

Highlight in Epilepsy 2011

รศ.นพ. คณิตพงษ์ ปราบพาล
หน่วยประสาทวิทยา ภาควิชาอายุรศาสตร์
คณะแพทยศาสตร์ ม. สงขลานครินทร์

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- Thailand vs. Other country

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Pharmacotherapy

New onset epilepsy

Refractory epilepsy

Status epilepticus

Conventional AED

New AED

Conventional AED

New AED

Surgery

New AED

Stimulants

Stimulation

Refractory
epilepsy

Pharmacotherapy

Epileptic surgery

1. Morrell et al. Responsive cortical stimulation for the treatment of medically intractable partial epilepsy. *Neurology* 2011; 77: 1295-304

- French et al. Lamotrigine XR conversion to monotherapy: first study using a historical control group. *Neurotherapeutics* 2012; 9: 176-84.
- Ng et al. Randomized, phase III study results of clobazam in LGS. *Neurology* 2011; 77: 1473-81.
- Zhang et al. Topiramate as an adjunctive treatment for refractory partial epilepsy in the elderly. *J Int Med Res* 2011; 39: 408-15.
- Verrotti et al. Efficacy of rufinamide in drug-resistant epilepsy: a meta-analysis. *Pediatr Neurol* 2011; 44: 347-9.
- French et al. Randomized, double-blind, placebo-controlled trial of ezogabine (retigabine) in partial epilepsy. *Neurology* 2011; 76: 1556-63.
- Lu et al. Efficacy and safety of adjunctive zonisamide in adult patients with refractory partial-onset epilepsy: a randomized, double-blind, placebo-controlled trial. *Clin Drug Invest* 2011; 31: 221-9.
- Blton et al. A randomized, double-blind, placebo-controlled, parallel-group study of rufinamide as adjunctive therapy for refractory partial-onset seizure. *Epilepsia* 2011; 52: 234-42.

1. Engel et al. Early surgical therapy for drug-resistant temporal lobe epilepsy: a randomized trial. *JAMA* 2012; 307: 922-30.

Status
epilepticus

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- Navarro et al. A prehospital randomized trial in convulsive status epilepticus. *Epilepsia* 2011; Suppl 8: 48-9.
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New onset epilepsy

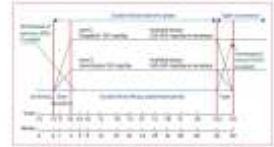
1. Kwan et al. Efficacy and safety of pregabalin versus lamotrigine in patients with newly diagnosed partial seizure: a phase 3, double-blind, randomised, parallel-group trial. *Lancet Neurol* 2011; 10: 881-90.
2. Fattore et al. A multicenter, randomized, placebo-controlled trial of levetiracetam in children and adolescents with newly diagnosed absence epilepsy. *Epilepsia* 2011; 52: 802-9.

Efficacy and safety of pregabalin versus lamotrigine in patients with newly diagnosed partial seizures: a phase 3, double-blind, randomised, parallel-group trial

Lancet Neurol 2011; 10: 881-90

Newly diagnosed epilepsy (> 16 yrs) with seizure ≥ 2 in 12 months
Europe and Asia
Pregabalin 150 mg/d VS. Lamotrigine 100 mg/d

Primary endpoint
proportion of patients who remained seizure-free for ≥ 6 months
Secondary endpoint
time to exit because of lack of efficacy (adverse event, any reason)
time to first seizure after dose-escalation phase
number of seizures during the dose-escalation
monthly seizure frequency
HAI
MOS-Sleep (Medical Outcome Sleep Study Scale)
Adverse event
314/330 in pregabalin ; 308/330 in lamotrigine



Efficacy and safety of pregabalin versus lamotrigine in patients with newly diagnosed partial seizures: a phase 3, double-blind, randomised, parallel-group trial

Lancet Neurol 2011; 10: 881-90

	Treatment difference estimate (pregabalin-lamotrigine; 95% CI)	Risk ratio* (95% CI)
Primary endpoint		
All months of seizure freedom	-0.18 (-0.33 to -0.03)	
Secondary endpoints		
During the efficacy assessment phase†		
Time to exit because of lack of efficacy	0.22 (0.03 to 0.41)	
Time to exit for any reason	0.06 (0.02 to 0.10)	
Time to first seizure	0.02 (0.00 to 0.04)	
Time to 6 months seizure freedom	0.28 (0.00 to 0.56)	
Up to the end of the efficacy assessment phase (including the dose-escalation phase)†		
Time to exit because of lack of efficacy	0.08 (0.05 to 0.14)	
Time to exit for any reason	0.29 (0.08 to 0.50)	

*No comparison of pregabalin to lamotrigine is risk ratio greater than 1, which means the event is more likely to occur in the pregabalin group than in the lamotrigine group; reference is set equal to 1, which means that the event is equally likely to occur in lamotrigine. †Pregabalin, n=330; lamotrigine, n=330. *Pregabalin, n=330; lamotrigine, n=330.

