exacerbated by early menopause and seizure medications. The experts for this exciting SIG will use the latest evidence to tackle challenges faced by women with catamenial epilepsy such as early menopause, hormonal replacement therapy and bone health. They will discuss the complex and multidirectional interaction between sex hormones, seizures and antiepileptic drugs (AEDs) as related to the care of women with catamenial epilepsy facing menopause.

8:00 a.m. – 11:00 a.m.

Scientific Exhibits

Convention Center

See Page 21.

8:00 a.m. – 2:00 p.m.

Poster Session Three

Convention Center, Hall A3, B3

8:00 a.m. - 10:00 a.m.
Enter behind Pavilion A, in Hall B3

10:00 a.m. - 2:00 p.m.
Enter behind the 400 aisle in Hall A3

See Pages 89-98.

8:45 a.m. - noon

Merritt-Putnam Symposium | Multiscale Imaging of Seizures and Epilepsy

Convention Center, General Assembly

Award Presentation: William G. Lennox Award

OVERVIEW

One of the grand goals of neuroscience is to understand how the interplay of structural, chemical and electrical signals in and between cells of brain tissue gives rise to function and disease. There is an emerging arsenal of tools and technologies for obtaining imaging data spanning multiple scales from molecules, cells, assemblies, networks and nervous systems that are poised to impact our understanding of epilepsy and advance therapeutic options for our patients. This session will present the latest evidence in neuroscience of which clinicians need to be aware in order to provide optimal patient care. Strategies for counseling patients and their families regarding the effect of surgery on memory circuits will be discussed.

LEARNING OBJECTIVES

Following participation in this session, learners should be able to:

- Describe the advances in structural and functional imaging and delineate their role in predicting epilepsy surgery outcomes and memory impact.
- Review and discuss the emerging evidence for cellular imaging in focal epilepsy.
- Counsel patients and families regarding the impact of surgery on memory circuits and the association with prognosis.

TARGET AUDIENCE

Basic, Intermediate and Advanced

PROGRAM

Chair: Gregory Worrell, M.D., Ph.D.

Introduction
Gregory Worrell, M.D., Ph.D.

Imaging Cells and Assemblies
Peyman Golshani, M.D.

Technologies for Imaging the Neural Circuits Underlying Seizure
Jin Hyung-Lee, Ph.D

Functional Imaging and Neurotransmitters in Epilepsy
Kate Davis, M.D.

Computational Neuroimaging: From Lesions to Networks
Andrea Bernasconi, M.D.

Functional Imaging of Memory Systems and Epilepsy
Jeffrey Binder M.D.

Conclusions
Gregory Worrell, M.D., Ph.D.

EDUCATION CREDIT
3.0 CME credits

Nurses may claim up to 3.0 contact hours for this session.

Pharmacists: AKH Inc., Advancing Knowledge in Healthcare approves this knowledge-based activity for 3.0 contact hours (0.3 CEUs). UAN 0077-9999-16-090-L01-P. Initial Release Date: 12/5/16.

9:00 a.m. - 10:30 a.m

Special Interest Groups

Cognitive and Behavioral Treatments | Effective Psychobehavioral Treatments in Adults and Children with Epilepsy: Closing the Practice and Research Gaps

Convention Center, Room 320 B

Coordinators: Janelle Wagner, Ph.D., and W. Curt LaFrance, M.D., M.P.H.

Speakers: Rosa Michaelis, M.D., and Avani Modi, Ph.D.

Given the comorbidities associated with epilepsy and the substantial impact epilepsy and its treatments have on health-related quality of life (HRQOL), psychological and behavioral health interventions aimed at enhancing psychological well-being and self-management are essential for persons with epilepsy (PWE). Psychological/behavioral health interventions include a broad range of interventions that use psychological or behavioral techniques designed to improve HRQOL, seizure frequency and severity and psychiatric/psychological comorbidities. A recent Cochrane review was conducted by the ILAE Psychobehavioral Treatment Task Force to assess the effects of such psychological treatments on HRQOL in PWE. ILAE Committee members will present this systematic review of 23 randomized, controlled trials, with a particular focus on intervention components (similarities and differences), strengths and weaknesses of selected outcomes and how these methodological variations make it challenging to compare findings across studies. Speakers will also present therapeutic recommendations and implications for future research design based on the findings of the Cochrane review. Both adult and pediatric interventions will be covered. The SIG format will involve brief presentations with significant time for discussion of and questions regarding findings and future directions.

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Convention Center, Room 361 A, D

Coordinators: Michael Funke, M.D., Ph.D., and Gretchen Von Allmen, M.D.

Speakers: Stefan Rampp, M.D., Pablo Cuesta, Ph.D., and Dario Englot, M.D., Ph.D.

Emerging evidence indicates that brain function and dysfunction actually results from a complex interplay or "network" of different brain areas, and that these networks have considerable plasticity, especially in children. Recently MEG has demonstrated utility in identifying abnormal functional connectivity patterns in the resting-state networks of patients with epilepsy compared to unaffected controls. However, much remains unknown about epileptic networks and their interplay with these resting state networks. Deciphering the epileptic networks themselves using MEG may provide new insights into the pathophysiology of epilepsy, and how these networks may influence treatment and prognosis. This program aims to showcase the most recent advances in this quickly evolving field and stimulate vivid discussion among international experts. Presentations include: Resting-state connectivity analysis: What's special about MEG, and what's unique about epilepsy? (Dario Englot, M.D., Ph.D.); Spike-related epileptic networks: Characterization of the whole brain activity during interictal spike events (Pablo Cuesta, Ph.D.); and, MEG focus localization using connectivity based methods in patients with focal pharmacoresistant epilepsies (Stefan Rampp, M.D.).

Pediatric Epilepsy Case Discussions | Pediatric Epilepsy Case Discussions

Convention Center, Ballroom A

Coordinators: Elaine Wyllie, M.D., and Ahsan Moosa Naduvil Velappil, M.D.

Speakers: Joseph Sullivan, M.D., Mary Connolly, M.D., and Erin Fedak Romanowski, M.D.

For over twenty years, this session has provided a rare platform for discussion of complex challenges in management of pediatric epilepsy. Each of five distinguished faculty present an enlightening case for discussion from their clinical practice, illustrating important new advances in diagnosis and treatment. The session's case-based format allows exploration of a wide range of topics at each session, as can be seen from some recent examples, including NMDA receptor encephalitis, stereotactic EEG in children, near-SUDEP, GLUT-1 deficiency and absence epilepsy syndrome, and epilepsy surgery for ESES secondary to early brain lesions. By offering this annual opportunity to share illustrative clinical experiences, AES fosters impactful interaction and communication among pediatric epilepsy specialists worldwide.

10:00 a.m. - 2:00 p.m.
Exhibit Hall

Convention Center, Hall A3, B3

2:15 p.m. - 3:00 p.m.
Lennox and Lombroso Lecture | EEG, The New Frontier

Convention Center, General Assembly

Lecturer: Jean Gotman, Ph.D.

EEG is an 80-year old clinical test for the evaluation of patients with epilepsy and most of what clinicians use today was discovered over 50 years ago. The last 10 years have seen however a set of major developments that provide improved diagnostic power and stronger links between human epilepsy and experimental models. We will review in particular how High Frequency Oscillations, seen from intracerebral and scalp electrodes, are becoming a new marker of epileptogenicity and how the combination of EEG and fMRI provides a unique non-invasive view of the whole brain during epileptic discharges.

3:15 p.m. - 4:45 p.m.
Investigators Workshops

Sessions run concurrently.

Novel Intracellular Signaling Cascades and Epilepsy: Is There Untapped Therapeutic Potential?

Convention Center, Room 351 A, D

Moderator: Chris Dulla, Ph.D.
Speakers: Gaia Novarino, Ph.D., Michele Jacob, Ph.D., and Angelique Bordey, Ph.D.

New Approaches to the Interictal EEG Challenge the Need to Record Seizures

Convention Center, Room 361 A, D

Moderator: Jean Gotman, Ph.D.
Speakers: Maeike Zijlmans, M.D., Ph.D., Francois Dubeau, M.D., and Andre Palmini, M.D.

3:15 p.m. - 5:15 p.m.
FDA Town Hall Update: Therapeutic Equivalence of Generic Antiepileptic Drugs

Convention Center, Room 371

This FDA town hall meeting will provide updates regarding bioequivalence study design considerations for antiepileptic drugs (AEDs), as well as research findings about generic AED equivalence studies in epilepsy patients. FDA's continued efforts on narrow therapeutic index (NTI) drug classification and modified release AED products will be discussed.

Summary of Generic Equivalence with Findings on Brand Variability

Michel Berg, M.D.

Exploring Generic Brittleness in Epilepsy Patients

Tricia Ting, M.D.

Bioequivalence Study Designs for Anti-epileptic Drugs

Lanyan (Lucy) Fang, Ph.D.

Therapeutic Equivalence of Generic Modified Release AED Products

Wenlei Jiang, Ph.D.

PANEL DISCUSSION

Moderators: Michael Privitera, M.D., and Xiaohui Jiang, Ph.D.

Panel Members: Tricia Ting, M.D., James Polli, Ph.D., Michel Berg, M.D., Norman Hershkowitz, M.D., Lanyan (Lucy) Fang, Ph.D., and Wenlei Jiang, Ph.D.

3:15 p.m. - 5:30 p.m.
Pediatric Epilepsy Highlights Session

Convention Center, Ballroom B

This session will showcase selected scientific abstracts focused on topics in clinical care and research in pediatrics epilepsy. Authors will present a six-minute overview of their work. Presentations are chosen from all submitted abstracts. Participants will be able to view posters and meet the authors at the end of the program.

1.028 Development of a Rapid Functional Assay That Predicts GLUT1 Diesease Severity | Saul Mullen

1.033 Malfunction of β-Catenin Pathways Leads to Infantile Spasms and Seizures | Antonella Pirone

1.182 Trends and Costs of Diagnosis and Treatment of Infantile Spasms | Sunita Misra

1.224 Atypical Cortical Development in Children with Benign Epilepsy with Centrotemporal Spikes (BECTS) | Hisako Fujiwara

1.333 Cognitive Change in Children after Frontal Lobe Resection | Lisa Ferguson

1.335 Frequency of Mosaicism in Parents of Children with Epileptic Encephalopathies | Candace Myers

2.143 Better Together? EEG-fMRI and ESI Improve Localization Accuracy and Predict Surgical Outcome in Paediatric Focal Epilepsy | Maria Centeno

2.249 Epilepsy Surgery for Infants under One Year of Age and Long-Term Seizure and Developmental Outcomes | Kenji Sugai

2.288 The Role of Non-Coding Variation in the Pathogenesis of Epileptic Encephalopathy | Gemma Carvill

2.304 "It Was Five Years of Hell": Parental Experiences of Navigating and Processing the Slow and Arduous Time to Pediatric Resective Epilepsy Surgery | Huibrie Pieters

3:15 p.m. – 5:30 p.m.

Platform Sessions: Three Concurrent Sessions

See page 88 for locations.

There will be three concurrent sessions consisting of selected key scientific abstracts. Authors will present a 10-minute overview of their work followed by a five-minute question and answer period.

5:45 p.m. – 8:15 p.m.

Pediatric State of the Art Symposium | Tuberous Sclerosis Complex (TSC): Understanding and Modifying Epileptogenesis

Convention Center, General Assembly

OVERVIEW

In Tuberous Sclerosis Complex (TSC), at least 70 percent of the patients will develop epilepsy and in the majority, the epilepsy will start before the age of one year. Especially when epileptic spasms occur and/or when the epilepsy becomes drug-resistant, the developmental and behavioral outcome will be unfavorable: Many children will develop intellectual disability and autistic behavior. Because we understand the genetic background in TSC, with the involvement of the mTOR pathway, TSC is becoming a model to study epileptogenesis at the clinical, EEG, imaging and molecular level. This will lead to earlier, better and preventive treatment of epilepsy in TSC. Also, a better and more targeted approach for the frequent behavioral problems will become possible.

LEARNING OBJECTIVES

Following participation in this session, learners should be able to:

- More rapidly diagnose epilepsy in TSC by utilizing clinical, electrophysiological, imaging and molecular biomarkers.
- Describe the preventive treatment of epilepsy in TSC.
- Develop optimal treatment strategies in TSC for epilepsy and behavioral problems.
- Discuss the burden of epilepsy in TSC: drug-resistant epilepsy, intellectual disability and behavioral problems.
- Describe the relationship between medical data and intellectual disability and behavioral problems.
- Delineate new treatment options for behavioral problems in TSC.

TARGET AUDIENCE

Intermediate and Advanced

PROGRAM

Co-Chairs: Jurriaan Peters, M.D., and Lieven Lagae, M.D.

Introduction
Jurriaan Peters, M.D.

Natural History of Epilepsy in TSC
Elizabeth Thiele, M.D.

Intellectual Disability and Behavior in TSC
Paolo Curatolo, M.D.

Current Treatment Options for Epilepsy in TSC
Darcy Krueger, M.D.

Imaging and EEG Biomarkers for TSC Related Epilepsy
Imaging - Jurriaan Peters, M.D.
EEG - Lieven Lagae, M.D.

Molecular Biomarkers in the Epileptogenesis of TSC
Michael Wong, M.D., Ph.D.

Targeting Early and Preemptive Treatment of Epilepsy in TSC to Modify Neurological Outcome
Sergiusz Jozwiak, M.D.

Conclusions
Lieven Lagae, M.D.

EDUCATION CREDIT

2.5 CME credits

Nurses may claim up to 2.5 contact hours for this session.

Pharmacists: AKH Inc., Advancing Knowledge in Healthcare approves this knowledge-based activity for 2.5 contact hours (0.25 CEUs). UAN 0077-9999-16-091-L01-P. Initial Release Date: 12/5/16.

COMMERCIAL SUPPORT ACKNOWLEDGMENT

Supported in part by educational grants from Lundbeck and GW Pharmaceuticals.

3:15 p.m. – 5:30 p.m. Platform Sessions: Three Concurrent Sessions

There will be three concurrent sessions consisting of selected key scientific abstracts. Authors will present a 10-minute overview of their work followed by a five-minute Q &A.

