

# 9<sup>th</sup> ASIAN & OCEANIAN EPILEPSY CONGRESS







MANILA, PHILIPPINES

22<sup>nd</sup> - 25<sup>th</sup> March 2012

# **Final Programme and Abstract Book**



www.epilepsymanila2012.org

# **LONDON 2012**

September 30th - October 4th

**10th European Congress on Epileptology** 



22<sup>nd</sup> - 25<sup>th</sup> March 2012

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# WELCOME MESSAGE FROM THE SCIENTIFIC ORGANISING COMMITTEE CHAIRS

Dear Friends and Colleagues,

On behalf of the Scientific Organising Committee, it gives us great pleasure to welcome you to the 9<sup>th</sup> Asian & Oceanian Epilepsy Congress (AOEC) taking place here in the exciting city of Manila for the next few days. This congress has been organised by the regional organisations of the International League Against Epilepsy and the International Bureau for Epilepsy.

More than 100 local, regional and international speakers will present a range of topics that are highly relevant to recent scientific, clinical and social developments in epilepsy. The Scientific Organising Committee and the Scientific Consultative Committee have been working arduously to prepare a scientific programme with international appeal. The main topics of the Congress are: "Epilepsy and the Developing Brain", "Epilepsy Genes and Beyond", "The Impact of Epilepsy and its Treatment" and "Epileptic Networks and Seizure Propagation". In addition to the main and the post main sessions are a range of parallel sessions which will focus on the pertinent issues of epilepsy practice and research, the Masakazu Seino Memorial Lecture, practical video sessions, lively debates, interactive workshops and topical satellite symposia. The Asian Epilepsy Academy (ASÉPA) has organised a series of didactic lectures by world renowned experts in their respective fields on interesting topics such as Seizure and Epilepsy Classification. Psychosocial Aspects of Epilepsy, Brain Stimulation, EEG Monitoring in the ICU and Autoimmune Seizure Disorders. ASEPA pre-congress workshops on Translational Research and Drug Resistant Epilepsy will also take place on Thursday 22<sup>nd</sup> March.

The Epilepsy & Society Symposium will take place on Saturday 24th March. This programme will be of great interest to both individuals living with epilepsy and for staff from community organisations supporting people living with epilepsy. Further details of this symposium are contained within this programme.

Make sure to attend the platform and poster sessions featuring the latest research and data on epilepsy; the quality of papers submitted this year was particularly high. The two best platform and two best poster presentations will receive the Tadokoro Award on Sunday 25th March.

We invite you to attend the Opening Ceremony on Thursday 22<sup>nd</sup> March, where we will award the Asian & Oceanian Outstanding Achievement Epilepsy Award and Outstanding Persons with Epilepsy Award in recognition of those with a distinguished contribution in the field of epilepsy in the region. This will be followed by Welcome Cocktails and a Cultural Night (called "Pagdiriwang" or Celebration) prepared for us by the Department of Tourism.

Manila is a blend of the past and the present, and of the east and the west. Its Hispanic-inspired historical and cultural sites dating back 400 years stand in striking contrast with sky scraping edifices. There is a large choice of malls, entertainment venues and restaurants to entertain you after the Congress. See the old walled city, Intramuros, enjoy the bayside breeze and the famous Manila Bay sunset.

We hope that the Congress meets your highest expectations and that you enjoy your stay in Manila.

With warm regards,



JOSEPHINE CASANOVA-GUTIERREZ Co-chair Scientific Organising Committee



ROBERT COLE Co-chair



BYUNG-IN LEE Co-chair Scientific Organising Committee Scientific Organising Committee

# WELCOME MESSAGE FROM THE PRESIDENTS OF THE INTERNATIONAL LEAGUE AGAINST EPILEPSY (ILAE) AND THE INTERNATIONAL BUREAU FOR EPILEPSY (IBE)

Dear Friends,

On behalf of both the International League Against Epilepsy (ILAE) and the International Bureau for Epilepsy (IBE), it is our pleasure to welcome you to Manila for the 9<sup>th</sup> Asian & Oceanian Epilepsy Congress.

With a legacy spanning over almost two decades, the congress has grown considerably in size and scope. It attracts researchers from all over Asia and Oceania and, indeed, the wider world. This year's scientific programme has been carefully selected to ensure comprehensive coverage of the key topics at the cutting edge of epileptology. The congress is also an ideal opportunity to meet those at the forefront of epilepsy research, and to share ideas with fellow epileptologists, neurologists and care providers.