†Table 3: Summary of results for predefined primary and secondary endpoints

Efficacy and safety of pregabalin versus lamotrigine in patients with newly diagnosed partial seizures: a phase 3, double-blind, randomised, parallel-group trial

Lancet Neurol 2011; 10: 881-90

Conclusions

Interpretation: Pregabalin has similar tolerability but seems to have inferior efficacy to lamotrigine for the treatment of newly diagnosed partial seizures in adults. Inferior efficacy of pregabalin might have been attributable to limitations in the study design, as treatment doses might have not been optimized adequately or early enough.

Role of the funding source:
The sponsor designed the study and provided assistance to select participants; data were collected by the investigators and held and analysed by the sponsor. The corresponding author had full access to all the data and had final responsibility for the decisions to submit for publication.

Interpretation: Pregabalin has similar tolerability but seems to have inferior efficacy to lamotrigine for the treatment of newly diagnosed partial seizures in adults. Inferior efficacy of pregabalin might have been attributable to limitations in the study design, as treatment doses might have not been optimized adequately or early enough.

Funding:

Refractory epilepsy

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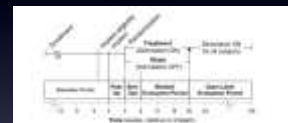
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Responsive cortical stimulation for the treatment of medically intractable partial epilepsy

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18-70 years
not been controlled ≥ 2
 ≥ 3 disabling seizure / month
localized 1 or 2 epileptogenic regions

Endpoint
seizure frequency
safety

Responsive cortical stimulation for the treatment of medically intractable partial epilepsy

Summary: Responsive cortical stimulation for the treatment of medically intractable partial epilepsy is associated with improvements in quality of life, and its effectiveness may be improved or optimized if responsive cortical stimulation may provide another effective treatment option for adults with medically intractable partial epilepsy.

Classification of Evidence: This study provides Class I evidence that responsive cortical stimulation is effective in significantly reducing seizure frequency for 12 weeks in adults who have failed 2 or more antiepileptic medication trials, 3 or more seizures per month, and 1 or 2 seizure-free intervals (SWI), NCT00612094

DISCLOSURE

Dr. Meo is an employee of and holds stock options in NeuroPace, Inc. is an employee of InVivo Metric or epilepsy clinical practice (20% effort) has received speaker honoraria from Cleveland Clinic, serves on the editorial board of *Neurostimulation*, is on the organizing committee of the American Society of Experimental Neurostimulation (AGENT), and is on the Board of Directors of the Epilepsy Research Foundation.

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From NeuroPace, Inc., Mountain View, CA.

Conflict of Interest Statement: Dr. Meo, which participated in acquisition of data, statistical analysis, study conception, and approval of this manuscript. Author disclosures are provided at the end of this article.

ORIGINAL CONTRIBUTION

Early Surgical Therapy for Drug-Resistant Temporal Lobe Epilepsy: A Randomized Trial

JAMA. 2011;305:1930-38

Author Disclosures: Dr. Williamson is on the advisory board for Abbott, Biogen, Bristol-Myers Squibb, Eisai, Genzyme, GlaxoSmithKline, Janssen-Cilag, Lundbeck, Merck, Novartis, Pfizer, Sanofi-Schering-Plough, and Takeda. Dr. Williamson is on the advisory board for Abbott, Biogen, Bristol-Myers Squibb, Eisai, Genzyme, GlaxoSmithKline, Janssen-Cilag, Lundbeck, Merck, Novartis, Pfizer, Sanofi-Schering-Plough, and Takeda. Dr. Williamson is on the advisory board for Abbott, Biogen, Bristol-Myers Squibb, Eisai, Genzyme, GlaxoSmithKline, Janssen-Cilag, Lundbeck, Merck, Novartis, Pfizer, Sanofi-Schering-Plough, and Takeda.