	NJAREMPK4Aú4RPALQJARHMLAJ *Convention Center, Ballroom A* *Moderators*: Melodie Winawer, M.D., M.S., and Eric Marsh, Ph.D.	**PLATFORM B: NEUROPHYSIOLOGY** *Convention Center, Room 310 B* *Moderators*: William Stacey, M.D., Ph.D., and Edward Cooper, M.D., Ph.D.	**PLATFORM C: CLINICAL** *Convention Center, Room 320 B* *Moderators*: Christopher Skidmore, M.D., and Jennifer Hopp, M.D.
3:15 p.m.	**A.01** Spontaneous Seizures and Behavioral Abnormalities in a Novel Open-Access Inducible Mouse Model of Dravet Syndrome | A. Mingorance, L. Goodwin, J. Morgan, S. Rizzo, M. Sasner	**B.01** Deep Brain Stimulation That Mimics Endogenous Synchrony Rapidly Terminates Temporal Lobe Seizures in Rats | D. Mogul, T. Sobayo	**C.01** Non-Adherence with Psychiatric Care among Patients with Psychogenic Nonepileptic Seizures | B. Tolchin, J. Ramel, B. Dworetzky, J. Zinser, G. Baslet **Selected for the Rebecca Goldberg Kaufman Honor***
3:30 p.m.	**A.02** Full Extrapolation of Efficacy from Adults to Children of Antiepileptic Drugs Indicated for the Treatment of Partial Onset Seizures: A Scientific and Regulatory Perspective | A. Men, S. Mehrotra, A. Bhattaram, K. Krudys, M. Bewernitz, R. Uppoor, M. Mehta, T. Liu, P. Sheridan, N. Hershkowitz, E. Bastings, B. Dunn	**B.02** In Vivo Interneuron Circuit Dysfunction in Chronically Epileptic Mice | T. Shuman, D. Aharoni, J. Taxidis, M. Javaherian, C. Kaba, D. Cai, K. Cheng, S. Flores, J. Hodson, N. Rao, A. Fariborzi, J. Lou, J. Daneshrad, C. Yang, S. Ghiaee, R. Manavi, M. Shtrahman, K. Bakhurin, M. Howard, S. Baraban, S. Masmanidis, P. Golshani	**C.02** Effects of Pharmaceutical Grade Cannabidiol on Seizure Severity | J. DeWolfe, E. Bebin, G. Cutter, Y. Liu, J. Szaflarski
3:45 p.m.	**A.03** Diffusion Tensor Imaging: A Non-Invasive Surrogate Marker of Intracranial High Frequency Oscillations | I. Orosz, S. Weiss, D. Woodworth, A. Yogi, H. Ullman, J. Qiao, V. Patel, I. Fried, J. Stern, B. Ellingson, R. Staba, J. Engel, N. Salamon	**B.03** Asynchronous Suppression of Superficial Cortex during Absence Seizures | J. Meyer, A. Maheshwari, J. Noebels, S. Smirnakis	**C.03** Antiepileptic Drug Prescribing Patterns in Pregnant Women with Epilepsy: Findings from the MONEAD Study | K. Meador, P. Pennell, R. May, E. Gerard, L. Kalayjian, N. Velez-Ruiz, P. Penovich, J. Cavitt, J. French, S. Hwang, A. Pack, M. Sam, E. Moore, D. Ippolito
4:00 p.m.	**A.04** Atomoxetine, A Clinically Used Medication to Treat ADHD, Reduces Seizure-Induced Respiratory Arrest | H. Feng, H. Zhang, H. Zhao	**B.04** A Computational Model for EEG Recovery in Postanoxic Encephalopathy | B. Ruijter, J. Hofmeijer, H. Meijer, M. van Putten	**C.04** Predictors of Efficacy In Patients with Adjunctive Everolimus Therapy for Treatment-Resistant Seizures Associated with Tuberous Sclerosis Complex | J. French, A. Wiemer-Kruel, P. Fan, P. de Vries, N. Berkowitz, A. Vaury, S. Peyrard, P. Curatolo
4:15 p.m.	**A.05** Long-Term Changes of Gamma Event Functional Connectivity after KA Induced Status Epilepticus | L. Li, J. Almajano, J. Engel, A. Bragin	**B.05** Prospective Serial EEG Study in Infants with Tuberous Sclerosis Complex (TSC) | G. de Bruyn, B. Verhelle, J. Vervisch, D. Domanska-Pakiela, E. Aronica, P. Curatolo, A. Jansen, F. Jansen, S. Jozwiak, K. Kotulska-Jóźwiak, D. Kwiatkowski, L. Lagae	**C.05** Seizure Control in Patients with Tumour Associated Epilepsy | A. Neal, A. Morokoff, T. O'Brien, P. Kwan
4:30 p.m.	**A.06** Loss of Function of the Circadian Molecular Clock Underlies Hyper-Excitability in Focal Epilepsy | J. Liu, P. Li, X. Fu, N. Smith, G. Valdez, C. Oluigbo, H. Goodkin, W. Gaillard	**B.06** Cortical Current Density Source Analysis Resolves Ambiguities in EEG/MEG Dipole Modeling | M. Wagner, J. Ebersole	**C.06** Design of Focal Brain Cooling Device for Suppression of Epileptic Seizures by Numerical Simulation Based on Pennes Bioheat Equation and Fundamental Equations of Fluid Dynamics | K. Hata, K. Fujiwara, M. Kano, T. Inoue, S. Nomura, H. Imoto, M. Suzuki
4:45 p.m.	**A.07** Models and Mechanisms of SPTAN1 Epileptic Encephalopathy | Y. Wang, T. Ji, K. Glanowska, S. Mojica-Perez, J. Dean, P. Jenkins, M. Uhler, G. Murphy, J. Parent	**B.07** Peri-Inter-Ictal High Frequency Oscillations (30-80Hz) and (80-200Hz) on Magnetoencephalography: A Potential Biomarker of Epilepsy Surgical Outcome in Patients with Drug Resistant Temporal and Extra-Temporal Epilepsies (DRE) | V. Jayabal, S. Nagarajan, S. Sinha, M. Narayanan, R. Chowdary Mundalmuri, A. Arima, R. Dawn Bharath, A. Mahadevan, J. R, M. Bhaskar Rao, T. Kandavel, P. Satishchandra	**C.07** Localization of Seizure Onset Zone Using Classification of Electrocortocographic Synchronization Pattern | B. Elahian, M. Yeasin, B. Mudigoudar, J. Wheless, A. Babajani-Feremi
5:00 p.m.	**A.08** Global and Local Sleep Homeostasis in Patients with Focal Epilepsy: A High-Density EEG Study | M. Boly, B. Jones, G. Findlay, E. Plumley, A. Mensen, B. Hermann, G. Tononi, R. Maganti	**B.08** Modeling Chd2-Linked Epilepsy in Mice | S. Abbasi, J. Frankowski, S. Lee, S. Smith, R. Hunt	**C.08** Phenotypic Analysis of 303 Multiplex Families with Common Epilepsies | S. Bellows, E. Consortium
5:15 p.m.	**A.09** Noninvasive Localization of Interictal High Frequency Oscillations with Simultaneous Magnetoencephalography and Scalp Electroencephalography in Children with Epilepsy | E. Tamilia, N. Tanaka, S. Stufflebeam, P. Pearl, J. Madsen, C. Papadelis	**B.09** Toll-Like Receptor 3 Increases Epileptogensis and Exacerbates Neuroinflammation | F. Benninger, A. Gross, R. Madar, T. Illouz, K. Griffioen, I. Steiner, D. Offen, E. Okun	**C.09** The Use of Cannabidiol for Seizure Management in Patients with Brain Tumor-Related Epilepsy | P. Warren, E. Bebin, J. Szaflarski

***The Rebecca Goldberg Kaufman Honor is awarded to the highest ranking abstract in the comorbidities topic category and is sessioned as Platform C.01. The abstract honored is selected by the Scientific Program Committee from more than 1,100 submitted abstracts.**

MONDAY, DECEMBER 5: POSTER SESSION THREE

8:00 a.m. – 10:00 a.m.
Poster Session Three

Convention Center, Hall A3, B3

Enter behind Pavilion A, in Hall B3

10:00 a.m. – 2:00 p.m.
Poster Session Three

Convention Center, Hall A3, B3

Enter behind the 400 aisle in Hall A3

TRANSLATIONAL RESEARCH

Mechanisms

3.001 The Regulation of Glutamate Transporter-1 (GLT1) and Aquaporin-4 (AQP4) Expression in an Epilepsy Model | J. Hubbard, J. Szu, J. Yonan, D. Binder

3.002 Interneuronopathy as a Non-Genetic Etiology of Infantile Spasms | A. Katsarou, S. Moshé, A. Galanopoulou

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3.267 Hippocampal Volume Loss following Stereotactic Laser Ablation for the Treatment of Temporal Lobe Epilepsies and Associated Changes in Neuropsychological Test Performance | C. Bermudez, W. Jermakowicz, C. Millan, K. McInerney, J. Jagid, M. Lowe, M. Palomeque, R. Ribot, E. Serrano, L. Tornes, N. Velez-Ruiz, A. Kanner, G. Rey

Pediatric

3.268 Hypermotor Seizures (HMS) in Children with Resective Epilepsy Surgery (RES) | A. Stewart, D. Gossett, G. Smith, M. Handler, P. Laoprasert

3.269 Cerebral Hemispherectomy in Children with Intractable Epilepsy: Patient Selection and Surgical Outcome | H. Chugani, M. Ilyas, E. Asano, A. Kumar, S. Sood

3.270 The Role of Intracranial Interictal Infraslow Activity (IISA) in Temporal Lobe Epilepsy (TLE) in Clinical Practice | P. Laoprasert, A. Stewart, M. Handler

3.271 Therapeutic Benefit of Responsive Neurostimulation in Two Pediatric Patients | S. Ghatan, M. Kokoszka, P. McGoldrick, F. Panov, S. Wolf

3.272 Electrocorticography and Intraoperative MRI-Guided Resection of Focal Cortical Dyplasia in Children: Concordance of Findings and Surgical Outcomes | D. Depositario-Cabacar, K. Havens, S. Magge, J. Murnick, T. Tsuchida, W. Gaillard, C. Oluigbo

3.273 Outcomes of MRI-Guided Thermal Ablation in Non-Lesional Intractable Childhood Epilepsy | C. Rao, D. Clarke, F. Perkins, M. Lee, L. Wasson, L. Seery, A. Hernandez, S. Malik, C. Keator, D. Donahue, M. Perry

3.274 Short-Term Cognitive and Adaptive Behavior Stability following Pediatric Hemispherectomy | A. Stefanatos, K. Sarill, J. Madsen, C. Vega, J. Bolton, K. Boyer

3.275 Pediatric Epilepsy Surgery: Results from a New Program | S. Winans, J. Robinson, A. Robison, M. VanHirtum-Das, E. Kiehna, M. Krieger, D. Holder

3.276 A Long Journey for Pediatric Epilepsy Surgery: From Desperation to Hope | L. Fried, D. Cataltepe, J. Riviello, T. McDonough, D. McBrian, A. Mandel, C. Akman

3.277 Long Term Followup in Patients with Surgical Resections of Hypothalamic Hamartomas | N. Kulkarni, J. Kerrigan

All Ages

3.278 Seizure Outcome after Epilepsy Surgery in Norway 2003-2012 | T. Svendsen, K. Alfstad, T. Torjussen, A. Egge, M. Lossius

3.279 Corpus Callosotomy in Patients with Refractory Epilepsy: Invasive EEG Findings and Seizure Outcomes | S. Pedavally, D. Madhavan, M. Puccioni, O. Taraschenko

3.280 Detecting Temporal "Plus" Orbito-Frontal Epilepsy in Surgery Candidates | M. La Vega-Talbott, M. Kokoszka, P. McGoldrick, S. Wolf, S. Ghatan

3.281 Magnetic Resonance-Guided Stereotactic Laser Amygdalohippocampotomy for Mesial Temporal Lobe Epilepsy is Noninferior to Open Temporal Lobe Surgery | M. Stern, J. Willie, R. Gross

3.282 Neuropsychology Outcomes following Trephine Epilepsy Surgery thru an Inferior Temporal Gyrus (ITG) Approach for Selective Amygdalohippocampectomy (AH) | F. Vale, R. Server, W. Clifton, S. Benbadis, M. Schoenberg

3.283 A Comparison of Surgical Treatments for Mesial Temporal Lobe Epilepsy | H. Waseem, A. Vivas, F. Vale

3.284 Multimodality Imaging for Intracranial Electrode Implantation in Epilepsy Surgery | A. Harrison, J. Betts, P. Parks, M. Goyal, P. Kankirawatana, R. Singh, C. Rozzelle, J. Blount

DIETARY

3.285 Ketogenic Diet for Refractory Status Epilepticus in an Infant Presenting with Leigh Syndrome Phenotype Due to Novel Lipoyltransferase 1 (LIPT1) Genetic Defect | R. Stowe, K. Houck, A. Nayak

3.286 Treatment of Intractable Epilepsy with Ketogenic Diet in a Patient with Glycine Encephalopathy | E. Segal, H. Pedro, K. Valdez-Gonzalez, A. Samuels

3.287 Modified Ketogenic Diets Improve Outcomes, Even in the Most Refractory Epilepsy Patients—Those Who Have Failed Epilepsy Surgery | J. Falco-Walter, K. Roehl, A. Balabanov

3.288 Management of Ketogenic Diet Treatment for a Child with POLG Alpers Syndrome | Y. Liu, M. Zak, H. Lowe, V. Chan, E. Donner, J. Kobayashi

3.289 A Ketogenic Diet Combined with Responsive Neurostimulation – A New Approach to Quantifying Efficacy of New Anti-Seizure Treatments | E. Felton, G. Bergey, B. Henry-Barron, E. Kossoff, M. Cervenka

3.290 A Pragmatic Study on Efficacy, Tolerability and Seizure Outcome of Ketogenic Diet Therapy in 74 South Indian Children with Pharmacoresistant Epilepsy | V. Puthenveettil, N. Baby, N. B, A. Roy

3.291 Can a Modified Atkins Diet be Implemented Safely During Pregnancy? | T. Williams, B. Henry-Barron, E. Kossoff, M. Cervenka

3.292 Ketogenic Diet Exhibits Neuroprotective but Not Anticonvulsant Effects on Lithium-Pilocarpine-Induced Status Epilepticus in Rats | W. Pereira, Y. dos Santos, I. Melo, O. Castro, T. Ataíde, D. Gitaí

3.293 Efficacy and Safety of Ketogenic Diet for Management of Refractory Status Epilepticus | R. Arya, K. Peariso, M. Gaínza Lein, J. Harvey, J. Brenton, H. Goodkin, M. Mikati, I. Sánchez Fernández, M. Stosic, D. Tchapyjnikov, A. Wilfong, T. Glauser, T. Loddenkemper

3.294 A Phase I/II Multicenter Ketogenic Diet Study for Super-Refractory Status Epilepticus | M. Cervenka, H. Sara, M. Koenig, B. Bar, B. Henry-Barron, E. Kossoff, A. Hartman, J. Probasco, D. Benavides, A. Venkatesan, E. Hagen, D. Dittrich, T. Stern, B. Radzik, M. Depew, F. Caserta, P. Nyquist, P. Kaplan, R. Geocadin

3.295 Reasons for Noncompliance on the Ketogenic Diet: A Single Center Prospective Study | A. Ruffin, G. Kumar

3.296 Ketone Production and Seizure Control with Orally Fed Medium Chain Triglyceride and Classic Ketogenic Diets | H. Lowe, S. Aimola, Y. Liu, M. Zak, V. Chan, J. Kobayashi, E. Donner

3.297 Modified Ketogenic Diet Treatments in Adults: Efficacy within Subgroups Based on Imaging Abnormalities, Seizure Type, and Diet Type | K. Roehl, J. Falco-Walter, A. Balabanov

3.298 Quality of Life in Children on Classic Ketogenic Diet | E. Fleming, E. Wirrell, P. Eric, K. Nickels, T. Cook, S. Eckert, M. Chinnow, L. Wong-Kisiel

3.299 Identifying Patient Profiles Associated with Difficult Ketogenic Diet Initiation and Risk of Hypoglycemia | S. Karia, L. Dority, D. Dellavalle

3.300 Sotos Syndrome and Ketogenic Diet | E. Fecske, L. Thompson, A. Abdelmoity

3.301 Synergestic Effect of Combining Vagus Nerve Stimulation with Ketogenic Diet Therapy in Patients with Drug Resistant Epilepsy | A. Abdelmoity, K. Pawar, A. Hall, M. Gustafson, E. Fecske, T. Myers, A. Hafez, S. Abdelmoity

3.302 Longitudinal Change of Thyroid Hormone Levels in Children with Epilepsy on Ketogenic Diet | Y. Lee, Y. Kim, G. Yeon, S. Nam

BEHAVIOR/NEUROPSYCHOLOGY/LANGUAGE

Adult

3.303 Reorganization of Verbal Memory in the Absence of Language Reorganization in Left Temporal Lobe Epilepsy Patients | S. Olson, G. Risse, R. Doss

3.304 Investigation of Role of Broca's Area in Speech Production Using High Gamma Electrocorticographic Recording | A. Babajani-Feremi, R. Rezaie, F. Boop, J. Wheless, A. Papanicolaou

3.305 Mapping Memory with Magnetic Source Imaging: Relationship to Wada Memory Performance and Mesial Temporal Sclerosis | G. Risse, R. Doss, W. Zhang

3.306 Topiramate Effects on Fluency of Spontaneous Speech | S. Pakhomov, A. Fieberg, L. Eberly, A. Birnbaum, I. Leppik, C. Barkley, B. Snyder, S. Marino