We look forward to meeting you over the coming days, and we wish you a valuable and enjoyable experience in Manila.

With best wishes,



Solomon L. MOSHÉ (USA) President ILAE





Mike GLYNN (Ireland) President IBE





WELCOME MESSAGE

On behalf of the Philippine Department of Tourism, I would like to welcome the delegates of the 9<sup>th</sup> Asian & Oceanian Epilepsy Congress (AOEC).

In addition to the exchange of knowledge and expertise, the AOEC event has always been an opportune occasion for the delegates from this medical and scientific field to strengthen regional camaraderie.

The Philippines, particularly its capital Manila, has been host to innumerable congresses and meetings of various natures and disciplines. Our long experience as host to international events coupled with our constant drive for excellence have made us confident that we can make the 9th AOEC event not just successful but, likewise, memorable.

The entire Philippine Department of Tourism (PDOT), through its attached agency, the Tourism Promotions Board (TPB), is committed to extending the utmost support to ensure the success of the 9<sup>th</sup> Asian & Oceanian Epilepsy Congress.

I would also like to take this opportunity to recognize the efforts of the International League Against Epilepsy / International Bureau for Epilepsy, with the support of the Philippine League Against Epilepsy, in bringing together global experts in the field and in providing the Filipinos the opportunity to enhance further their capabilities, interact and collaborate with their counterparts from the region.

Mabuhay and welcome to the Philippines!



RAMON R. JIMENEZ, JR. Secretary of Tourism

22<sup>nd</sup> - 25<sup>th</sup> March 2012

# **GENERAL CONGRESS INFORMATION**

## **Scientific Organising Committee**

Josephine CASANOVA-GUTIERREZ (Philippines), Co-chair Robert COLE (Australia), Co-chair Byung-In LEE (South Korea), Co-chair

Ding DING (China) Simon HARVEY (Australia) Patrick KWAN (Hong Kong) Andrew PAN (Singapore) Vinod SAXENA (India) Tatsuya TANAKA (Japan)

# Scientific Consultative Committee

Leonor CABRAL-LIM (Philippines), Co-chair John DUNNE (Australia), Co-chair

Hasan AZIZ (Pakistan) Victoria BAEL (Philippines) Peter BERGIN (New Zealand) Andrew BLEASEL (Australia) Denise CHAPMAN (Australia) Yotin CHINVARUN (Thailand) Imelda DAVID (Philippines) Jeannie DESIREE-KHONGHUN (Philippines) Shinichi HIROSE (Japan) Seung Bong HONG (South Korea) Akio IKEDA (Japan) Yushi INOUE (Japan) Satish JAIN (India) Sunao KANEKO (Japan) Heung Dong KIM (South Korea) Shichuo LI (China) Weiping LIAO (China) Shih Hui LIM (Singapore) Yung-Yang LIN (Taiwan)

Guoming LUAN (China) Man Mohan MEHNDIRATTA (India) Vrushali Vinod NADKARNI (India) Terry O'BRIEN (Australia) Marilvn ORTIZ (Philippines) Jiong QIN (China) Kurupath RADHAKRISHAN (India) Belinda SANCHEZ (Philippines) Ingrid SCHEFFER (Australia) Graeme SHEARS (Australia) Ernie SOMERVILLE (Australia) Maria Felicidad SOTO (Philippines) Chong Tin TAN (Malaysia) Grace TAN (Singapore) Jing-Jane TSAI (Taiwan) Yuan-Fu TSENG (Taiwan) Annanit VISUDTIBHAN (Thailand) Masako WATANABE (Japan)

# ASSOCIATED COMMITTEES SUPPORTING THE 9<sup>th</sup> ASIAN & OCEANIAN EPILEPSY CONGRESS

Commission on Asian and Oceanian Affairs (CAOA) The Asian Epilepsy Academy (ASEPA) IBE Vice Presidents of Western Pacific and South East Asia IBE Western Pacific and South East Asia regional executive committees Philippine League Against Epilepsy (PLAE) Epilepsy Awareness and Advocacy Inc (EAA) Executive members of the International League Against Epilepsy (ILAE) Executive members of the International Bureau for Epilepsy (IBE)