Design, Setting, and Participants: This early randomized surgical therapy for drug-resistant temporal lobe epilepsy (TLE) trial was conducted at 14 US epilepsy centers. The 38 patients (18 men and 20 women, aged 12-52 years) had medically refractory TLE and were randomized to early (n = 19) or delayed (n = 19) surgery. The primary outcome was seizure freedom at 1 year. Secondary outcomes included seizure freedom at 2 years, seizure freedom at 5 years, and quality of life. The study was registered at ClinicalTrials.gov (NCT00107332) and approved for 2 years. Neurostimulation was used for the trial as indicated in the protocol.

Conclusions: Among patients with medically refractory TLE, earlier surgery (vs delayed surgery) resulted in a lower probability of seizure during year 1 of follow-up. Postoperative AED treatment was similar. Given the alternative treatments of the trial, this result should be interpreted with caution.

Status epilepticus

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Intramuscular versus Intravenous Therapy for Prehospital Status Epilepticus

Robert E. Shinnick, M.D., Victor Durkalski, Ph.D., Daniel L. Bernstein, M.D., Brian G. Simon, M.D., Arthur Pearson, M.D., Sara Pineda, Ph.D., and William H. Meo, M.D. for the NETT Investigators*

- Children with BW \geq 13 kg, and adult
- Convulsive seizure > 5 min.
- Drug Kit
 - BW \geq 40 kg. IM midazolam 10 mg or IV lorazepam 4 mg
 - BW 13-40 kg. IM midazolam 2 mg or IV lorazepam 2 mg
- Primary outcome: termination of seizure before arrival in the ER
- Secondary outcome:
 - time from study-box opening to termination of convulsions
 - time from initiation of active-drug administration to termination of seizure
 - the frequency and duration of hospitalization and of admission to the ICU
 - the frequency of acute ET and acute seizure recurrence

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Intramuscular versus Intravenous Therapy for Prehospital Status Epilepticus

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CONCLUSIONS

For subjects in status epilepticus, intramuscular midazolam is at least as safe and effective as intravenous lorazepam for prehospital seizure cessation. (Funded by the National Institute of Neurological Disorders and Stroke and others; ClinicalTrials.gov number, NCT00809146.)

PROPORTION of subjects admitted was significantly lower in the IM group than in IV group. Median time to administration of active treatment was significantly shorter by the IM than IV. Onset of action occurred sooner after IV than after IM.

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Antiepileptic drug selection for people with HIV/AIDS: Evidence-based guidelines from the ILAE and AAN

*Gretchen L. Birbeck, Jacqueline A. French, †Evelio Perucca, †David H. Simpson, †Henry Friedman, †Jerry M. George, †Jason P. Okun, †David B. Clark, †Helen Hackett, and †Diane H. Lary for the Quality Standards Subcommittee of the American Academy of Neurology and the Adult Task Force of the Commission on Therapeutic Strategies of the International League Against Epilepsy

- Patients receiving phenytoin may require a 10% lower steady-state dosage increase of about 50% to maintain unchanged serum concentrations (Level C).
 - Patients receiving valproic acid may require a 20% lower dosage reduction to maintain unchanged serum valproic acid concentrations (Level C).
 - Combination of valproic acid and efavirenz may not require efavirenz dosage adjustment (Level C).
 - Patients receiving lamotrigine may require a 50% higher dosage increase of about 50% to maintain unchanged lamotrigine serum concentrations (Level C).
 - Combination of lamotrigine and efavirenz may not require lamotrigine dosage adjustment (Level C).
 - Combination of lamotrigine and efavirenz may not require efavirenz dosage adjustment (Level C).
 - Patients may be counseled that it is unclear whether dosage adjustment is necessary when other AEDs and ARVs are combined (Level U).
- It may be important to avoid B1-AEDs in people on ARV regimens that include PI or NRTIs, as pharmacokinetic interactions may result in toxicity, failure, and/or clinical implications for disease progression and development of ARV resistance. If such regimens are required for disease control, patients may be monitored through pharmacokinetic assessments to ensure efficacy of the ARV regimen (Level C).

PRACTICE

NICE epilepsy guidance “may be detrimental to patient care”

New NICE guidelines on epilepsy have come under fire by several experts who say that they do not reflect clinical experience and focus too much on drug cost effectiveness. David Holmes reports.