3.307 Social Conversations Regarding Epilepsy and Driving: Big Data Techniques | J. von Gaudecker, J. Buelow, D. Groves, W. Miller

3.308 Effects of Lunar Phase and Climate Factors on Frequency of Non-Epileptic Seizures; A Retrospective Study | S. Hejazi, V. Shah, S. Izadyar

3.309 Goal-Setting in a Self-Management Education Program for People with Epilepsy (PWE): PAUSE to Learn Your Epilepsy | M. Chesaniuk, N. Nabulsi, P. Shafer, J. Loeb, D. Pandey

3.310 Affective Outcome Following Stereotactic Laser Ablation for the Treatment of Temporal Lobe Epilepsies | K. McInerney, C. Bermudez, W. Jermakowicz, C. Millan, J. Jagid, G. Rey, M. Lowe, M. Palomeque, R. Ribot, E. Serrano, L. Tornes, N. Velez Ruiz, A. Kanner

3.311 Top-Down Facilitation of Visual Object Recognition | C. Kadipasaoglu, K. Forseth, M. Rollo, N. Tandon

Pediatric

3.312 Role of Neurodevelopmental Psychology in a Pediatric Epilepsy Clinic Center: Evaluation of Developmental Delays and Autism Spectrum Disorder | M. Wojnaroski, A. Patel

3.313 The SEEN Study: A Population-Based Study of Neurobehavior in Young Children with Epilepsy and a Comparison Group with Non-Epilepsy Related Neurodisability | C. Reilly, P. Atkinson, A. Memon, C. Gillberg, B. Neville, K. Das, C. Jones, R. Scott

3.314 Behaviour Difficulties and Sleep Disturbance in a Large Cohort of Patients with Sturge-Weber Syndrome | A. Brunklaus, D. Steel, S. Gittins, I. Newsom-Davis, C. Hawkins, S. Aylett

3.315 Delineating Speech Deficits in Dravet Syndrome | S. Turner, A. Brown, M. Arpone, V. Anderson, A. Morgan, I. Scheffer

3.316 Baseline Neurobehavioral Comorbidities Impact 10-Year Outcomes in Childhood Onset Epilepsy | J. Jones, D. Almane, M. Hanson, B. Morrison, B. Hermann

3.317 Risk of Verbal Function after Resective Surgery in Children with Drug- Resistant Epilepsy | S. Baba, E. Kerr, O. Muthaffar, A. Ochi, C. Go, E. Widjaja, J. Drake, J. Rutka, E. Donner, C. Snead III, H. Otsubo

3.318 Premorbid Hyperactivity Prior to New-Onset Epilepsy Predicts Anti-Epileptic Drug Behavioral Side Effects One Month after Initiation | S. Guilfoyle, K. Junger, S. Ollier, B. Hater, C. Hamilton, A. Modi

3.319 New Onset Pediatric Epilepsy (NOPE) Clinic: Measurable Declines Identified at 1-Year Follow-Up and Cognitive Differences Associated with Treatment and Recent Seizures | K. Kaszynski, E. Adams, J. Doss, P. Brown, F. Ritter

3.320 Executive Dysfunction Is Predictive of Elevated Depression Features in Youth with Temporal Lobe Epilepsy | W. Schraegle, N. Nussbaum, J. Titus

3.321 New Onset Pediatric Epilepsy (NOPE) Clinic Neuropsychological Evaluation Yields Higher than Normal Prevalence of Attention and Learning Problems Shortly after Epilepsy Diagnosis | E. Adams, J. Doss, M. Palmquist, P. Brown, F. Ritter

GENETICS

Human Studies

3.322 IQSEC1 - A Novel Gene Mutation in a Family with Dravet Like Syndrome | T. Abd Majid, A. Al-Otaibi, H. Aldhalaan, A. AlAsmari

3.323 Increased Epilepsy Prevalence in Adults with 22q11 Deletion Syndrome | R. Wither, F. Borlot, N. Butcher, E. Chow, A. Bassett, D. Andrade

3.324 GRIN1 Polymorphisms Do Not Affect Susceptibility or Phenotype in NMDA Receptor Encephalitis | G. Day, H. Prüss, S. Benseler, T. Paton, A. Paterson, D. Andrade

3.325 Characterizing Epilepsy Phenotypes in Patients with Intellectual Disability, Autism Spectrum Disorder, Developmental Delay, Congenital Abnormalities and Chromosomes 15q11.2, 15q13.3 and 16p13.11 Microdeletions | F. Nascimento, R. Wennberg, D. Aljaafari, A. Tahiri, E. Young, J. Stavropoulos, D. Andrade

3.326 Prevalence of Copy Number Variation in Adults with Epilepsy and Intellectual Disability | F. Borlot, A. Bassett, B. Regan, J. Stavropoulos, D. Andrade

3.327 Hunting for the Genetic Cause in SCN1A Mutation Negative Dravet Syndrome Patients | A. Muir, C. Myers, G. Hollingsworth, A. Schneider, I. Scheffer, H. Mefford

3.328 Characteristics of SCN1A Mutation Locations in a Cohort of 285 Japanese Dravet Syndrome Patients | A. Ishii, J. Watkins, D. Chen, S. Hirose, M. Hammer

3.329 An Informatics Infrastructure for KCNQ2 Encephalopathy Research Including a Patient Registry, Database, Curation Platform, and Website | N. Joshi, M. Taglialatela, S. Weckhuysen, G. Nesbitt, E. Cooper

3.330 Genetic Testing for Complex Epilepsy Patients – Lessons from a 2 Year Experience | M. Pietryga, A. Patel

3.331 Genetic Risk Patterns in Sudden Death Are Linked with Age-Dependent Splice Variant Expression | T. Klassen, C. Troskie, P. Boutet, R. Siu, A. Smith

HEALTH SERVICES

Delivery of Care

3.332 Anti-Epileptic Drug (AED) Monotherapy Use in Older Medicare Beneficiaries with New-Onset Epilepsy | J. Richman, R. Martin, E. Faught, J. Szaflarski, E. Funkhouser, L. Piper, L. Juarez, C. Dai, M. Pisu

3.333 Adherence to Antiepileptic Drugs (AEDs) in Older Americans with Epilepsy | M. Pisu, L. Piper, J. Richman, R. Martin, J. Szaflarski, E. Funkhouser, C. Dai, L. Juarez, E. Faught

3.334 Improving Testing During Seizures in a Pediatric Epilepsy Monitoring Unit: A Quality Improvement (QI) Process | C. Garcia Gracia, J. Bautista, L. Bell, C. Gary, C. Kenik, J. Hartman, S. Sykes, A. Moosa

3.335 Healthcare Utilization Characteristics and Demographics of Patients Prescribed Intranasal Midazolam Versus Rectal Diazepam | S. Nunley, P. Glynn, J. Vidaurre, D. Albert, A. Patel

3.336 Bone Health Assessment in Veterans—Are We Doing Enough? | K. McMillan, S. Gowda`, A. Van Cott, M. Pugh

3.337 Is Quality of Care Associated with Patient Reported Outcomes? | M. Pugh, S. Chan, J. Cramer

3.338 Designing a Pediatric Epilepsy Population Management Dashboard | T. Hill, B. Hafeez, J. Ancker, Z. Grinspan

EPIDEMIOLOGY

3.339 Impact of Epilepsy and Seizures on Siblings of Children with Epilepsy in the Seizures and Outcomes Study | A. Bumbut, M. Ardini, W. Gaillard, B. Kroner

3.340 Relationship between Severity of Traumatic Brain Injury with the Onset Time of Post Traumatic Epilepsy | K. Mejía Rojas, I. Lagger, L. Orellana, M. Pacha, G. Ernst, O. Martinez

3.341 Clinical Variables Associated with Acute Symptomatic Seizures in the Emergency Room | M. Holtkamp, S. Reinecke, A. Kowski

3.342 Seizures and Other Clinical Manifestations Prior to Diagnosis of Tuberous Sclerosis Complex | J. Wheless, N. Engel-Nitz, C. Blauer-Peterson, J. Johnson, Q. Said, T. Gipson

3.343 The Quality of National Birth Certificate Data for Neonatal Seizure Epidemiology | K. Berry, M. Pesko, D. Hesdorffer, R. Shellhaas, J. Seirup, Z. Grinspan

3.344 An Update on the Veterans Health Administration Epilepsy Centers of Excellence Registry Project | R. Rehman, P. Kelly, A. Husain

3.345 Spanish-MINDSET: Usability and Feasibility of Epilepsy Self-Management Decision Support for Spanish Speaking Patients | R. Sepulveda, J. Chong, D. Labiner, N. Halavacs, C. Begley, R. Shegog, K. Martin

3.346 Transition from Paediatric to Adult Health Care: Readiness and Experiences of Adolescents and Young Adults (AYAs) Diagnosed with Epilepsy in Childhood | A. Dasiewicz, G. Zou, C. Camfield, K. Speechley

3.347 Comorbidity Differs between Persons Hospitalized with Tractable and Intractable Epilepsy: U.S. Nationwide Inpatient Sample (NIS)/ Healthcare Cost and Utilization Project (HCUP), 1998-2006 and 2009-2011 | Y. Luo, M. Zack

3.348 Re-Visiting Racial Disparities in Access to Surgical Management of Intractable Temporal Lobe Epilepsy and Short-Term Post-Operative Outcomes | K. Sharma, P. Kalakoti, E. Disbrow, R. Riel-Romero, C. Notarianni, A. Minagar, E. Gonzalez-Toledo, A. Nanda, H. Sun

CASE STUDIES

3.349 A Case of SUDEP after Improved Seizure Control with the Aspire SR Vagal Nerve Stimulator | M. Etienne, M. McCauley, R. Venor

3.350 Resection of Focal Cortical Dysplasia Is Effective Even with Co-Existing Dravet Syndrome Due to SCN1A Mutation | S. Tomko, P. Ann, M. Takeoka

3.351 Alpha Coma as a Complication of Ethylene Glycol Poisoning | M. Nguyen, L. Ernst

3.352 Resuscitation and Sudden Unexpected Death In Epilepsy (SUDEP): A Report of Six Near-SUDEPs from the North American SUDEP Registry (NASR) | K. Doumlele, O. Devinsky, D. Friedman

3.353 Rapidly Progressive Temporal Lobe Epilepsy Associated with Hippocampal Sclerosis and Glutamic Acid Decarboxylase 65 (GAD65) Antibody- A Case Report and Literature Review | S. Wu, N. Issa, S. Rose, P. Warnke, J. Jacobsen, V. Towle, J. Tao

3.354 Severe Refractory Epilepsy in Adults following Remote Childhood Cancer Treatment with Cranial Radiotherapy | A. Chalissery, A. Gunko, E. Maloney, H. ElNaggar, G. Mullins, D. Colin, P. Widdess-Walsh, R. Kilbride, N. Delanty

3.355 Linear Nevus Sebaceous Syndrome with Pharmacoresistant Epilepsy in a Cognitively Normal Adult | J. Nguyen, B. Krishnaiah, J. Acharya, V. Acharya

3.356 A Case of Bannayan-Riley-Ruvalcaba Syndrome with Normal Brain MRI | J. Nguyen, B. Krishnaiah, V. Acharya, J. Acharya

3.357 De Novo DNM1L Mutation in a Teenager with Myoclonic Super-Refractory Status Epilepticus | C. Ryan, A. Fine, A. Cohen, B. Schiltz, E. Wirrell, M. Patterson, E. Payne

3.358 CACN1A Mutation in an Infant with Focal Epilepsy and Ataxia | R. Luke, S. Verma

3.359 Vagal Nerve Stimulation for Super Refractory Status Epilepticus in Children | P. Pichon Zentil, P. Chee

3.360 TBC1D24 Gene Mutations: Two Brothers with Novel Phenotypic Presentations | S. Alick, L. Morgan

Convention Center, Room 310 B

Coordinators: Susan T. Herman, M.D., and Kitti Kaiboriboon, M.D.

Speakers: Nicholas Abend, M.D., MSCE, Saurabh Sinha, M.D., Ph.D., and Brandon Westover, M.D., Ph.D.

Long-term continuous EEG (CEEG) monitoring is a valuable method for diagnosis of epilepsy and for detection of seizures in patients with altered mental status. CEEG is a labor-intensive process, requiring 1 – 2 hours for review and interpretation of 24 hours of CEEG. Quantitative EEG trends, or graphical displays of EEG characteristics, can aid in identification of abnormal segments of EEG, making review more efficient and facilitating real-time communication of EEG abnormalities. In this session, speakers will review commonly employed quantitative EEG techniques and discuss their use in the detection of seizures, ischemia and other abnormalities in the epilepsy monitoring unit and intensive care unit. Presentations will highlight practical aspects of using quantitative EEG trends, including pros and cons of various trends and pitfalls in use of trends. Presentations include: QEEG Trends for Seizure Detection (Saurabh Sinha, M.D., Ph.D.); QEEG Trends in Neonates and Children (Nicholas Abend, M.D., MSCE); and, QEEG Trends for Identification of Ischemia (Brandon Westover, M.D., Ph.D.)

Neuropharmacology | Antiepileptic Drug Selection in Special Population

Convention Center, Room 361 A, D

Coordinators: Mitra Habibi, Pharm.D., and Archana Shrestha, M.D.

Speakers: Mitra Habibi, Pharm.D., Chantal O'Brien, M.D., and Kim Tallian, Pharm.D.

This SIG focuses on individuals with a common interest in neuropharmacology and epilepsy. Epilepsy can affect various different patient populations, including the elderly, people with other chronic disorders and those with limited income. Anticonvulsant medications are the mainstay of treatment for people with epilepsy and there are many medication options including generics available. However, the use of antiepileptic drugs in these special populations of patients can pose significant problems. For instance elderly patients are usually on multiple drugs and their age related pharmacokinetic changes put them at higher risk for important drug-drug interactions and adverse events. Also, access to these medications due to their cost can be an issue for patients. The special populations being treated and their needs and risks must be considered when selecting anticonvulsant medications. The SIG this year will focus on anticonvulsant medication selection in specific populations of patients with epilepsy.

Neuropsychology | Cognition and Epilepsy Across the Lifespan

Convention Center, Room 371 A, D

Coordinator: Gail L. Risse, Ph.D.

Speakers: Elizabeth Adams, Ph.D., Marilyn Jones-Gotman, Ph.D., and Bruce Hermann, Ph.D.

The effects of chronic seizure activity on cognitive function remain controversial. This SIG will explore the evidence for cognitive impairment in pediatric new onset epilepsy, cognition in aging persons with chronic epilepsy, including metabolic and vascular risk factors, and cognition in aging temporal lobectomy patients.

Psychogenic Nonepileptic Seizures | Global Perspectives on Psychogenic Nonepileptic Seizures (PNES): Research and Clinical Experiences across Countries and Cultures

Convention Center, Room 370 A, D

Coordinator: Sigita Plioplys, M.D.

Speakers: A. Ali Asadi-Pooya, M.D., Chrisma Pretorius, Ph.D., Claire DeSouza, M.D., A. DeMarinis Palombo, M.D., Dong Zhou, M.D., Kette Valente, M.D., Kousuke Konemoto, M.D., and Yacov Ezra, M.D.

This SIG will provide a forum for the PNES experts from Japan, China, Israel, Iran, South Africa, Canada, Chile and Brazil to disseminate their research and clinical data at the AES. The discussion about the multicultural PNES research and clinical experience will improve the knowledge and encourage development of global strategies for PNES diagnosis and treatment. Specific topics for presentations will include: demographic and clinical characteristics of PNES patients in Asia, the Middle East, Africa, North America and South America; cultural differences in provider and patient perceptions of PNES; barriers in effective care and different approaches to PNES management in the context of limited access to the vEEG; and lack of access to research data that is specific to certain countries and/or cultures. A multidisciplinary hospital-based model of services for pediatric PNES, developed at the Hospital for Sick Children in Toronto will be presented. The limitations in PNES education and clinical training will be highlighted to reflect that it is a global problem.