# **GENERAL CONGRESS INFORMATION**

# **Facilities Timetable**

	Thursday	Friday	Saturday	Sunday
Registration	07:30 – 18:30	07:00 - 18:00	07:00 – 18:00	07:00 – 13:00
Speakers Ready Room	07:30 – 17:30	07:00 – 17:30	07:00 – 17:30	07:00 - 11:00
Posters on Display	-	09:00 - 17:00	09:00 - 17:00	-
Exhibition	-	09:00 - 17:00	09:00 - 17:00	09:00 - 12:00
Coffee Break Morning	-	10:30 - 11:00	10:30 - 11:00	10:30 - 11:00
Coffee Break Afternoon	-	16:00 – 16:30	16:00 – 16:30	-
Lunch	-	12:30 - 13:30	12:30 – 13:30	-
Internet Area	-	09:00 – 17:00	09:00 – 17:00	09:00 – 12:00

# **Certificate of Attendance**

A Certificate of Attendance will be available for all delegates for collection from the registration area on the 2<sup>nd</sup> floor of the SMX Convention Center on Sunday.

# **Coffee Breaks**

Coffee, tea and snacks will be served in the exhibition area in Function Room 5 on the  $2^{nd}$  floor of the SMX Convention Center from 10:30 - 11:00 on Friday, Saturday and Sunday and also from 16:00 - 16:30 on Friday and Saturday.

# Exhibition

A trade exhibition will be held in conjunction with the 9<sup>th</sup> Asian & Oceanian Epilepsy Congress. This is an integral part of the event, offering delegates the opportunity to learn about the latest developments in products and services relevant to the field of epilepsy. The exhibition area is located in Function Room 5 on the 2<sup>nd</sup> floor of the SMX Convention Center.

# **Internet Area**

Internet stations are located within the exhibition area in Function Room 5 on the 2<sup>nd</sup> floor of the SMX Convention Center; please note that these are open during exhibition hours only.

### Language

English is the official language of the 9th Asian & Oceanian Epilepsy Congress.

# Liability and Insurance

The Congress Organiser will not accept any liability for personal injury or loss/damage to property/ belongings of participants or accompanying persons, either during or following the congress, tours or their stay in Manila. It is therefore recommended that participants arrange their own personal health, accident and travel insurance.

# Lunch

Lunch will be served in the exhibition area in Function Room 5 on the  $2^{nd}$  floor of the SMX Convention Center on Friday and Saturday 12:30 – 13:30.

# **Message Board**

A message board can be found beside the registration desk outside Function Room 1 on the 2<sup>nd</sup> floor of the SMX Convention Center. Delegates are invited to check it regularly.

## **Posters**

Posters are exhibited in Function Room 5 on the  $2^{nd}$  floor of the SMX Convention Center. Posters will be on display from 09:00 - 17:00 on Friday and Saturday. Poster presenters are required to set up their posters between 08:00 - 09:00 on Friday morning. Posters must be removed between 17:00 - 18:00 on Saturday.

# Registration

The registration area is located outside Function Room 1 on the  $2^{nd}$  floor of the SMX Convention Center; congress bags can also be collected from this point. Please note that name badges must be worn at all times.

# **Replacement Badges**

Please note that a fee of US\$50 will be charged for a replacement badge.

# **Secretariat Office**

Members of the Congress Secretariat can be contacted at the registration area which is located outside Function Room 1 on the  $2^{nd}$  floor of the SMX Convention Center.

For queries arising after the congress, please contact:

Áine Mitchell, 9th Asian & Oceanian Congress Secretariat, ILAE/IBE Congress Secretariat, 7 Priory Hall, Stillorgan, Dublin 18, Ireland. Tel : +353 1 2056720 Fax : +535 1 2056156 Email : manila@epilepsycongress.org Website : www.epilepsymanila2012.org

# **Smoking Policy**

The SMX Convention Center is a non-smoking area.

# **Speakers Ready Room**

The Speakers Ready Room is located in Meeting Room 10 on the 2<sup>nd</sup> floor of the SMX Convention Center. Facilities to review and amend presentations will be available to all speakers and technical assistance will also be provided in this room. Please note that all speakers should submit their final PowerPoint presentations to the main desk in the Speakers Ready Room no later than 2 hours in advance of their session. Speakers in early morning session are required to submit their material before 17:00 on the day prior to their scheduled session.