Chief clinical adviser to the guideline: “The Prince of Wales’s chair of childhood epilepsy”

National Clinical Guidelines Centre, Royal College of Physicians, London SW1P 3BZ, UK; TCD, National Child Health Research Development Centre, London W12 0NS, UK; Young Epilepsy, London W12 0NS, UK

HISTORICAL REVIEW

The causes of epilepsy: Changing concepts of etiology of epilepsy over the past 150 years

Blaise D. Sharves

MCL Institute of Neurology, University College London, London, United Kingdom

SPECIAL REPORT

International consensus clinical practice statements for the treatment of neuropsychiatric conditions associated with epilepsy

*Hille P. Korn, †Seeh Muzali, †Frank Beeg, †Bertrand de Toffi, †Alan Ettinger, †Kousuke Kanemoto, †Andreas Kanner, †Steven Kemp, †Ervan Padam Krishnamoorthy, †W. Carl LaFrance Jr, †Marco Mula, †Bertho Schmitz, †Ludwig Tassiartz van Elst, †Julien Triller, and †Tara J. Williamson

International League Against Epilepsy
Epilepsia, Vol. 61, No. 10, 2020

SPECIAL REPORT

Epilepsy imaging study guideline criteria: Commentary on diagnostic testing study guidelines and practice parameters

*William D. Gaillard, †Helen Cross, †John S. Duncan, †Hermann Stefan, †William H. Theodore, and †Tara Farnon on Practice Parameter Imaging Guidelines for the International League Against Epilepsy, Commission for Diagnostics

Epilepsia, Vol. 61, No. 10, 2020

Mechanisms of epileptogenesis and potential treatment targets

David Holmes (epilepsy@ucl.ac.uk)

Epilepsia, Vol. 61, No. 10, 2020

Prevention of epileptogenesis after brain trauma is an unmet medical challenge. Recent molecular profiling studies have provided an insight into molecular changes that contribute to formation of long-term neuronal networks, including gene regulatory networks or neuronal plasticity, cell death, proliferation, and inflammation or immune responses. These mechanisms have been targeted to prevent epileptogenesis in animal models. Promising effects have been obtained using immunomodulators, antibodies blocking activation of receptors in molecular cell-gene therapy, blocking expression of neurotrophic factors, pharmacological neuroprotection, or even with neuronal autologous drugs by administering them before the appearance of genetic epilepsy. Further studies are needed to clarify the optimal time window and optimal specificity of treatment. Questions related to safety events also need further consideration. Interestingly, the more experimental studies emphasize that the complex process of epileptogenesis can be genetically modified, and that epileptogenesis as a complex indication might not be as inoperable as once.

Management of refractory status epilepticus in adults: still more questions than answers

Andreas Kanner (epilepsy@ucl.ac.uk)

Epilepsia, Vol. 61, No. 10, 2020

Refractory status epilepticus (RSE) is defined as status epilepticus that continues despite treatment with benzodiazepines and antiepileptic drug. RSE should be treated promptly to prevent morbidity and mortality. However, since evidence is limited to support the choice of specific treatments, major independent outcome problems are yet to be resolved and new data should be actively targeted. No clear recommendations for adults suggest that the appropriate use of benzodiazepines for RSE should be defined in the clinical situation. In combination with anti-oxidant-coagulation, focal RSE without treatment of electrocatalytic angle initially is approached conservatively, generally with induction of anaesthesia or generalised convulsive therapy if the disorder is life threatening. In addition, propofol or ketamine on the one hand and midazolam, benzodiazepines, such as additional anaesthetics, after withdrawal of antiepileptic or immunomodulatory compounds, or immunomodulatory approaches, for immunomodulation treatment or hyperthermia, have been used in prolonged RSE. Treatment settings need to be further evaluated in a good evidence, in a clinical practice after completion of systematic literature. Well-designed prospective studies of RSE are urgently needed.

NEUROLOGICAL PROGRESS

Therapeutic Devices for Epilepsy

Robert S. Fisher, MD, PhD

Therapeutic devices provide new options for treating drug-resistant epilepsy. These devices act by a variety of mechanisms to modulate neuronal activity. Only vagus nerve stimulation (VNS), which continues to develop new technology, is approved for use in the United States. Deep brain stimulation of anterior thalamus for partial epilepsy recently was approved in Europe and several other countries. Responsive neurostimulation, which delivers stimuli to 1 or 2 patients but is responsive to a detected seizure, recently constituted a successful neuroscience trial. Several other trials of brain stimulation are in planning or underway. Transcutaneous magnetic stimulation (TMS) may provide a noninvasive method to stimulate cortex. Controlled studies of TMS are still an ongoing, which may depend on whether a seizure focus is near a possible region for stimulation. Seizure detection devices in the form of shake detectors or portable neurostimulators are possible candidates of an ongoing brain-stimulus network, or gates of access to the thalamus of stimulation. Prediction of seizures from neural signals of electroencephalography (EEG) is in early stages. Prediction appears to be possible in a subpopulation of groups with refractory seizures, and a clinical trial of an implanted prediction device is underway. Coding of response or hyperexcitability in the epileptic epileptic RSE activity and seizures, but engineering problems remain in the implementation. Comparison to a new technique that can control excitability of specific populations of neurons with light, inhibition of epileptic activity has been demonstrated in experimental animals, but use in humans will require more work, in general, devices provide useful prediction for seizure unprovoked seizures, but with a different risk profile than with drug therapy. Optimization of use of devices for therapy for epilepsy will require further development and clinical experience.