Tuberous Sclerosis | Novel and Investigational Therapeutic Approaches to Epilepsy in Tuberous Sclerosis

Convention Center, Room 320 B

Coordinator: Michael Wong, M.D., Ph.D.

Speakers: Darcy Krueger , M.D., Ph.D., Elizabeth Thiele, M.D., Ph.D., and Rohini Coorg, M.D.

Epilepsy occurs commonly in tuberous sclerosis complex (TSC) and is often resistant to available treatments. Progress in understanding the pathophysiology of epilepsy in TSC provides potential opportunities for novel therapeutic approaches in TSC. Furthermore, TSC is often viewed as a model disease, in which pathophysiological and therapeutic advances may be relevant to other types of genetic and acquired epilepsies. For example, mTOR inhibitors are currently being investigated as treatments for drug-resistant epilepsy in TSC and also have potential preventative, antiepileptogenic effects, but are starting to be considered in other types of genetic cortical malformation involving mTORopathies, such as focal cortical dysplasia. While cannabinoids (CBD) have been a hot area of epilepsy research primarily focused on Dravet and Lennox-Gastaut syndromes, clinical trials of CBD in TSC patients are now under way. In terms of novel surgical approaches, laser ablation has become a cutting-edge surgical option for epilepsy and may be particularly amenable to the multifocal tubers of TSC. In this SIG, we will review these and other novel therapeutic approaches to epilepsy in TSC. Darcy Krueger from the Cincinnati Children's Hospital Medical Center will present the latest clinical trial updates related to both antiseizure and antiepileptogenic indications for mTOR inhibitors. Elizabeth Thiele, M.D., Ph.D., of Massachusetts General Hospital will discuss the potential use of CBD in TSC. Rohini Coorg, M.D., of Baylor College of Medicine will present her institution's experience with laser ablation for drug-resistant epilepsy in TSC. Ample time will be reserved for discussion of other potential novel therapies in various stages of research, such as preventative approaches with vigabatrin or ketogenic diet and targeting other aspects of the mTOR pathway.

8:45 a.m. - 10:45 a.m.

Hot Topics Symposium

Convention Center, Ballroom B, C

OVERVIEW

The ROSE trial has just been completed, and this symposium will present two talks offering the first comprehensive review of this study, which compared stereotactic radiation and open surgery for treatment of drug resistant temporal lobe epilepsy. As the recent addition of several new drugs to treat epilepsy adds further complexity of choosing appropriate therapy, this symposium will review how these agents should be incorporated into clinical practice, discussing efficacy, side effects, cost effectiveness and mechanisms of action. The final talk will review new personal monitoring apps developed for patients to track, detect and manage seizures. These apps are gaining widespread use and offer new opportunities for managing seizures.

LEARNING OBJECTIVES

Following participation in this symposium, learners should be able to:

- Discuss the use of stereotactic radiation for treatment of drug resistant temporal lobe epilepsy and advise patients regarding treatment options.
- Discuss the use of new anticonvulsant medications and better plan antiepileptic drug therapy.
- Describe various apps available on smartphones and other portable electronic devices and advise patients on the use of these apps.

TARGET AUDIENCE

Intermediate and Advanced

PROGRAM

Chair: Michael Sperling, M.D.

Introduction
Michael Sperling, M.D.

ROSE (Radiosurgery or Open Surgery for Epilepsy) Trial: Efficacy and Adverse Events
Nicholas Barbaro, M.D.

ROSE (Radiosurgery or Open Surgery for Epilepsy) Trial: Efficacy and Adverse Events: Secondary Outcomes and Health Care Utilization
Mark Quigg, M.D.

How Should Recently Approved Antiepileptic Drugs Be Incorporated into Clinical Practice?
Elinor Ben-Menachem, M.D., Ph.D.

Seizure Apps and Non-EEG Seizure Detection
Gregory Krauss, M.D.

Conclusions
Michael Sperling, M.D.

EDUCATION CREDIT

2.0 CME credits

Nurses may claim up to 2.0 contact hours for this session.

Pharmacists: AKH Inc., Advancing Knowledge in Healthcare approves this knowledge-based activity for 2.0 contact hours (0.2 CEUs). UAN 0077-9999-16-092-L01-P. Initial Release Date: 12/6/16.

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8:45 a.m. - 10:45 a.m.

Scientific Symposium | The Neurobiology of Brain Stimulation in Epilepsy: Targets, Networks and Cascades

Convention Center, Ballroom A

OVERVIEW

Brain stimulation is an evolving treatment for medically refractory epilepsy. This session will convey the basic anti-seizure mechanisms of brain stimulation. The history of brain stimulation will be presented as well as a discussion of the sites of stimulation in animal models. How electrical current spreads through brain tissue will be explained. Conceptualization of brain stimulation paradigms as ablative forces will be discussed, and information on how stimulation of a network can modulate an epileptogenic focus will be addressed. Examples of how chronic stimulation can modulate protein pathways and lead to neuroplastic changes will be presented; potential mechanisms of improved efficacy over time will be covered. Clinical studies have sometimes proceeded with only limited understanding of the basic mechanisms. As competing and complementary technologies emerge, new treatment algorithms should be developed to direct patients to the best treatment options. Understanding of the neurobiology and mechanisms of brain stimulation may illuminate clinical trials and treatment decisions, contributing to the development of anti-seizure therapeutic best practices utilizing this approach.

LEARNING OBJECTIVES

Following participation in this session, learners should be able to:

- Discuss the anti-seizure mechanisms of brain stimulation for medically refractory epilepsy.
- Provide an overview of empirical observations of various sites of anti-seizure stimulation.
- Describe how anti-seizure stimulation can both disrupt and drive local neuronal circuits.
- Delineate the network effects of anti-seizure stimulation.
- List chronic the neuronal changes that may drive long term benefits of brain stimulation for epilepsy.

TARGET AUDIENCE

Intermediate and Advanced

PROGRAM

Chair: Kevin Graber, M.D.

Introduction and Overview of Brain Stimulation in Models and Patients: Targets and Empirical Effects
Kevin Graber, M.D.

Mechanisms of Seizure Control with Local Circuit Stimulation
Dominique Durand, Ph.D.

Modulation of Epileptic Networks with Electrical Stimulation
Kristl Vonck, M.D., Ph.D.

Positive Cascades: Improving Efficacy and Outcomes
Esther Krook-Magnuson, Ph.D.

Conclusions
Kevin Graber, M.D.

EDUCATION CREDIT

2.0 CME credits

Nurses may claim up to 2.0 contact hours for this session.

Pharmacists: AKH Inc., Advancing Knowledge in Healthcare approves this knowledge-based activity for 2.0 contact hours (0.2 CEUs). UAN 0077-9999-16-093-L01-P. Initial Release Date: 12/6/16.

11:00 a.m. - 12:30 p.m. Session One
12:45 p.m. - 2:15 p.m. Session Two

Skills Workshops

Advance registration and tickets are required for these sessions. An additional $50 registration fee applies for each workshop; maximum of 30 people per session. Sessions run concurrently.

Basic EEG in Epilepsy: Fundamentals and Interpretation

Convention Center, Room 320 C

11:00 a.m. – 12:30 p.m.
12:45 p.m. – 2:15 p.m.

Moderator: Gregory D. Cascino, M.D.

The routine EEG recording remains essential in the care and management of individuals with seizures and suspected epilepsy. The EEG is used for diagnosis, classification of seizure type and identification of a specific epileptic syndrome. EEG findings may be of prognostic importance and be used to assess the efficacy of treatment. Use of appropriate EEG methodology and recognition of artifact and benign variant patterns are essential for satisfactory clinical studies. This workshop will review basic methodologies of EEG for the evaluation and treatment of pediatric and adult patients with seizure disorders. This will include use of appropriate EEG techniques and fundamentals of EEG recordings. Recognition of benign variant alterations and ictalinterictal epileptogenic discharges will be addressed. The presentations will also discuss the importance of EEG to identify characteristics of specific epilepsies and epileptic syndromes.

Basics of Neuroimaging Acquisition and Processing: What the Clinician Needs to Know

Convention Center, Room 310 C

11:00 a.m. – 12:30 p.m.

Please note this Workshop occurs only once.

Moderators: R. Edward Hogan, M.D., Andrea Bernasconi, M.D., and Matthias Koepp, M.D.

Basic principles of image acquisition and post-image-acquisition-processing have important implications for correlation with clinical history and EEG findings in the diagnosis and treatment of epilepsy. The workshop will focus on pertinent basic principles of acquisition and processing of MRI, PET, and SPECT. The program will review the basic steps of structural the MR image acquisition and processing using a simple algorithm ("processing pipeline" figures), and discussion of common pitfalls (movement, inhomogeneity, causes for miss-segmentation, manual corrections). For PET, we will concentrate primarily on imaging glucose metabolism with FDG emphasizing best practice of data acquisition and analysis, but also cursory mention the current state-of-play of clinical use of novel PET ligands. Discussion of SPECT will include important issues of administration of radiopharmaceutical in the epilepsy monitoring unit, as well as basics of image acquisition, co-registration and normalization for subtraction SPECT studies.

Genetic Testing in Epilepsy Patients

Convention Center, Room 310 B

11:00 a.m. – 12:30 p.m.
12:45 p.m. – 2:15 p.m.

Moderators: Alica M. Goldman, M.D., Ph.D., and Annapurna Poduri, M.D.

Novel detection platforms have accelerated scientific discoveries of genes relevant to patients with epilepsy of all ages. These research findings are being used in institutions and commercial laboratories. Selection of patients that would most benefit from the genetic investigations, identification of the appropriate tests and reporting of results are increasingly complex. This skills workshop will review available testing platforms and outline case scenarios driven testing algorithms. The aim is to provide a practical clinical guide in selecting patients, testing methods, and the workflow involved in ordering, submitting and reporting genetic tests.

The workshop is designed to be an interactive, case-driven discussion and a practical guide for clinical care. Participants are encouraged to submit questions and cases to Drs. Goldman and Poduri. The goal is to address pressing questions and discuss real life cases within the context of genetic testing driven diagnostics and care.

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Convention Center, Room 320 B

11:00 a.m. – 12:30 p.m.
12:45 p.m. – 2:15 p.m.

Moderator: Dennis Spencer, M.D.

Over the past thirty years, resection for medically intractable epilepsy has become a standard treatment option. However, in many instances successful surgery is not possible without defining the potential resective volume by intracranial electrophysiology. Imaging and stereotactic navigation have made great strides and epilepsy centers have many choices regarding types of electrodes, number of contacts needed and how they are delivered.

This is an interactive workshop where two – three cases are presented illustrating different problems to be solved in defining a region of epileptogenesis. The participants work in groups to provide a consensus intracranial study. A guest experienced epileptologist will provide his or her institution's approach to the case and we will then describe what was done and the outcome. There will be an attempt to discuss as many alternative approaches as possible, balancing the invasiveness of the various procedures and risk versus benefit.

Neurostimulation/VNS

Convention Center, Room 310 A

11:00 a.m. – 12:30 p.m.
12:45 p.m. – 2:15 p.m.

Moderator: Mohamad Koubeissi, M.D.

Neurostimulation is now an accepted treatment option for patients with refractory epilepsy. Two devices are approved by the FDA for patients with epilepsy: the vagus nerve stimulation (VNS) and the responsive neurostimulator (RNS). This workshop will discuss and instruct on how to use these devices effectively. After the workshop, participants should be able to identify appropriate patients, understand how implantation is carried out and how to program the devices. Side effects and how to practically manage them will be discussed as well.

Optimal Use of Neuroimaging in Diagnosing and Treating Epilepsy

Convention Center, Room 320 A

11:00 a.m. – 12:30 p.m.
12:45 p.m. – 2:15 p.m.

Moderators: John Stern, M.D., and David Millett, M.D., Ph.D.

Neuroimaging is an essential tool in the diagnosis and treatment of epilepsy. It has opened a window on the pathological substrate underlying epilepsy, ranging from subtle gliotic lesions and cortical malformations to larger, more extensive structural disturbances. This workshop will review the techniques used to diagnose epilepsy, emphasizing both basic MRI customized for epilepsy and advanced neuroimaging techniques. We will review a rational approach to the use of neuroimaging, highlight specific techniques that enhance diagnostic ability, along with newer fMRI and other functional imaging methods. Interpretation of scans and various findings will be reviewed in this practical session.

Treating Patients with Psychogenic Nonepileptic Seizures

Convention Center, Room 310 C

12:45 p.m. – 2:15 p.m.

Please note this Workshop occurs only once.

Moderator: W. Curt LaFrance, Jr., M.D., M.P.H.

Psychogenic nonepileptic seizures (PNES) are prevalent and disabling and are often identified in seizure monitoring units. Neurologists readily diagnose PNES, but the majority of providers do not feel equipped to treat patients with PNES. Psychogenic NES present in adults and children with neurologic signs, psychological stressors and comorbid psychiatric disorders. For years, neurologists, psychiatrists and psychologists have accumulated data about NES phenomenology, epidemiology, risks, comorbidities and prognosis. The role of the neurologist and mental health providers in the diagnosis and management of these patients will be discussed, and common obstacles that preclude proper treatment will be reviewed. ILAE Task Force recommendations and randomized clinical trial data will be presented, including pharmacologic and non-pharmacologic interventions. Participants will observe treatment of patients with PNES using a validated intervention shown to reduce seizures, improve comorbidities and quality of life. Session participants will view video vignettes from in-session interactions between clinicians (including epileptologists and mental health workers) providing PNES treatment. During this workshop participants will learn the elements of the 12-session intervention, using the seizure treatment workbook.

11:00 a.m. – 5:00 p.m.

Preclinical Common Data Elements: Update on the AES/ILAE/NINDS Initiative

Hilton, Lanier Grand Ballroom A, Level Four

Co-chairs: Helen Scharfman, Ph.D., Jacqueline French, M.D., and Asla Pitkanen, M.D., Ph.D.

In human epilepsy clinical trials, the use of common forms for obtaining key data points (common data element forms) has contributed greatly to the acquisition of high quality data that can be compared across sites. The AES/ILAE translational task force of the ILAE with the partnership of NINDS have developed common data element forms for key aspects of pre-clinical translational research that include behavior, physiology, pharmacology and EEG studies. This open forum will be the first opportunity for the communities who will be the end-users of these forms to evaluate them and provide comments. The feedback received will be critically important for the development of broadly community-accepted usable forms.

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, -
Washington, D.C.
December 1 - 5

2018
New Orleans, LA
November 30 - December 4

2019
Baltimore, MD
December 6 - 10

2020
Seattle, WA
December 4 - 8

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AESnet.org/annual_meeting

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- Exclusive Door Drop (Friday)
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- CME Grant: Interprofessional Care Symposium
- CME Grant: Hot Topics Symposium
- Grant to Support AES PI-CME Education System
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- CME Grant: Annual Fundamentals Symposium
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GeneDx

Monteris Medical

Neuralynx, Inc.

PMT Corporation

Brain Sentinel

EXHIBITOR LOCATIONS

EXHIBIT HALL MAP

Third Floor Convention Center

Exhibit Hall A3, B3

Innovation Pavilions

Convention Center, Hall B3

Visit the Innovation Pavilions to experience in-depth education and training from exhibitors—featuring some of the latest research and technology related to the treatment and prevention of epilepsy.