### **Sponsors**

The International League Against Epilepsy (ILAE), the International Bureau for Epilepsy (IBE) and the 9<sup>th</sup> Asian & Oceanian Epilepsy Congress Scientific Organising Committee (AOEC SOC) would like to thank the Philippine Department of Tourism for their contribution to this year's AOEC.



The 9<sup>th</sup> Asian & Oceanian Epilepsy Congress acknowledges the support of the following companies through the Philippine League Against Epilepsy:

Abbott, Ambica, AstraZeneca, GlaxoSmithKline, Innogen, Medichem, Natrapharm, Pfizer and Torrent.

# **Venue Information**

The 9<sup>th</sup> Asian & Oceanian Epilepsy Congress will be held at the SMX Convention Center.

Venue address:

SMX Convention Center, Seashell Lane, Mall of Asia Complex, Pasay City, 1300 Philippines. Website: www.smxconventioncenter.com

# **Wheelchair Access**

All conference rooms in the SMX Convention Center are wheelchair accessible.

# **SOCIAL EVENTS**

# **Welcome Ceremony**

The Welcome Ceremony of the 9<sup>th</sup> Asian & Oceanian Epilepsy Congress will take place in Function Room 4 on the 2<sup>nd</sup> floor of the SMX Convention Center on Thursday  $22^{nd}$  March at 18:30.

# **Welcome Reception**

All delegates are invited to join the Welcome Reception which will be held following the Welcome Ceremony in Function Rooms 2 & 3.



# **CONGRESS FLOOR PLANS**





# PRACTICAL INFORMATION ON MANILA

#### About Manila

Manila, also known as the Pearl of the Orient, is located in Southern Luzon, the largest of more than 7,000 islands that make up the nation known as the Philippines. The city flanks Manila Bay, and is divided into northern and southern sections by the Pasig River. Manila serves not only as the country's capital, but also as its financial, publishing, and business centre.

Many who travel to Manila find that the cosmopolitan capital of the Philippines is the most exciting city in Asia, offering a blend of cultures, a good supply of historic sights and places to see, and unforgettable experiences. Another plus for Manila is that the city is as lively at night as it is during the day. The city caters for all religious and dietary needs so everyone will feel welcome.

# **Airport Tax**

Passenger Terminal Fee is levied on all passengers embarking for: International Travel: PHP 750 (US\$17) Domestic Travel: PHP 200 (US\$17) As per February 2012

# **Airport Transfers**

Transfers to the airport at US\$8 can be booked at the designated Intas Destination desk which is outside Function Room 3 on the 2<sup>nd</sup> floor of the SMX Convention Center.

# **Business Hours**

Private and government offices are open either from 08:00 to 17:00 or from 09:00 to 18:00. Some private companies hold office on Saturdays from 09:00 to 12:00. Most shopping malls, department stores, and supermarkets are open from 10:00 to 20:00 daily. There are 24-hour convenience stores and drugstores. Banks are open from 09:00 to 15:00, Mondays to Fridays, with automated teller machines (ATM) operating 24 hours.

# **Credit Cards and ATMs**

Credit cards are accepted at most restaurants and shops, the most widely used being MasterCard, Visa, American Express and Diners Club. ATMs are widely available throughout the city of Manila.

#### Currency

Philippine Peso (PHP) currency is used in Manila, which is composed of 100 centavos. Coins come in denominations of 5, 10 and 25 centavos, 1, 5 and 10 peso; and 20, 50, 100, 200, 500 and 1,000 notes. Currency exchange facilities are available in most banks, hotels and airports and operate normal business hours.

# **Eating Out**

Manila is a prime location for outstanding restaurants that are ready to serve all catering requests. Overall, these first-class food service providers offer delicious cuisine and highly efficient service for truly pleasurable fine dining experiences.

# Electricity

The electrical current is 220 volts, 60Hz. Two-pin flat blade attachments and two-pin round plugs are used.

# Тах

A 12% VAT tax applies to all food, shopping and hotel purchases in Manila.

# Taxis

When taking a taxi in Manila, it is important to look for an official metered taxi. There are taxi ranks in various locations throughout the city.

# **Time Zone**

Manila is 8 Hours ahead of Greenwich Mean Time in March.