ANN NEUROL 2019;85:1171-1181

Research in Thailand

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Research in Thailand

Short Report
Epilepsy & Behavior, Volume 111, Issue 1, May 2012, Pages 173-178

4 newly identified loci for benign adult familial myoclonic epilepsy
Tara Hering, Ingrida Roca-Trillo, Mark Goodfellow, Lisa Parsons, Heidi Smith, Steve Mead

Factors associated with knowledge and attitudes in persons with epilepsy
Joseph Rattapornchai¹, Susanna Rattapornchai¹, Sornrat Kijjanakul¹, Nitsara Rattapornchai¹, Sornrat Rattapornchai¹, R. Rattapornchai¹, Rattapornchai Research Group¹

Abstract
Background: Knowledge and attitudes of persons with epilepsy (PWE) are important for their social and psychological well-being. This study aimed to identify factors associated with knowledge and attitudes in PWE.

Research in Thailand

Abdominal epilepsy: an assessment of non-convulsive status epilepticus
Dimitris S. Stafylidis, C. Stefanidis, S. Stefanidis

Abstract
Background: Abdominal epilepsy (AE) is a rare form of focal epilepsy. The aim of this study was to assess the clinical features and outcome of AE.

Research in Thailand

Mutation screening of the CDKL5 gene in cryptogenic infantile intractable epilepsy and review of clinical sensitivity
Uthairat Huanont¹, Puchit Rattapornchai¹, Chantana Phinyo¹, Tawana Srisa¹, Puchit Rattapornchai¹, Sornrat Rattapornchai¹, Rattapornchai Research Group¹

Abstract
Background: CDKL5 gene mutations are associated with infantile intractable epilepsy (IIE). This study aimed to identify CDKL5 mutations in Thai IIE patients.

Research in Thailand

Characterization of glucose homeostasis and lipid profile in adult, seizure-free, epileptic patients in Asian population
K. Phatphai¹, K. Limapichai¹, P. Sathirapanya¹, S. Sathiracharawong¹ and A. Gease²

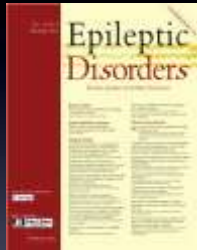
Abstract
Background: Epilepsy is associated with metabolic abnormalities. This study aimed to characterize glucose homeostasis and lipid profile in adult, seizure-free epileptic patients.

Research in Thailand

Clinical characteristics, etiology and long-term outcome of epilepsy partialis continua in adult patients in Thailand
Karnpotee Phatphai¹, Kitt Limapichai¹, Puchit Rattapornchai¹, Susanna Rattapornchai¹, Alan Gease²

Abstract
Background: Epilepsy partialis continua (EPC) is a rare form of focal epilepsy. This study aimed to describe the clinical characteristics, etiology, and long-term outcome of EPC in adult patients in Thailand.

Research in Thailand



Original article

Montreal Cognitive Assessment in cryptogenic epilepsy patients with normal Mini-Mental State Examination scores

Kompoeng Phatthana, Pongpan Ngamwongwan
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Received 15 October 2011; accepted 15 January 2012

OBJECTIVE: The objective of this study was to evaluate the Montreal Cognitive Assessment (MoCA) in cryptogenic epilepsy patients with normal Mini-Mental State Examination (MMSE) scores. The study was conducted in a tertiary care hospital in Bangkok, Thailand. The study included 100 patients with cryptogenic epilepsy who had normal MMSE scores. The MoCA was administered to all patients. The results showed that the MoCA scores were significantly lower than the MMSE scores. The study also found that the MoCA scores were significantly lower in patients with cryptogenic epilepsy compared to patients with idiopathic generalization epilepsy. The study suggests that the MoCA is a more sensitive measure of cognitive function than the MMSE in cryptogenic epilepsy patients with normal MMSE scores.

KEYWORDS: cryptogenic epilepsy; cognitive function; Montreal Cognitive Assessment; Mini-Mental State Examination