QARSPCAVü4CDBDKÁDP4q	SUNDAY, DECEMBER 4	MONDAY, DECEMBER 5
Noon - 6:00 p.m.	10:00 a.m. - 4:00 p.m.	10:00 a.m. - 2:00 p.m.
Pavilion A - **Sunovion Pharmaceuticals Inc.**	Pavilion A - **Sunovion Pharmaceuticals Inc.**	Pavilion A - **Sunovion Pharmaceuticals Inc.**
Pavilion B - **Lundbeck**	Pavilion B - **Lundbeck**	Pavilion C - **GW Pharmaceuticals**
Pavilion C - **NeuroPace, Inc.**	Pavilion C - **NeuroPace, Inc.**	Pavilion D - **Tuberous Sclerosis Alliance**
Pavilion D - **LivaNova**	Pavilion D - **Eisai Inc.**	Pavilion E - **UCB, Inc.**
Pavilion E - **UCB, Inc.**	Pavilion E - **UCB, Inc.**	

EXHIBITOR CATEGORIES

BOOTH/TABLE

Accreditation/Credentialing

ABRET Neurodiagnostic Credentialing & Accreditation 16
American Board of Clinical Neurophysiology 17
American Board of Psychiatry and Neurology 332
DigiTrace EEG Services 117
National Association of Epilepsy Centers 637

Brain Mapping

Ad-Tech Medical Instrument Corp. 710
ANT North America 340
Compumedics USA 427
Elekta, Inc. 621
FHC, Inc. 827
Guger Technologies OG 915
Nihon Kohden America, Inc. 413
PMT Corporation 200
Ripple LLC 440
York Instruments 343

Clinical Tools

Child Neurology Foundation 7
Compumedics USA 427
Electrical Geodesics, Inc. 418
Evogen Precision Medicine 341
IntraDiagnostics 734
Monteris Medical 212
MVAP Medical Supplies, Inc. 113
Neuralynx, Inc. 412
SeizureTracker.com 20

Diagnostics

Alliance Family of Companies 841
Blackrock NeuroMed 527
Cadwell Industries, Inc. 521
Compumedics USA 427
Evogen Precision Medicine 341
Fulgent Diagnostics 922
GeneDx 719
IntraDiagnostics 734
Invitae 825
MediMax Tech, Inc. 106
MNG Laboratories 101
MVAP Medical Supplies, Inc. 113
Neurotech, LLC 641
Perfect Phorm, LLC 132
PMT Corporation 200
Rhythmlink International, LLC 241
RSC 110
York Instruments 343

BOOTH/TABLE

Education

American Epilepsy Society 326
The Anita Kaufmann Foundation 11
ASET - The Neurodiagnostic Society 14
Bridge the Gap - SYNGAP Education and Research Foundation 25
Epilepsy Foundation 5, 716
Epilepsy Foundation SUDEP Institute and North American SUDEP Registry 9
Epilepsy Foundation Texas - Houston/ Dallas-Fort Worth/West Texas 108
Glut1 Deficiency Foundation 4
Hope for Hypothalamic Hamartoma 8
Hope4Harper 22
Informal Learning Center 327
MVAP Medical Supplies, Inc. 113
NINDS-National Institute of Neurological Disorders and Stroke 921
Oxford University Press 229
UCSF Medical Center 913
VA Epilepsy Centers of Excellence 10

EEG Systems

ANT North America 340
Blackrock NeuroMed 527
Cadwell Industries, Inc. 521
Compumedics USA 427
CortiCare 124
CREmedical Corp. 914
DigiTrace EEG Services 117
Electrical Geodesics, Inc. 418
Global Neuro-Diagnostics, LP 635
Guger Technologies OG 915
Lifelines Neurodiagnostic Systems, Inc. 818
Natus Neurology Incorporated 627
Neurotech, LLC 641
Nihon Kohden America, Inc. 413
Persyst Development Corporation 321
Pinnacle Technology, Inc. 740
PMT Corporation 200
Rhythmlink International, LLC 241
Ripple LLC 440
RSC 110
York Instruments 343

Electrodes

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Alpha MED Scientific Inc. 127
ANT North America 340
Cadwell Industries, Inc. 521
CREmedical Corp. 914

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MediMax Tech, Inc. 106
MVAP Medical Supplies, Inc. 113
Natus Neurology Incorporated 627
PMT Corporation 200
Rhythmlink International, LLC 241
Ripple LLC 440

Genetic Services

Ambry Genetics 233
Courtagen Life Sciences, Inc. 118
Evogen Precision Medicine 341
Fulgent Diagnostics 922
GeneDx 719
Invitae 825
The North American AED Pregnancy Registry 336
Pairnomix 829

Hospital/Medical Center

NINDS-National Institute of Neurological Disorders and Stroke 921
Penn State Hershey Medical Center 843
Texas Children's Hospital 136
UCSF Medical Center 913
VA Epilepsy Centers of Excellence 10

Imaging

ANT North America 340
CREmedical Corp. 914
Guger Technologies OG 915
York Instruments 343

Medical Device

Ad-Tech Medical Instrument Corp. 710
Brain Sentinel 742
Cadwell Industries, Inc. 521
Empatica 837
FHC, Inc. 827
Guger Technologies OG 915
Integra LifeSciences 823
LivaNova 513
Monteris Medical 212
MRI Interventions 216
Neuralynx, Inc. 412
NeuroPace, Inc. 213
Nihon Kohden America, Inc. 413
Peachtree BioResearch Solutions 228
PMT Corporation 200
Rhythmlink International, LLC 241
Ripple LLC 440
Smart Monitor Corp. 736
York Instruments 343

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Ripple LLC 440

Mobile App
Empatica 837
SeizureTracker.com 20

Monitoring Systems
Brain Sentinel 742
DigiTrace EEG Services 117
Electrical Geodesics, Inc. 418
emka TECHNOLOGIES Inc. 832
Empatica 837
Global Neuro-Diagnostics, LP 635
Integra LifeSciences 823
Natus Neurology Incorporated 627
Neurotech, LLC 641
Nihon Kohden America, Inc. 413
Perfect Phorm, LLC 132
Persyst Development Corporation 321
RSC 110
SeizureTracker.com 20
Smart Monitor Corp. 736

Other
Alpha MED Scientific Inc. 127
Citizens United for Research in Epilepsy (CURE) 21
Epilepsy Foundation 5, 716
Medtech Surgical 727
National Association of Epilepsy Centers 637
NINDS-National Institute of Neurological Disorders an Stroke 921
Nutricia 142
ROW Foundation 3

Patient Advocacy Group
The Anita Kaufmann Foundation 11
Bridge the Gap - SYNGAP Education and Research Foundation 25
Child Neurology Foundation 7
Citizens United for Research in Epilepsy (CURE) 21
Dravet Syndrome Foundation 2
Epilepsy Foundation SUDEP Institute and North American SUDEP Registry 9
Epilepsy Foundation Texas - Houston/Dallas-Fort Worth/West Texas 108
The FamilieSCN2a Foundation 15
Glut1 Deficiency Foundation 4
Hope for Hypothalamic Hamartoma 8
Hope4Harper 22
The Jack Pribaz Foundation 13
LGS Foundation 6
Tuberous Sclerosis Alliance 23

Pharmaceutical
Aprecia Pharmaceuticals Company 733
BioMarin Pharmaceutical 100, 105
Eisai Inc. 501
GW Pharmaceuticals 421
Lundbeck 601
Mallinckrodt Pharmaceuticals 104, 911
OWP Pharmaceuticals, Inc. 917
Peachtree BioResearch Solutions 228
Sage Therapeutics 918
Sun Neurosciences 433
Sunovion Pharmaceuticals Inc. 800
Supernus Pharmaceuticals, Inc. 301
UCB, Inc. 401
Upsher-Smith Laboratories, Inc. 711, 720

Practice Services
Global Neuro-Diagnostics, LP 635
Perfect Phorm, LLC 132

Professional Society
American Clinical Magnetoencephalography Society (ACMEGS) 19
American Clinical Neurophysiology Society (ACNS) 18
American Epilepsy Society 326
ASET - The Neurodiagnostic Society 14
The North American AED Pregnancy Registry 336

Publications
ASET - The Neurodiagnostic Society 14
Child Neurology Foundation 7
Demos Medical Publishing 924
John Libbey EUROTEXT 226
Neurology Reviews 208
NINDS-National Institute of Neurological Disorders and Stroke 921
Oxford University Press 229
Practical Neurology 831
Taylor & Francis 833
Wiley 416
Wolters Kluwer Health 227

Recruiter
Penn State Hershey Medical Center 843
RosmanSearch 718

Research Aids
Blackrock Neuromed, LLC 527
Empatica 837
Epilepsy Foundation SUDEP Institute and North American SUDEP Registry 9
NINDS-National Institute of Neurological Disorders and Stroke 921
The North American AED Pregnancy Registry 336
Peachtree BioResearch Solutions 228

Seizure Detection
Cadwell Industries, Inc. 521
Compumedics 427
DigiTrace EEG Services 117
Electrical Geodesics, Inc. 418
emka TECHNOLOGIES Inc. 832
Empatica 837
Evogen Precision Medicine 341
MVAP Medical Supplies, Inc. 113
NeuroPace, Inc. 213
Nihon Kohden America, Inc. 413
Perfect Phorm, LLC 132
Persyst Development Corporation 321
Pinnacle Technology, Inc. 740
Rhythmlink International, LLC 241
RSC 110
Smart Monitor Corp. 736

Software
ANT North America 340
emka TECHNOLOGIES Inc. 832
FHC, Inc. 827
Monteris Medical 212
MRI Interventions 216
Neuralynx, Inc. 412
Persyst Development Corporation 321
SeizureTracker.com 20

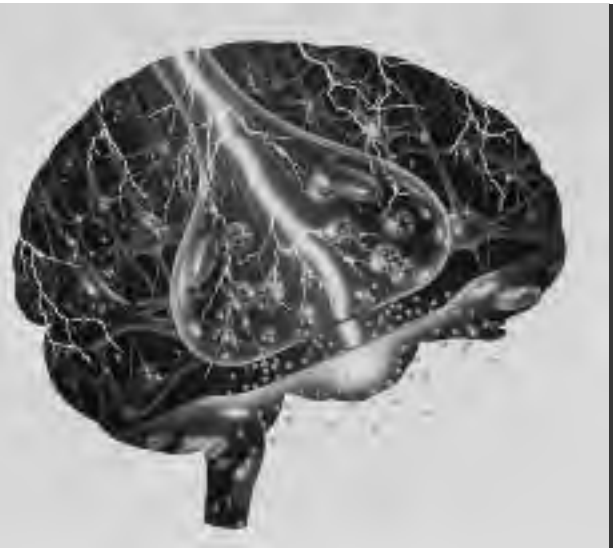
Surgical Tools
Monteris Medical 212

Testing
Alliance Family of Companies 841
GeneDx 719
MediMax Tech, Inc. 106
Natus Neurology Incorporated 627

Video Tools
captureproof 237
Child Neurology Foundation 7
Perfect Phorm, LLC 132
Pinnacle Technology, Inc. 740
RSC 110

INDUSTRY-SUPPORTED SYMPOSIUM

NAVIGATING THE SPECTRUM OF DISEASE: EXPERT PERSPECTIVES ON THE RECOGNITION AND MANAGEMENT OF GENERALIZED SEIZURES CME



THURSDAY, DECEMBER 1, 2016

REGISTRATION: 7:00 PM TO 7:30 PM

PROGRAM: 7:30 PM TO 9:00 PM

LOCATION: GEORGE R. BROWN CONVENTION CENTER
1001 AVENIDA DE LAS AMERICAS, HOUSTON, TEXAS 77010

ROOM: CONVENTION CENTER BALLROOM A (LEVEL 3)

Patients with generalized seizures can be challenging to diagnose and manage. Because of the complex nature and possible comorbidities, it can be difficult to develop management strategies for generalized seizures. This symposium's expert lectures and discussion will address these challenges.

AGENDA:

All Seizure Types Are Not the Same: Characteristics of Generalized Seizures
Generalized Seizures in Practice: Common Comorbidities
Personalizing Treatment for Generalized Seizures
Practical Management Considerations for Patients With Generalized Seizures

Tracy A. Glauser, MD
Moderator
Cincinnati, Ohio

Gregory L. Krauss, MD
Baltimore, Maryland

Steve S. Chung, MD
Phoenix, Arizona

Bernhard J. Steinhoff, MD, PhD
Kehl-Kork, Germany

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www.medscape.org/sites/townhall/public/seizures

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AES 2016 // DEC 2-6

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Featured speakers and topics at our Innovation Pavilion:

Saturday, December 3rd
12:30–1:30 pm

- **7 Year outcomes**
  Eric Geller, MD, Saint Barnabas Medical Center

  Barbara Jobst, MD, Dartmouth Hitchcock Medical Center
- **Biomarkers: What does long-term ambulatory ECoG data tell us?**
  Martha Morrell, MD, NeuroPace, Inc., Stanford University

Sunday, December 4th
12:30–1:30 pm

- **Long-term SUDEP data**
  Orrin Devinsky, MD, NYU Langone Medical Center
- **Case presentations and patient selection**
  Michel J. Berg, MD, University of Rochester

  Lawrence Hirsch, MD, Yale University

  Vikram Rao, MD, PhD, University of California, San Francisco

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Important Safety Information

- ONFI is contraindicated in patients with a history of hypersensitivity to the drug or its ingredients. Hypersensitivity reactions have included serious dermatological reactions.
- ONFI causes somnolence and sedation. In clinical trials, somnolence or sedation was reported at all effective doses and was dose-related. In general, somnolence and sedation begin within the first month of treatment and may diminish with continued treatment. Prescribers should monitor patients for somnolence and sedation, particularly with concomitant use of other central nervous system (CNS) depressants. Prescribers should caution patients against engaging in hazardous activities that require mental alertness, such as operating dangerous machinery or motor vehicles, until the effect of ONFI is known.
- ONFI has a CNS depressant effect. Patients should be cautioned against the simultaneous use with other CNS depressant drugs or alcohol, and cautioned that the effects of other CNS depressant drugs or alcohol may be potentiated.
- As with all antiepileptic drugs (AEDs), ONFI should be gradually withdrawn to minimize the risk of precipitating seizures, seizure exacerbation, or status epilepticus. Withdrawal symptoms have been reported following abrupt discontinuation of ONFI; the risk of withdrawal symptoms is greater with higher doses.
- Serious dermatological reactions, including Stevens-Johnson syndrome (SJS) and toxic epidermal necrolysis (TEN), have been reported with ONFI in both children and adults during the post-marketing period. ONFI should be discontinued at the first sign of rash, unless the rash is clearly not drug-related.
- Patients with a history of substance abuse should be under careful surveillance when receiving ONFI or other psychotropic agents because of the predisposition of such patients to habituation and dependence. In clinical trials, cases of dependency were reported following abrupt discontinuation of ONFI. The risk of dependence increases with increasing dose and duration of treatment.
- AEDs, including ONFI, increase the risk of suicidal thoughts or behavior in patients. Patients, their caregivers, and families should be informed of the risk and advised to monitor and report any emergence or worsening of depression, suicidal thoughts or behavior, or any unusual changes in mood or behavior, or thoughts of self-harm. If these symptoms occur, consider whether it may be related to the AED or illness, because epilepsy itself can increase these risks.
- Based on animal data, ONFI may cause fetal harm and should only be used during pregnancy or while nursing if the potential benefit justifies the potential risk.
- The most commonly observed adverse reactions reported in an LGS randomized, double-blind, placebo-controlled, parallel group clinical trial of patients who received clobazam as adjunctive therapy (≥10% in any treatment group and at least 5% greater than placebo, respectively) were somnolence or sedation (32% vs. 15%), somnolence (25% vs. 12%), pyrexia (17% vs. 3%), lethargy (15% vs. 5%), aggression (14% vs. 5%), drooling (14% vs. 3%), irritability (11% vs. 5%), ataxia (10% vs. 3%), and constipation (10% vs. 0%).

Please see Brief Summary of Prescribing Information on the following pages. For full Prescribing Information, Medication Guide, and Instructions for Use go to ONFI.com for more information.

Onfi® (clobazam)℞
10 mg, 20 mg Tablets
2.5 mg/mL Oral Suspension

Ready to Fight

Lundbeck Your Partner in Epilepsy™

ONFI® (clobazam) tablets, for oral use, Ⓒ
ONFI® (clobazam) oral suspension, Ⓒ

Brief Summary of Prescribing Information

(See package insert for full Prescribing Information or visit www.ONFI.com)

Rx Only

INDICATIONS AND USAGE – ONFI® (clobazam) is indicated for the adjunctive treatment of seizures associated with Lennox-Gastaut syndrome (LGS) in patients 2 years of age or older.