# Tipping

A service charge of 10% is usually added to hotel and restaurant bills. If not, a 10% tip is appropriate for waiters.

# **Transport in Manila**

A complimentary shuttle service between the SMX Convention Center and the official 9<sup>th</sup> Asian & Oceanian Epilepsy Congress Hotels will be available to delegates who booked their accommodation through the 9<sup>th</sup> Asian & Oceanian Epilepsy Congress website. Timetables for these shuttle services will be on display in the various hotel reception areas and also on display in the registration area on the 2<sup>nd</sup> floor of the SMX Convention Center.

# **GENERAL PROGRAMME INFORMATION**

# AOEA – The Asian and Oceanian Outstanding Achievement Epilepsy Award

The Asian and Oceanian Outstanding Achievement Epilepsy Award (AOEA) will be presented to 6 awardees during the Welcome Ceremony on Thursday 22<sup>nd</sup> March. The award is bestowed on Leonor CABRAL-LIM (Philippines), Yushi INOUE (Japan), Sunao KANEKO (Japan), Kurupath RADHAKRISHNAN (India), Pongsakdi VISUDHIPHAN (Thailand) and Liwen WU (China).

# ASEPA EEG Certification Examination Part 1

The ASEPA EEG Certification Examination Part 1 will take place on Thursday  $22^{nd}$  March from 08:00 - 12:00.

# **ASEPA Workshops**

**Clinical Epilepsy Workshop:** "Drug-resistant epilepsy: recognize it, treat it" will take place on Thursday 22<sup>nd</sup> March from 08:00 – 11:30.

**Basic Science Workshop:** "Translational research" will take place on Thursday 22<sup>nd</sup> March from 08:30 – 12:00.

**Registration:** To attend the ASEPA pre-congress workshops, you must register for them at the onsite registration desk. The registration fee for each workshop is US\$20 for congress delegates and US\$40 for non-congress delegates.

# **Epilepsy & Society Symposium**

An exciting programme that will be of great interest to both individuals living with epilepsy and to staff from community organisations supporting people with epilepsy will take place on Saturday 24<sup>th</sup> March, 08:30 – 17:15 in Meeting Rooms 7-9. This programme has been developed by local and regional committees of the International Bureau for Epilepsy (IBE). The main topics will focus on "Anxiety and depression in epilepsy", "The impact of epilepsy" and "Empowering people with epilepsy".

Please note that a separate registration is required for this programme; please enquire at the onsite registration desk.

# **Outstanding Person with Epilepsy Awards**

The Outstanding Person with Epilepsy Award will be presented to 7 awardees during the Welcome Ceremony on Thursday 22<sup>nd</sup> March. The award is bestowed on Yung-Chich CHEN (Taiwan), Fai Ming HUNG (Hong Kong), Baldwin Chua KHO (Philippines), Hongquan LI (China), Martin RAFFAELE (Australia), Purevjav TSOGTSAIKHAN (Mongolia) and Yashoda WAKANKAR (India).

# **Tadokoro Award**

In order to encourage young researchers in epileptology in the region, there will be best presentation prizes for both platform and poster presentations. Dr. Tadokoro is a retired epileptologist from Japan who contributed generously to the activities of the ILAE Commission on Asian and Oceanian Affairs (CAOA).

The first and second prize for both platform and poster presentation are US\$300 and US\$200 respectively and the recipients will be announced on Sunday 25<sup>th</sup> March before the main session.

# **Travel Bursary Awards**

The Travel Bursary Award scheme was established to assist delegates to attend the 9<sup>th</sup> Asian & Oceanian Epilepsy Congress. A particular emphasis was given to those coming from developing regions, which are locally active in the field of epilepsy. A total of 32 Travel Bursary Awards were provided for the 9<sup>th</sup> Asian & Oceanian Epilepsy Congress. Funding for these awards was provided by the International League Against Epilepsy (ILAE) and the International Bureau for Epilepsy (IBE) and the ILAE Commission on Asian and Oceanian Affairs (CAOA).