CONTRAINDICATIONS – ONFI is contraindicated in patients with a history of hypersensitivity to the drug or its ingredients. Hypersensitivity reactions have included serious dermatological reactions.

WARNINGS AND PRECAUTIONS – Somnolence or Sedation: ONFI causes somnolence and sedation. In clinical trials, somnolence or sedation was reported at all effective doses and was dose-related. In general, somnolence and sedation begin within the first month of treatment and may diminish with continued treatment. Prescribers should monitor patients for somnolence and sedation, particularly with concomitant use of other central nervous system depressants. Prescribers should caution patients against engaging in hazardous activities requiring mental alertness, such as operating dangerous machinery or motor vehicles, until the effect of ONFI is known. **Potentiation of Sedation from Concomitant Use with Central Nervous System Depressants:** Since ONFI has a central nervous system (CNS) depressant effect, patients or their caregivers should be cautioned against simultaneous use with other CNS depressant drugs or alcohol, and cautioned that the effects of other CNS depressant drugs or alcohol may be potentiated. **Withdrawal Symptoms:** Abrupt discontinuation of ONFI should be avoided. ONFI should be tapered by decreasing the dose every week by 5-10 mg/day until discontinuation *[see Dosage and Administration]*. As with all antiepileptic drugs, ONFI should be withdrawn gradually to minimize the risk of precipitating seizures, seizure exacerbation, or status epilepticus. Withdrawal symptoms have been reported following abrupt discontinuance of benzodiazepines. The more severe withdrawal symptoms have usually been limited to patients who received excessive doses over an extended period of time, followed by an abrupt discontinuation. Generally milder withdrawal symptoms have been reported following abrupt discontinuance of benzodiazepines taken continuously at therapeutic doses for several months. **Serious Dermatological Reactions:** Serious skin reactions, including Stevens-Johnson syndrome (SJS) and toxic epidermal necrolysis (TEN), have been reported with ONFI in both children and adults during the post-marketing period. Patients should be closely monitored for signs or symptoms of SJS/TEN, especially during the first 8 weeks of treatment initiation or when re-introducing therapy. ONFI should be discontinued at the first sign of rash, unless the rash is clearly not drug-related. If signs or symptoms suggest SJS/TEN, use of this drug should not be resumed and alternative therapy should be considered *[see Contraindications]*. **Physical and Psychological Dependence:** Patients with a history of substance abuse should be under careful surveillance when receiving ONFI or other psychotropic agents because of the predisposition of such patients to habituation and dependence *[see Drug Abuse and Dependence]*. **Suicidal Behavior and Ideation:** Antiepileptic drugs (AEDs), including ONFI, increase the risk of suicidal thoughts or behavior in patients taking these drugs for any indication. Patients treated with any AED for any indication should be monitored for the emergence or worsening of depression, suicidal thoughts or behavior, and/or any unusual changes in mood or behavior. The increased risk of suicidal thoughts or behavior with AEDs was observed as early as one week after starting drug treatment with AEDs and persisted for the duration of treatment assessed. Because most trials included in the analysis did not extend beyond 24 weeks, the risk of suicidal thoughts or behavior beyond 24 weeks could not be assessed. The risk of suicidal thoughts or behavior was generally consistent among drugs in the data analyzed. The finding of increased risk with AEDs of varying mechanisms of action and across a range of indications suggests that the risk applies to all AEDs used for any indication. The relative risk for suicidal thoughts or behavior was higher in clinical trials for epilepsy than in clinical trials for psychiatric or other conditions, but the absolute risk differences were similar for the epilepsy and psychiatric indications. Anyone considering prescribing ONFI or any other AED must balance the risk of suicidal thoughts or behavior with the risk of untreated illness. Should suicidal thoughts and behavior emerge during treatment, the prescriber needs to consider whether the emergence of these symptoms in any given patient may be related to the illness being treated. Patients, their caregivers, and families should be informed that AEDs increase the risk of suicidal thoughts and behavior and should be advised of the need to be alert for the emergence or worsening of the signs and symptoms of depression, any unusual changes in mood or behavior, or the emergence of suicidal thoughts, behavior, or thoughts about self-harm. Behaviors of concern should be reported immediately to healthcare providers. *[Please see Warnings and Precautions in the full Prescribing Information for complete details.]*

ADVERSE REACTIONS – Because clinical trials are conducted under widely varying conditions, adverse reaction rates observed in the clinical trials of a drug cannot be directly compared to rates in the clinical trials of another drug and may not reflect the rates observed in practice. During its development for the adjunctive treatment of seizures associated with LGS, ONFI was administered to 333 healthy volunteers and 300 patients with a current or prior diagnosis of LGS, including 197 patients treated for 12 months or more. The conditions and duration of exposure varied greatly and included single- and multiple-dose clinical pharmacology studies in healthy volunteers and two double-blind studies in patients with LGS (Study 1 and 2) *[see Clinical Studies]*. Only Study 1 included a placebo group, allowing comparison of adverse reaction rates on ONFI at several doses to placebo. Adverse Reactions Leading to Discontinuation in an LGS Placebo Controlled Clinical Trial (Study 1): The adverse reactions associated with ONFI treatment discontinuation in ≥1% of patients in decreasing order of frequency included lethargy, somnolence, ataxia, aggression, fatigue, and insomnia. Most Common Adverse Reactions in an LGS Placebo Controlled Clinical Trial (Study 1): Table 3 in the full Prescribing Information lists the adverse reactions that occurred in ≥5% of ONFI treated patients (at any dose), and at a rate greater than placebo treated patients, in the randomized, double-blind, placebo-controlled, parallel group clinical study of adjunctive AED therapy for 15 weeks (Study 1).

Table 3. Adverse Reactions Reported for ≥5% of Patients and More Frequently than Placebo in Any Treatment Group

		ONFI Dose Level			
	Placebo N=59 %	Low[a] N=58 %	Medium[b] N=62 %	High[c] N=59 %	All ONFI N=179 %
Gastrointestinal Disorders					
Vomiting	5	9	5	7	7
Constipation	0	2	2	10	5
Dysphagia	0	0	0	5	2
General Disorders and Administration Site Conditions					
Pyrexia	3	17	10	12	13
Irritability	5	3	11	5	7
Fatigue	2	5	5	3	5
Infections and Infestations					
Upper respiratory tract infection	10	10	13	14	12
Pneumonia	2	3	3	7	4
Urinary tract infection	0	2	5	5	4
Bronchitis	0	2	0	5	2
Metabolism and Nutrition Disorders					
Decreased appetite	3	3	0	7	3
Increased appetite	0	2	3	5	3
Nervous System Disorders					
Somnolence or Sedation	15	17	27	32	26
Somnolence	12	16	24	25	22
Sedation	3	2	3	9	5
Lethargy	5	10	5	15	10
Drooling	3	0	13	14	9
Ataxia	3	3	2	10	5
Psychomotor hyperactivity	3	3	3	5	4
Dysarthria	0	2	2	5	3
Psychiatric Disorders					
Aggression	5	3	8	14	8
Insomnia	2	2	5	7	5
Respiratory Disorders					
Cough	0	3	5	7	5

[a] Maximum daily dose of 5 mg for ≤30 kg body weight; 10 mg for >30 kg body weight

[b] Maximum daily dose of 10 mg for ≤30 kg body weight; 20 mg for >30 kg body weight

[c] Maximum daily dose of 20 mg for ≤30 kg body weight; 40 mg for >30 kg body weight

Post Marketing Experience: These reactions are reported voluntarily from a population of uncertain size; therefore, it is not possible to estimate their frequency or establish a causal relationship to drug exposure. Adverse reactions are categorized by system organ class. ***Blood Disorders:*** Anemia, eosinophilia, leukopenia, thrombocytopenia; ***Eye Disorders:*** Diplopia, vision blurred; ***Gastrointestinal Disorders:*** Abdominal distention; ***General Disorders and Administration Site Conditions:*** Hypothermia; ***Investigations:*** Hepatic enzyme increased; ***Musculoskeletal:*** Muscle spasms; ***Psychiatric Disorders:*** Agitation, anxiety, apathy, confusional state, depression, delirium, delusion, hallucination; ***Renal and Urinary Disorders:*** Urinary retention; ***Respiratory Disorders:*** Aspiration, respiratory depression;

Skin and Subcutaneous Tissue Disorders: Rash, urticaria, angioedema, and facial and lip edema.

DRUG INTERACTIONS – Effect of ONFI on Other Drugs: ONFI is a weak CYP3A4 inducer. As some hormonal contraceptives are metabolized by CYP3A4, their effectiveness may be diminished when given with ONFI. Additional non-hormonal forms of contraception are recommended when using ONFI *[see Clinical Pharmacology, Patient Counseling Information]*. ONFI inhibits CYP2D6. Dose adjustment of drugs metabolized by CYP2D6 may be necessary *[see Clinical Pharmacology]*. **Effect of Other Drugs on ONFI:** Strong and moderate inhibitors of CYP2C19 may result in increased exposure to N-desmethylclobazam, the active metabolite of clobazam. This may increase the risk of dose-related adverse reactions. Dosage adjustment of ONFI may be necessary when co-administered with strong CYP2C19 inhibitors (e.g., fluconazole, fluvoxamine, ticlopidine) or moderate CYP2C19 inhibitors (e.g., omeprazole) *[see Clinical Pharmacology]*. **CNS Depressants and Alcohol:** Concomitant use of ONFI with other CNS depressants may increase the risk of sedation and somnolence *[see Warnings and Precautions]*.

USE IN SPECIFIC POPULATIONS – Pregnancy: Pregnancy Category C. Risk Summary: There are no adequate and well-controlled studies in pregnant women. In animal studies, administration of clobazam during pregnancy resulted in developmental toxicity, including increased incidences of fetal malformations, at plasma exposures for clobazam and its major active metabolite, N-desmethylclobazam, below those expected at therapeutic doses in patients. ONFI should be used during pregnancy only if the potential benefit justifies the potential risk to the fetus. Available human data on the risk of teratogenicity associated with benzodiazepines are inconclusive. There is insufficient evidence in humans to assess the effect of benzodiazepine exposure during pregnancy on neurodevelopment. Administration of benzodiazepines immediately prior to or during childbirth can result in a syndrome of hypothermia, hypotonia, respiratory depression, and difficulty feeding. In addition, infants born to mothers who have taken benzodiazepines during the later stages of pregnancy can develop dependence, and subsequently withdrawal, during the postnatal period. Data for other benzodiazepines suggest the possibility of adverse developmental effects (including long-term effects on neurobehavioral and immunological function) in animals following prenatal exposure to benzodiazepines at clinically relevant doses. Data: *Animal* - In a study in which clobazam (150, 450, or 750 mg/kg/day) was orally administered to pregnant rats throughout the period of organogenesis, embryofetal mortality and incidences of fetal skeletal variations were increased at all doses. The low effect dose for embryofetal developmental toxicity in rats (150 mg/kg/day) was associated with plasma exposures (AUC) for clobazam and its major active metabolite, N-desmethylclobazam, lower than those in humans at the maximum recommended human dose (MRHD) of 40 mg/day. Oral administration of clobazam (10, 30, or 75 mg/kg/day) to pregnant rabbits throughout the period of organogenesis resulted in decreased fetal body weights, and increased incidences of fetal malformations (visceral and skeletal) at the mid and high doses, and an increase in embryofetal mortality at the high dose. Incidences of fetal variations were increased at all doses. The highest dose tested was associated with maternal toxicity (ataxia and decreased activity). The low effect dose for embryofetal developmental toxicity in rabbits (10 mg/kg/day) was associated with plasma exposures for clobazam and N-desmethylclobazam lower than those in humans at the MRHD. Oral administration of clobazam (50, 350, or 750 mg/kg/day) to rats throughout pregnancy and lactation resulted in increased embryofetal mortality at the high dose, decreased pup survival at the mid and high doses, and alterations in offspring behavior (locomotor activity) at all doses. The low effect dose for adverse effects on pre- and postnatal development in rats (50 mg/kg/day) was associated with plasma exposures for clobazam and N-desmethylclobazam lower than those in humans at the MRHD. ***Pregnancy Registry:*** To provide information regarding the effects of *in utero* exposure to ONFI, physicians are advised to recommend that pregnant patients taking ONFI enroll in the North American Antiepileptic Drug (NAAED) Pregnancy Registry. This can be done by calling the toll free number 1-888-233-2334, and must be done by patients themselves or their caregiver. Information on the registry can also be found at the website http://www.aedpregnancyregistry.org/. **Nursing Mothers:** ONFI is excreted in human milk. Because of the potential for serious adverse reactions in nursing infants from ONFI, a decision should be made whether to discontinue nursing or to discontinue the drug, taking into account the importance of the drug to the mother. **Pediatric Use:** Safety and effectiveness in patients less than 2 years of age have not been established. In a study in which clobazam (4, 36, or 120 mg/kg/day) was orally administered to rats during the juvenile period of development (postnatal days 14 to 48), adverse effects on growth (decreased bone density and bone length) and behavior (altered motor activity and auditory startle response; learning deficit) were observed at the high dose. The effect on bone density, but not on behavior, was reversible when drug was discontinued. The no-effect level for juvenile toxicity (36 mg/kg/day) was associated with plasma exposures (AUC) to clobazam and its major active metabolite, N-desmethylclobazam, less than those expected at therapeutic doses in pediatric patients. **Geriatric Use:** Clinical studies of ONFI did not include sufficient numbers of subjects aged 65 and over to determine whether they respond differently from younger subjects. However, elderly subjects appear to eliminate clobazam more slowly than younger subjects based on population pharmacokinetic analysis. For these reasons, the initial dose in elderly patients should be 5 mg/day. Patients should be titrated initially according to weight to 10-20 mg/day. Patients may be titrated further to a maximum daily dose of 20 or 40 mg depending on weight, if tolerated *[see Dosage and Administration, Clinical Pharmacology]*. **CYP2C19 Poor Metabolizers:** Concentrations of clobazam's active metabolite, N-desmethylclobazam, are higher in CYP2C19 poor metabolizers than in extensive metabolizers. For this reason, the initial dose in patients known to be CYP2C19 poor metabolizers should be 5 mg/day. Dose titration should proceed slowly according to weight to 10-20 mg/day, and may be titrated further depending on weight to a maximum daily dose of 20 or 40 mg on day 21 based upon clinical response *[see Dosage and Administration, Clinical Pharmacology]*. **Renal Impairment:** The pharmacokinetics of ONFI were evaluated in patients with mild and moderate renal impairment. There were no significant differences in systemic exposure (AUC and C_{max}) between patients with mild or moderate renal impairment and healthy subjects. No dose adjustment is required for patients with mild and moderate renal impairment. There is essentially no experience with ONFI in patients with severe renal impairment or ESRD. It is not known if clobazam or its active metabolite, N-desmethylclobazam, is dialyzable *[see Dosage and Administration, Clinical Pharmacology]*. **Hepatic Impairment:** ONFI is hepatically metabolized; however, there are limited data to characterize the effect of hepatic impairment on the pharmacokinetics of ONFI. For this reason, the initial dose in patients with mild to moderate hepatic impairment (Child-Pugh score 5-9) should be 5 mg/day. These patients should be titrated according to weight to 10-20 mg/day, and may be titrated further depending on weight to a maximum daily dose of 20 or 40 mg on day 21 based upon clinical response. There is inadequate information about metabolism of ONFI in patients with severe hepatic impairment. Therefore no dosing recommendation in those patients can be given *[see Dosage and Administration, Clinical Pharmacology]*.