# SCIENTIFIC PROGRAMME - FULL PROGRAMME TIMETABLE

Thursday 22 <sup>nd</sup> March		Friday 23rd March					
ASEPA Pre-Congress Workshop Drug-resistant epilepsy: recognise it, treat it 08:00-11:30 Meeting Room 5 Translational research		ASEPA Didactic Lecture: Classification of seizures and epilepsy 07:30-08:15 Function Room 3 ASEPA Didactic Lecture: Psychological issues in epilepsy					
		08:15-09:00 Function Room 3 Main Session Epilepsy genes and beyond 09:00-10:30 Function Room 4					
	08:30-12:00 Meeting Room 4		Coffe	e Break		10:30	EpiNet Project
		Post Main Section	10.00	Pa	allel Session	IS 11:00-	12:30
		The genetics of epilepsy 11:00-12:30 Function Room 4	Psy Func	chosis and epilepsy tion Room 3	Neuroimag epileps Function Ro	ing of sy com 2	Epilepsy and sleep Function Room 1
		Lunch & Posters 12:30 - 13:30					
Chairman's Symposium Epileptic networks and seizure propagation		Satellite Symposium UCB Epilepsy: from diagnosis to management challenges 13:30-15:00 Function Room 4					
Function	Room 4	Platform Sessions 15:00-16:00					
Masakazu Seino Memorial Lecture High fequency oscillations; a new marker of the		Clinical epileptology Basic science Function Room 3 Function Room 2		Clinica	al neurophysiology and neuroimaging Function Room 1		
epileptoge 15:30- Function	nic region 16:30 Room 4	Coffee Break 16:00 - 16:30					
Research highlights of young investigators in the Asian & Oceanian region 16:30-18:00	The need to advocate for epilepsy funding; how advocacy is working around the world 16:30-18:00	Video Seizures: focal generalized? 16:30-17:30 Function Room	or 3	Debate Add-on or substitute after the first drug failure? 16:30-17:30 Function Room 2		Glob	Workshop al Campaign Against Epilepsy 16:30-17:30 Function Room 1
Function Room 4 Meeting Rooms 8 & 9 Welcome Ceremony 18:30-19:30 Function Room 4		An updated v	iew on	Satellite Syn clinical uses of 17:3 Functio	nposium Elek magnetoence 0-19:00 n Room 4	t <b>a</b> ephalogr	aphy in epilepsy
Welcome Reception 19:30-20:30 Function Rooms 2 & 3							

# SCIENTIFIC PROGRAMME - FULL PROGRAMME TIMETABLE



Function Room 4	Meeting Room 4	Meeting Room 5
	ASEPA Pre-Congress Workshop Translational research 08:30-12:00	ASEPA Pre-Congress Workshop Drug-resistant epilepsy: recognise it, treat it 08:00-11:30
Chairman's Symposium Epileptic networks and seizure propagation 14:00-15:30		
Masakazu Seino Memorial Lecture High frequency oscillations; a new marker of the epileptogenic region 15:30-16:30		
Research highlights of young investigators in the Asian & Oceanian region 16:30-18:00		
Welcome Ceremony 18:30-19:30		
Welcome Reception 19:30-20:30		

Meeting Room 7	Meeting Rooms 8&9
ASEPA EEG Certification Examinations Part 1 08:00-12:00	
	ILAE/CAOA Chapter Convention 11:00-13:00
	The need to advocate for epilepsy funding; how advocacy is working around the world 16:30-18:00

#### 08:00 – 11:30 ASEPA Pre-congress workshop

#### Meeting Room 5

**Drug resistant epilepsy: recognise it, treat it** *Chair: KWAN, P (Hong Kong)* 

#### What is drug resistant epilepsy?

Natural history of treated epilepsy: relevance to diagnosing pharmacoresistance *KWAN, P (Hong Kong)* 

Natural history of treated epilepsy: are children and adults the same? *KIM*, *H D* (South Korea)

Classifying drug response in clinical practice *KWAN*, *P* (Hong Kong)

#### Why does drug resistance occur?

'Pseudoresistance'? Psychological comorbidities and lifestyle issues WILSON, S (Australia)

Hypothesised mechanisms of pharmacoresistance: lost in translation? BLEASEL, A (Australia)

Evaluation of drug-resistant epilepsy BLEASEL, A (Australia)

#### How to treat drug resistant epilepsy

Latest development in pharmacological treatment of drug-resistant epilepsy PERUCCA, *E* (*Italy*)

Latest development in non-pharmacological treatment of drug-resistant epilepsy COOK, M (Australia)

Using pharmacological and non-pharmacologial therapies to treat drug-resistant epilepsy COOK, M (Australia)

#### 08:30 - 12:00 ASEPA Pre-congress Workshop

Meeting Room 4

#### Translational research

Chairs: LIAO, W (China) & O'BRIEN, T (Australia)

Animal models of early life epilepsies? MOSHÉ, S L (USA)

Use of gene expression systems to study cellular effects of human epilepsy mutations *LIAO*, *W* (*China*)

New insights into cellular mechanisms of anti-epileptic drugs *FRENCH, C (Australia)* 

Using experimental models to provide insights into the mechanism of genetic generalised epilepsy YAMAKAWA, K (Japan)

Using rat models to study the mechanisms of acquired limbic epilepsy and develop new treatment approaches *TANAKA*, *T*(*Japan*)

Whole animal in-vivo imaging in animal epilepsy models: MRI, PET *O'BRIEN*, *T*(*Australia*)

#### 14:00 - 15:30 Main Session

#### **Function Room 4**

Chairman's Symposium: Epileptic networks and seizure propagation Chairs: CASANOVA-GUTIERREZ, J (Philippines), COLE, R (Australia) & LEE, B I (South Korea)

Insights from animal models into epileptic networks *TANAKA*, *T*(*Japan*)

Functional and structural connectivity imaging JACKSON, G (Australia)

Epilepsy networks in epilepsy surgery *MATHERN, G (USA)* 

#### 15:30 – 16:30 The Masakazu Seino Memorial Lecture

**Function Room 4** 

#### High frequency oscillations; a new marker of the epileptogenic region

Chair: LIM, S H (Singapore)

Speaker: GOTMAN, J (Canada)

**16:30 – 18:00** The need to advocate for epilepsy funding: How advocacy is working around the world Meeting Rooms 8 & 9

Chairs: GYLNN, M (Ireland), LEE, B I (South Korea) & MOSHÉ, S L (USA)

What are demonstration projects and how can they help to improve advocacy for epilepsy? *DE BOER, H (Netherlands)* 

Awareness campaign in Australia COLE, R (Australia)

What is the significance of the European Declaration on Epilepsy passed by the European Parliament last year? *PERUCCA, E (Italy)* 

What is the Pan American Health Organisation initiative for Epilepsy? *ACEVEDO, C (Chile)* 

Advocacy for epilepsy in the USA and what will the IOM report mean for people with epilepsy? *MATHERN, G (USA)* 

Multidimensional approaches to the advocacy of epilepsy in Taiwan *TSAI*, *J*-*J* (*Taiwan*)

How to mobilize government to fund epilepsy; the Chinese experience Promote the "International day for epilepsy" in the Western Pacific (WP) / South East Asian (SEA) region *LI*, *S* (*China*)

#### 16:30 – 18:00 Research Highlights of Young Investigators in the Asian & Function Room 4 Oceanian Region

Chairs: HARVEY, S (Australia) & NADKARNI V V (India)

EEG and ECoG for understanding epilepsy and sleep *CHO*, *J R* (*South Korea*)

Clinical presentations in the early phase of PCDH19 related epilepsy *HIGURASHI*, *N*(*Japan*)

Pharmacogenomics screening of HLA-B\*1502 in epilepsy patients: How we do it at the UKM Medical Center *MIAN*, *TS*(*Malaysia*)

Involvement of monocyte chemoattractant protein-1 in neural progenitor migration and epileptogenesis of rats with status epilepticus HUNG, Y-W (Taiwan)

Investigating psychological outcomes of paediatric epilepsy surgery MICALLEF, S (Australia)

# SATELLITE SYMPOSIUM

# **Epilepsy**:

# from diagnosis to management challenges

# Friday 23<sup>rd</sup> March, 13:30 –15:00, Function Room 4

Chair: Dr Josephine Casanova-Gutierrez, FPNA

PROGRAMME				
13:30 – 13:55	Diagnosing epilepsy – what are the key ingredients? <b>Dr Byung In Lee</b>			
13:55 – 14:20	Challenges in the treatment of uncontrolled epilepsy. <b>Dr Ernest Somerville</b>			
14:20 – 14:45	First becoming last. Selecting an optimal therapeutic choice. <b>Dr Patrick Kwan</b>			
14:45 – 15:00	A critical comparative review of AEDs. Dr Selim Benbadis			



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# SCIENTIFIC PROGRAMME - FRIDAY 23<sup>RD</sup> MARCH