DRUG ABUSE AND DEPENDENCE – Controlled Substance: ONFI contains clobazam which is a Schedule IV controlled substance. **Abuse:** ONFI can be abused in a similar manner as other benzodiazepines, such as diazepam. The pharmacological profile of ONFI is similar to that of other benzodiazepines listed in Schedule IV of the Controlled Substance Act, particularly in its potentiation of GABAergic transmission through its action on $GABA_A$ receptors, which leads to sedation and somnolence. The World Health Organization epidemiology database contains reports of drug abuse, misuse, and overdoses associated with clobazam. **Dependence:** In clinical trials, cases of dependency were reported following abrupt discontinuation of ONFI. The risk of dependence is present even with use of ONFI at the recommended dose range over periods of only a few weeks. The risk of dependence increases with increasing dose and duration of treatment. The risk of dependence is increased in patients with a history of alcohol or drug abuse. *Withdrawal:* Abrupt discontinuation of ONFI causes withdrawal symptoms. As with other benzodiazepines, ONFI should be withdrawn gradually *[see Dosage and Administration, Warnings and Precautions]*. In ONFI clinical pharmacology trials in healthy volunteers, the most common withdrawal symptoms after abrupt discontinuation were headache, tremor, insomnia, anxiety, irritability, drug withdrawal syndrome, palpitations, and diarrhea *[see Warnings and Precautions]*. Other withdrawal reactions to clobazam reported in the literature include restlessness, panic attacks, profuse sweating, difficulty in concentrating, nausea and dry retching, weight loss, blurred vision, photophobia, and muscle pain and stiffness. In general, benzodiazepine withdrawal may cause seizures, psychosis, and hallucinations *[see Warnings and Precautions]*.

OVERDOSAGE – Signs and Symptoms of Overdosage: Overdose and intoxication with benzodiazepines, including ONFI, may lead to CNS depression, associated with drowsiness, confusion and lethargy, possibly progressing to ataxia, respiratory depression, hypotension, and, rarely, coma or death. The risk of a fatal outcome is increased in cases of combined poisoning with other CNS depressants, including alcohol. **Management of Overdosage:** The management of ONFI overdose may include gastric lavage and/or administration of activated charcoal, intravenous fluid replenishment, early control of airway and general supportive measures, in addition to monitoring level of consciousness and vital signs. Hypotension can be treated by replenishment with plasma substitutes and, if necessary, with sympathomimetic agents. The efficacy of supplementary administration of physostigmine (a cholinergic agent) or of flumazenil (a benzodiazepine antagonist) in ONFI overdose has not been assessed. The administration of flumazenil in cases of benzodiazepine overdose can lead to withdrawal and adverse reactions. Its use in patients with epilepsy is typically not recommended.

Lundbeck
Deerfield, IL 60015, U.S.A.

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CLB-L-00009

BANZEL®
(rufinamide)

BANZEL (rufinamide) BRIEF SUMMARY—See package insert for full Prescribing Information

Dosage Information

Pediatric patients (1 year to less than 17 years)

The recommended starting daily dose of BANZEL in pediatric patients with Lennox-Gastaut Syndrome is approximately 10 mg/kg/day administered in two equally divided doses. The dose should be increased by approximately 10 mg/kg increments every other day until a maximum daily dose of 45 mg/kg/day, not to exceed 3200 mg/day, administered in two equally divided doses, is reached. It is not known whether doses lower than the target doses are effective.

Adults (17 years and older)

The recommended starting daily dose of BANZEL in adults with Lennox-Gastaut Syndrome is 400 to 800 mg per day administered in two equally divided doses. The dose should be increased by 400–800 mg every other day until a maximum daily dose of 3200 mg, administered in two equally divided doses, is reached. It is not known whether doses lower than 3200 mg are effective.

Administration Information

Administer BANZEL with food. BANZEL film-coated tablets can be administered whole, as half tablets or crushed.

BANZEL oral suspension should be shaken well before every administration. The provided adapter and calibrated oral dosing syringe should be used to administer the oral suspension. The adapter which is supplied in the product carton should be inserted firmly into the neck of the bottle before use and remain in place for the duration of the usage of the bottle. The dosing syringe should be inserted into the adapter and the dose withdrawn from the inverted bottle. The cap should be replaced after each use. The cap fits properly when the adapter is in place.

Dosing in Patients Undergoing Hemodialysis

Hemodialysis may reduce exposure by a limited (about 30%) extent. Accordingly, adjusting the BANZEL dose during the dialysis process should be considered.

Dosing in Patients with Hepatic Disease

Use of BANZEL in patients with hepatic impairment has not been studied. Therefore, use in patients with severe hepatic impairment is not recommended. Caution should be exercised in treating patients with mild to moderate hepatic impairment.

Dosing in Patients Taking Valproate

Patients taking valproate should begin BANZEL at a dose lower than 10 mg/kg per day in pediatric patients or 400 mg per day in adults.

CONTRAINDICATIONS

BANZEL is contraindicated in patients with Familial Short QT syndrome.

WARNINGS AND PRECAUTIONS

Suicidal Behavior and Ideation

Antiepileptic drugs (AEDs), including BANZEL, increase the risk of suicidal thoughts or behavior in patients taking these drugs for any indication. Patients treated with any AED for any indication should be monitored for the emergence or worsening of depression, suicidal thoughts or behavior, and/or any unusual changes in mood or behavior.

Pooled analyses of 199 placebo-controlled clinical trials (mono- and adjunctive therapy) of 11 different AEDs showed that patients randomized to one of the AEDs had approximately twice the risk (adjusted Relative Risk 1.8, 95% CI:1.2, 2.7) of suicidal thinking or behavior compared to patients randomized to placebo. In these trials, which had a median treatment duration of 12 weeks, the estimated incidence rate of suicidal behavior or ideation among 27,863 AED-treated patients was 0.43%, compared to 0.24% among 16,029 placebo-treated patients, representing an increase of approximately one case of suicidal thinking or behavior for every 530 patients treated. There were four suicides in drug-treated patients in the trials and none in placebo-treated patients, but the number is too small to allow any conclusion about drug effect on suicide.

The increased risk of suicidal thoughts or behavior with AEDs was observed as early as one week after starting drug treatment with AEDs and persisted for the duration of treatment assessed. Because most trials included in the analysis did not extend beyond 24 weeks, the risk of suicidal thoughts or behavior beyond 24 weeks could not be assessed.

The risk of suicidal thoughts or behavior was generally consistent among drugs in the data analyzed. The finding of increased risk with AEDs of varying mechanisms of action and across a range of indications suggests that the risk applies to all AEDs used for any indication. The risk did not vary substantially by age (5-100 years) in the clinical trials analyzed. Table 1 shows absolute and relative risk by indication for all evaluated AEDs.

Table 1: Absolute and Relative Risk of Suicidal Behavior and Ideation

Indication	Placebo Patients with Events Per 1000 Patients	Drug Patients with Events Per 1000 Patients	Relative Risk: Incidence of Events in Drug Patients/ Incidence in Placebo Patients	Risk Difference: Additional Drug Patients with Events Per 1000 Patients
Epilepsy	1.0	3.4	3.5	2.4
Psychiatric	5.7	8.5	1.5	2.9
Other	1.0	1.8	1.9	0.9
Total	2.4	4.3	1.8	1.9

The relative risk for suicidal thoughts or behavior was higher in clinical trials for epilepsy than in clinical trials for psychiatric or other conditions, but the absolute risk differences were similar for the epilepsy and psychiatric indications.

Anyone considering prescribing BANZEL or any other AED must balance the risk of suicidal thoughts or behavior with the risk of untreated illness. Epilepsy and many other illnesses for which AEDs are prescribed are themselves associated with morbidity and mortality and an increased risk of suicidal thoughts and behavior. Should suicidal thoughts and behavior emerge during treatment, consider whether the emergence of these symptoms in any given patient may be related to the illness being treated.

Patients, their caregivers, and families should be informed that AEDs increase the risk of suicidal thoughts and behavior and should be advised of the need to be alert for the emergence or worsening of the signs and symptoms of depression, any unusual changes in mood or behavior, or the emergence of suicidal thoughts, behavior, or thoughts about self-harm. Behaviors of concern should be reported immediately to healthcare providers.

Central Nervous System Reactions

Use of BANZEL has been associated with central nervous system-related adverse reactions in the controlled clinical trial of patients 4 years or older with Lennox-Gastaut Syndrome. The most significant of these can be classified into two general categories: 1) somnolence or fatigue, and 2) coordination abnormalities, dizziness, gait disturbances, and ataxia.

Somnolence was reported in 24% of BANZEL-treated patients compared to 13% of patients on placebo, and led to study discontinuation in 3% of BANZEL-treated patients compared to 0% of patients on placebo. Fatigue was reported in 10% of BANZEL-treated patients compared to 8% of patients on placebo patients. It led to study discontinuation in 1% of BANZEL-treated patients and 0% of patients on placebo patients.

Dizziness was reported in 2.7% of BANZEL-treated patients compared to 0% of patients on placebo, and did not lead to study discontinuation.

Ataxia and gait disturbance were reported in 5.4% and 1.4% of BANZEL-treated patients, respectively, compared to no patients on placebo. None of these reactions led to study discontinuation.

Accordingly, patients should be advised not to drive or operate machinery until they have gained sufficient experience on BANZEL to gauge whether it adversely affects their ability to drive or operate machinery.

QT Shortening

Formal cardiac ECG studies demonstrated shortening of the QT interval (mean = 20 msec, for doses ≥2400 mg twice daily) with BANZEL. In a placebo-controlled study of the QT interval, a higher percentage of BANZEL-treated subjects (46% at 2400 mg, 46% at 3200 mg, and 65% at 4800 mg) had a QT shortening of greater than 20 msec at T_{max} compared to placebo (5-10%).

Reductions of the QT interval below 300 msec were not observed in the formal QT studies with doses up to 7200 mg per day. Moreover, there was no signal for drug-induced sudden death or ventricular arrhythmias.

The degree of QT shortening induced by BANZEL is without any known clinical risk. Familial Short QT syndrome is associated with an increased risk of sudden death and ventricular arrhythmias, particularly ventricular fibrillation. Such events in this syndrome are believed to occur primarily when the corrected QT interval falls below 300 msec. Non-clinical data also indicate that QT shortening is associated with ventricular fibrillation.

Patients with Familial Short QT syndrome should not be treated with BANZEL. Caution should be used when administering BANZEL with other drugs that shorten the QT interval.

Multi-organ Hypersensitivity Reactions

Multi-organ hypersensitivity syndrome, a serious condition sometimes induced by antiepileptic drugs, has occurred in association with BANZEL therapy in clinical trials. One patient experienced rash, urticaria, facial edema, fever, elevated eosinophils, stuporous state, and severe hepatitis, beginning on day 29 of BANZEL therapy and extending over a course of 30 days of continued BANZEL therapy with resolution 11 days after discontinuation. Additional possible cases presented with rash and one or more of the following: fever, elevated liver function studies, hematuria, and lymphadenopathy. These cases occurred in pediatric patients less than 12 years of age, within 4 weeks of treatment initiation, and were noted to resolve and/or improve upon BANZEL discontinuation. The syndrome has been reported with other anticonvulsants and typically, although not exclusively, presents with fever and rash associated with other organ system involvement. Because this disorder is variable in its expression, other organ system signs and symptoms not noted here may occur. If this reaction is suspected, BANZEL should be discontinued and alternative treatment started. In addition, rare cases of DRESS (Drug Reaction with Eosinophilia and Systemic Symptoms) and Stevens-Johnson syndrome have been reported in association with rufinamide therapy post marketing. If an antiepileptic drug hypersensitivity syndrome is suspected, rufinamide should be discontinued and alternative treatment started.

All patients who develop a rash while taking BANZEL must be closely supervised.

Withdrawal of AEDs

As with all antiepileptic drugs, BANZEL should be withdrawn gradually to minimize the risk of precipitating seizures, seizure exacerbation, or status epilepticus. If abrupt discontinuation of the drug is medically necessary, the transition to another AED should be made under close medical supervision. In clinical trials, BANZEL discontinuation was achieved by reducing the dose by approximately 25% every 2 days.

Status Epilepticus

Estimates of the incidence of treatment emergent status epilepticus among patients treated with BANZEL are difficult because standard definitions were not employed. In a controlled Lennox-Gastaut Syndrome trial, 3 of 74 (4.1%) BANZEL-treated patients had episodes that could be described as status epilepticus in the BANZEL-treated patients compared with none of the 64 patients in the placebo-treated patients. In all controlled trials that included patients with different epilepsies, 11 of 1240 (0.9%) BANZEL-treated patients had episodes that could be described as status epilepticus compared with none of 635 patients in the placebo-treated patients.

Leukopenia

BANZEL has been shown to reduce white cell count. Leukopenia (white cell count < 3X10⁹ L) was more commonly observed in BANZEL-treated patients 43 of 1171 (3.7%) than placebo-treated patients, 7 of 579 (1.2%) in all controlled trials.

ADVERSE REACTIONS

The following serious adverse reactions are described below and elsewhere in the labeling:

- Suicidal Behavior and Ideation
- Central Nervous System Reactions
- QT Shortening
- Multi-organ Hypersensitivity Reactions
- Leukopenia

Clinical Trials Experience

Because clinical trials are conducted under widely varying conditions, adverse reaction rates observed in the clinical trials of a drug cannot be directly compared to rates in the clinical trials of another drug and may not reflect the rates observed in practice.

Adverse Reactions in Adult and Pediatric Patients ages 4 to 17 years of age

In the pooled, double-blind, adjunctive therapy studies in adult and pediatric patients (age 4 to 17 years of age), the most common (≥10%) adverse reactions in BANZEL-treated patients, in all doses studied (200 to 3200 mg per day), with a higher frequency than in patients on placebo were: headache, dizziness, fatigue, somnolence, and nausea.

Table 2 lists adverse reactions that occurred in at least 3% of pediatric patients (ages 4 to less than 17 years) with epilepsy treated with BANZEL in controlled adjunctive studies and were numerically more common in patients treated with BANZEL than in patients on placebo.

At the target dose of 45 mg/kg per day for adjunctive therapy in pediatric patients (ages 4 to less than 17 years), the most common (≥3%) adverse reactions with an incidence greater than in placebo for BANZEL were somnolence, vomiting, and headache.

Table 2: Adverse Reactions in Pediatric Patients (Ages 4 to less than 17 years) in Pooled Double-Blind Adjunctive Trials

Adverse Reaction (BANZEL [N=187] vs Placebo [N=182])
Somnolence (17% vs 9%), Vomiting (17% vs 7%), Headache (16% vs 8%), Fatigue (9% vs 8%), Dizziness (8% vs 6%), Nausea (7% vs 3%), Influenza (5% vs 4%), Nasopharyngitis (5% vs 3%), Decreased appetite (5% vs 1%), Rash (4% vs 2%), Ataxia (4% vs 1%), Diplopia (4% vs 1%), Bronchitis (3% vs 2%), Sinusitis (3% vs 2%), Psychomotor hyperactivity (3% vs 1%), Upper abdominal pain (3% vs 2%), Aggression (3% vs 2%), Ear infection (3% vs 1%), Disturbance in attention (3% vs 1%), and Pruritus (3% vs 0%)

Table 3 lists adverse reactions that occurred in at least 3% of adult patients with epilepsy treated with BANZEL (up to 3200 mg/day) in adjunctive controlled studies and were numerically more common in patients treated with BANZEL than in patients on placebo. In these studies, either BANZEL or placebo was added to current AED therapy.

At all doses studied of up to 3200 mg per day given as adjunctive therapy in adults, the most common (≥3%) adverse reactions, and with the greatest increase in incidence compared to placebo, for BANZEL were dizziness, fatigue, nausea, diplopia, vision blurred, and ataxia.

Table 3: Adverse Reactions in Adults in Pooled Double-Blind Adjunctive Trials

Adverse Reaction (BANZEL [N=823] vs Placebo [N=376])
Headache (27% vs 26%), Dizziness (19% vs 12%), Fatigue (16% vs 13%), Nausea (12% vs 9%), Somnolence (11% vs 9%), Diplopia (9% vs 3%), Tremor (6% vs 5%), Nystagmus (6% vs 5%), Blurred vision (6% vs 2%), Vomiting (5% vs 4%), Ataxia (4% vs 0%), Upper abdominal pain (3% vs 2%), Anxiety (3% vs 2%), Constipation (3% vs 2%), Dyspepsia (3% vs 2%), Back pain (3% vs 1%), Gait disturbance (3% vs 1%), and Vertigo (3% vs 1%)

Discontinuation in Controlled Clinical Studies

In controlled, double-blind, adjunctive clinical studies, 9% of patients and adult patients receiving BANZEL as adjunctive therapy and 4% receiving placebo discontinued as a result of an adverse reaction. The adverse reactions most commonly leading to discontinuation of BANZEL (>1%) used as adjunctive therapy were generally similar in adults and children.

In pediatric patients (ages 4 to less than 17 years) double-blind adjunctive clinical studies, 8% of patients receiving BANZEL as adjunctive therapy (at the recommended dose of 45 mg/kg per day) and 2% receiving placebo discontinued as a result of an adverse reaction. The adverse reactions most commonly leading to discontinuation of BANZEL (>1%) used as adjunctive therapy are presented in Table 4.

Table 4: Most Common Adverse Reactions Leading to Discontinuation in Pediatric Patients (Ages 4 to less than 17 years) in Pooled Double-Blind Adjunctive Trials

Adverse Reaction (BANZEL [N=187] vs Placebo [N=182])
Somnolence (2% vs 1%), Rash (2% vs 1%), Fatigue (2% vs 0%), and Vomiting (1% vs 2%)

In adult double-blind, adjunctive clinical studies, 10% of patients receiving BANZEL as adjunctive therapy (at doses up to 3200 mg per day) and 6% receiving placebo discontinued as a result of an adverse reaction. The adverse reactions most commonly leading to discontinuation of BANZEL in this adjunctive therapy are presented in Table 5.

Table 5: Most Common Adverse Reactions Leading to Discontinuation in Adult Patients in Pooled Double-Blind Adjunctive Trials

Adverse Reaction (BANZEL [N=823] vs Placebo [N=376])
Dizziness (3% vs 1%), Fatigue (2% vs 1%), Headache (2% vs 1%), Nausea (1% vs 0%), and Ataxia (1% vs 0%)

Pediatric Patients ages 1 to less than 4 years

In a multicenter, parallel group, open-label study comparing BANZEL (45 mg/kg per day) adjunctive treatment (n=25) to any adjunctive treatment with an AED of the investigator's choice (n=11) in pediatric patients 1 year to less than 4 years of age with inadequately controlled Lennox-Gastaut Syndrome, the adverse reaction profile was generally similar to that observed in adults and pediatric patients 4 years of age and older treated with BANZEL. Adverse reactions that occurred in at least 2 (8%) BANZEL-treated patients and with a higher frequency than in the AED comparator group were: vomiting (24%), somnolence (16%), bronchitis (12%), constipation (12%), cough (12%), decreased appetite (12%), rash (12%), otitis media (8%), pneumonia (8%), decreased weight (8%), gastroenteritis (8%), nasal congestion (8%), and pneumonia aspiration (8%).

Other Adverse Reactions Observed During Clinical Trials

BANZEL has been administered to 1978 individuals during all epilepsy clinical trials (placebo-controlled and open-label). Adverse reactions occurring during these studies were recorded by the investigators using terminology of their own choosing. To provide a meaningful estimate of the proportion of patients having adverse reactions, these events were grouped into standardized categories using the MedDRA dictionary. Adverse events occurring at least three times and considered possibly related to treatment are included in the System Organ Class listings below. Terms not included in the listings are those already included in the tables above, those too general to be informative, those related to procedures, and terms describing events common in the population. Some events occurring fewer than 3 times are also included based on their medical significance. Because the reports include events observed in open-label, uncontrolled observations, the role of BANZEL in their causation cannot be reliably determined.

Events are classified by body system and listed in order of decreasing frequency as follows: *frequent* adverse events—those occurring in at least 1/100 patients; *infrequent* adverse events—those occurring in 1/100 to 1/1000 patients; *rare*—those occurring in fewer than 1/1000 patients.

Blood and Lymphatic System Disorders: *Frequent:* anemia. *Infrequent:* lymphadenopathy, leukopenia, neutropenia, iron deficiency anemia, thrombocytopenia.

Cardiac Disorders: *Infrequent:* bundle branch block right, atrioventricular block first degree.

Metabolic and Nutritional Disorders: *Frequent:* decreased appetite, increased appetite.

Renal and Urinary Disorders: *Frequent:* pollakiuria. *Infrequent:* urinary incontinence, dysuria, hematuria, nephrolithiasis, polyuria, enuresis, nocturia, incontinence.

DRUG INTERACTIONS

Effects of BANZEL on Other AEDs:

Population pharmacokinetic analysis of average concentration at steady state of carbamazepine, lamotrigine, phenobarbital, phenytoin, topiramate, and valproate showed that typical rufinamide C_{avss} levels had little effect on the pharmacokinetics of other AEDs. Any effects, when they occur, have been more marked in the pediatric population.

Table 6 summarizes the drug-drug interactions of BANZEL with other AEDs.

Table 6: Summary of drug-drug interactions of BANZEL with other antiepileptic drugs

AED Co-administered	Influence of Rufinamide on AED concentration[a]	Influence of AED on Rufinamide concentration
Carbamazepine	Decrease by 7 to 13%[b]	Decrease by 19 to 26% Dependent on dose of carbamazepine
Lamotrigine	Decrease by 7 to 13%[b]	No Effect
Phenobarbital	Increase by 8 to 13%[b,c]	Decrease by 25 to 46%[b,d] Independent of dose or concentration of phenobarbital
Phenytoin	Increase by 7 to 21%[b,c]	Decrease by 25 to 46%[b,d] Independent of dose or concentration of phenytoin
Topiramate	No Effect	No Effect
Valproate	No Effect	Increase by <16 to 70%[b,c] Dependent on concentration of valproate
Primidone	Not investigated	Decrease by 25 to 46%[b,d] Independent of dose or concentration of primidone
Benzodiazepines[e]	Not investigated	No Effect

a) Predictions are based on BANZEL concentrations at the maximum recommended dose of BANZEL.

b) Maximum changes predicted to be in pediatric patients and in adult patients who achieve significantly higher levels of BANZEL, as the effect of rufinamide on these AEDs is concentration-dependent.

c) Larger effects in pediatric patients at high doses/concentrations of AEDs.

d) Phenobarbital, primidone and phenytoin were treated as a single covariate (phenobarbital-type inducers) to examine the effect of these agents on BANZEL clearance.

e) All compounds of the benzodiazepine class were pooled to examine for 'class effect' on BANZEL clearance.

Phenytoin: The decrease in clearance of phenytoin estimated at typical levels of rufinamide (C_{avss} 15 μg/mL) is predicted to increase plasma levels of phenytoin by 7 to 21%. As phenytoin is known to have non-linear pharmacokinetics (clearance becomes saturated at higher doses), it is possible that exposure will be greater than the model prediction.

Effects of Other AEDs on BANZEL

Potent cytochrome P450 enzyme inducers, such as carbamazepine, phenytoin, primidone, and phenobarbital, appear to increase the clearance of BANZEL (see Table 6). Given that the majority of clearance of BANZEL is via a non-CYP-dependent route, the observed decreases in blood levels seen with carbamazepine, phenytoin, phenobarbital, and primidone are unlikely to be entirely attributable to induction of a P450 enzyme. Other factors explaining this interaction are not understood. Any effects, when they occurred, were likely to be more marked in the pediatric population.

Valproate

Patients stabilized on BANZEL before being prescribed valproate should begin valproate therapy at a low dose, and titrate to a clinically effective dose. Similarly, patients on valproate should begin at a BANZEL dose lower than 10 mg/kg per day (pediatric patients) or 400 mg per day (adults).

Effects of BANZEL on Hormonal Contraceptives

Female patients of childbearing age should be warned that the concurrent use of BANZEL with hormonal contraceptives may render this method of contraception less effective. Additional non-hormonal forms of contraception are recommended when using BANZEL.

USE IN SPECIFIC POPULATIONS

Pregnancy

Pregnancy Category C

There are no adequate and well-controlled studies in pregnant women. BANZEL should be used during pregnancy only if the potential benefit justifies the potential risk to the fetus. Rufinamide produced developmental toxicity when administered orally to pregnant animals at clinically relevant doses.

Rufinamide was administered orally to rats at doses of 20, 100, and 300 mg/kg per day and to rabbits at doses of 30, 200, and 1000 mg/kg per day during the period of organogenesis (implantation to closure of the hard palate); the high doses are associated with plasma AUCs ~2 times the human plasma AUC at the maximum recommended human dose (MRHD, 3200 mg per day). Decreased fetal weights and increased incidences of fetal skeletal abnormalities were observed in rats at doses associated with maternal toxicity. In rabbits, embryo-fetal death, decreased fetal body weights, and increased incidences of fetal visceral and skeletal abnormalities occurred at all but the low dose. The highest dose tested in rabbits was associated with abortion. The no-effect doses for adverse effects on rat and rabbit embryo-fetal development (20 and 30 mg/kg per day, respectively) were associated with plasma AUCs ~0.2 times that in humans at the MRHD.

In a rat pre- and postnatal development study (dosing from implantation through weaning) conducted at oral doses of 5, 30, and 150 mg/kg per day (associated with plasma AUCs up to ~1.5 times that in humans at the MRHD), decreased offspring growth and survival were observed at all doses tested. A no-effect dose for adverse effects on pre- and postnatal development was not established. The lowest dose tested was associated with plasma AUC < 0.1 times that in humans at the MRHD.

Pregnancy Registry

To provide information regarding the effects of in utero exposure to BANZEL, physicians are advised to recommend that pregnant patients taking BANZEL enroll in the North American Antiepileptic Drug Pregnancy Registry. This can be done by calling the toll free number 1-888-233-2334, and must be done by patients themselves. Information on the registry can also be found at the website https://www.aedpregnancyregistry.org.

Nursing Mothers

Rufinamide is likely to be excreted in human milk. Because of the potential for serious adverse reactions in nursing infants from BANZEL, a decision should be made whether to discontinue nursing or discontinue the drug taking into account the importance of the drug to the mother.

Pediatric Use

Safety and effectiveness have been established in pediatric patients 1 to 17 years of age. The effectiveness of BANZEL in pediatric patients 4 years of age and older was based upon an adequate and well-controlled trial of BANZEL that included both adults and pediatric patients 4 years of age and older with Lennox-Gastaut Syndrome. The effectiveness in patients 1 to less than 4 years was based upon a bridging pharmacokinetic and safety study. The pharmacokinetics of rufinamide in the pediatric patients, ages 1 to less than 4 years of age is similar to children older than 4 years of age and adults.

Safety and effectiveness in pediatric patients below the age of 1 year has not been established.

Geriatric Use

Clinical studies of BANZEL did not include sufficient numbers of subjects aged 65 and over to determine whether they respond differently from younger subjects. In general, dose selection for an elderly patient should be cautious, usually starting at the low end of the dosing range, reflecting the greater frequency of decreased hepatic, renal, or cardiac function, and of concomitant disease or other drug therapy.

Pharmacokinetics of rufinamide in the elderly are similar to that in the young subjects.

Renal Impairment

Rufinamide pharmacokinetics in patients with severe renal impairment (creatinine clearance < 30 mL/min) was similar to that of healthy subjects. Dose adjustment in patients undergoing dialysis should be considered.

Hepatic Impairment

Use of BANZEL in patients with severe hepatic impairment (Child-Pugh score 10 to 15) is not recommended. Caution should be exercised in treating patients with mild (Child-Pugh score 5 to 6) to moderate (Child-Pugh score 7 to 9) hepatic impairment.

OVERDOSAGE

Because strategies for the management of overdose are continually evolving, it is advisable to contact a Certified Poison Control Center to determine the latest recommendations for the management of an overdose of any drug.

One overdose of 7200 mg per day BANZEL was reported in an adult during the clinical trials. The overdose was associated with no major signs or symptoms, no medical intervention was required, and the patient continued in the study at the target dose.

Treatment or Management of Overdose: There is no specific antidote for overdose with BANZEL. If clinically indicated, elimination of unabsorbed drug should be attempted by induction of emesis or gastric lavage. Usual precautions should be observed to protect the airway. General supportive care of the patient is indicated including monitoring of vital signs and observation of the clinical status of the patient.

Hemodialysis: Standard hemodialysis procedures may result in limited clearance of rufinamide. Although there is no experience to date in treating overdose with hemodialysis, the procedure may be considered when indicated by the patient's clinical state.

BAN20630

For the adjunctive treatment of seizures **associated with LGS in adults and children,**

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- 32.7% median reduction of total seizures in the BANZEL® group vs 11.7% for placebo ($P<0.002$)*[1,2]
- 42.5% median reduction in tonic-atonic seizures (drop attacks) in the BANZEL® group vs 1.4% increase for placebo ($P<0.0001$)*[1,2]

Learn more at Booth 501

*A 12-week, randomized, double-blind, multicenter, placebo-controlled, parallel-group trial to assess the effectiveness of BANZEL (rufinamide) to reduce inadequately controlled seizures associated with LGS in patients (N=138, intent to treat) being treated with 1-3 concomitant, stable-dose AEDs. These were primary efficacy endpoints in the pivotal trial.[1-3]

Indication:

BANZEL® is indicated for the adjunctive treatment of seizures associated with Lennox-Gastaut Syndrome (LGS) in pediatric patients 1 year of age and older, and in adults.

Important Safety Information

Contraindication:

BANZEL is contraindicated in patients with Familial Short QT syndrome.

Warnings:

AEDs increase the risk of suicidal thoughts or behavior in patients. Patients, their caregivers, and families should be informed of the risk and advised to monitor and report any emergence or worsening of depression, suicidal thoughts or behavior, or any unusual changes in mood or behavior, or thoughts of self-harm. If these symptoms occur, consider if it may be related to the AED or illness because epilepsy itself can increase these risks.

Use of BANZEL has been associated with central nervous system–related adverse reactions, such as somnolence or fatigue, coordination abnormalities, dizziness, gait disturbances, and ataxia.

Precautions:

Formal cardiac ECG studies demonstrated shortening of the QT interval (mean = 20 msec, for doses ≥ 2400 mg twice daily) with BANZEL. Caution should be used when administering BANZEL with other drugs that shorten the QT interval.

Multi-organ hypersensitivity syndrome has been reported in association with BANZEL therapy. In clinical trials, hypersensitivity reactions occurred in children less than 12 years of age and within 4 weeks of starting BANZEL therapy. In addition, rare cases of Drug Reaction with Eosinophilia and Systemic Symptoms and Stevens-Johnson syndrome have been reported in association with rufinamide therapy post marketing. If any of these reactions are suspected, BANZEL should be discontinued and alternative treatment started. All patients who develop a rash while taking BANZEL must be closely supervised.

As with all AEDs, BANZEL should be gradually withdrawn to minimize the risk of increased seizure frequency.

Adverse reactions:

In the pooled, double-blind, adjunctive therapy studies in adults and pediatric patients ages 4 and older, the most commonly observed (≥10%) adverse reactions with BANZEL vs placebo, respectively, were headache (25% vs 20%), dizziness (17% vs 10%), fatigue (15% vs 9%), somnolence (13% vs 9%), and nausea (11% vs 7%).

In a multicenter, parallel group, open-label study in pediatric patients (1 year to less than 4 years of age) the most commonly observed (≥10%) adverse reactions and with a higher frequency with BANZEL vs any other AED, respectively, were vomiting (24% vs 9%), somnolence (16% vs 0%), constipation (12% vs 9%), cough (12% vs 9%), bronchitis (12% vs 0%), rash (12% vs 9%), and decreased appetite (12% vs 9%).

The material in this booth is based on the US Prescribing Information and may not be consistent with prescribing information in other countries

For more information, please visit www.BANZEL.com/hcp

References: 1. Glauser et al. Rufinamide for generalized seizures associated with Lennox-Gastaut syndrome. *Neurology*. 2008;70(21):1950-1958. **2.** BANZEL® (rufinamide) prescribing information, Eisai Inc.

Please see Brief Summary of full Prescribing Information on the adjacent pages.

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