Function Room 5	Function Room 4	Function Room 3	
Poster Set-up 07:30-09:00		ASEPA Didactic Lecture Classification of seizures and epilepsy 07:30-08:15	
		ASEPA Didactic Lecture Psychological issues in epilepsy 08:15-09:00	
	Main Session Epilepsy genes and beyond 09:00-10:30		
	Coffee Break - E	Exhibition Area	
	Post Main Session The genetics of epilepsy 11:00-12:30	Parallel Session Psychosis and epilepsy 11:00-12:30	
Posters on Display 09:00-17:00	Lunch - Exhibition Area		
	Satellite Symposium UCB Epilepsy: from diagnosis to management challenges 13:30-15:00		
		Platform Session Clinical epileptology 15:00-16:00	
	Coffee Break - Exhibition Area		
		Video Seizures: focal or generalized? 16:30-17:30	
	Satellite Symposium Elekta An updated view on clinical uses of magnetoencephalography in epilepsy 17:30-19:00		

# SCIENTIFIC PROGRAMME - FRIDAY 23<sup>RD</sup> MARCH

Function Room 2	Function Room 1
Coffee Break - Exhibition Area	EpiNet Project 10:30-10:50
Parallel Session Neuroimaging of epilepsy 11:00-12:30	Parallel Session Epilepsy and sleep 11:00-12:30
Lunch - Exf	nibition Area
Platform Session Basic science 15:00-16:00	Platform Session Clinical neurophysiology and neuroimaging 15:00-16:00
Coffee Break -	Exhibition Area
Debate Add-on or substitute after the first drug failure? 16:30-17:30	Workshop Global Campaign Against Epilepsy 16:30-17:30

# SCIENTIFIC PROGRAMME - FRIDAY 23RD MARCH

07:30 - 08:15 ASEPA Didactic Lecture Chair: MANNAN, M (Bangladesh)

> Classification of seizures and epilepsy LEE, BI (South Korea)

**08:15 – 09:00 ASEPA Didactic Lecture** Chair: GAMAGE, R (Sri Lanka)

> **Psychological issues in epilepsy** WIEBE, S (Canada)

#### 09:00 - 10:30 Main Session

**Function Room 4** 

**Function Room 3** 

Function Room 3

#### **Epilepsy genes and beyond** *Chair: KANEKO, S (Japan)*

Validation criteria for genetic animal models of epilepsy OKADA, *M*(*Japan*)

Epilepsy genetics: the impact on clinical practice *SCHEFFER, I (Australia)* 

Epilepsy genetics: generalized epilepsy versus partial epilepsy *LIAO*, *W* (*China*)

#### 11:00 – 12:30 Post Main Session

#### **Function Room 4**

**The genetics of epilepsy** *Chair: HIROSE, S (Japan)* 

Future approaches towards the genetics of epilepsy *DIBBENS, L (Australia)* 

Mitochondrial disorders and epilepsy CHAE, J H (South Korea)

Genetics of progressive myoclonic epilepsy SATISHCHANDRA, P (India)

# SCIENTIFIC PROGRAMME - FRIDAY 23<sup>RD</sup> MARCH

## 11:00 – 12:30 Parallel Session

#### **Psychosis and epilepsy: a multi-disciplinary approach** *Chair: WATANABE, M (Japan)*

Psychotic illness in patients with epilepsy; under-recognized but serious complications of long-standing epilepsy KANEMOTO, K (Japan)

Psychiatric comorbidity in patients with epilepsy PARK, S P (South Korea)

Psychosis of epilepsy: its mechanisms and rationale management CONDE, B (Philippines)

# 11:00 – 12:30 Parallel Session

# Function Room 2

Function Room 1

**Neuroimaging of epilepsy: therapeutic implications** *Chair: CHINVARUN, Y (Thailand)* 

SPECT and PET CHINVARUN, Y (Thailand)

fMRI; how are we now? JACKSON, G (Australia)

MRS; does it have value for therapeutic implications yet? LEE, S K (South Korea)

#### 11:00 – 12:30 Parallel Session

#### Epilepsy and sleep

Chair: SOTO, M F (Philippines)

The quality of the sleep in patients with epilepsy *DESUDCHIT*, *T* (*Thailand*)

Epilepsy syndromes that feature activity only in sleep *ZHANG*, *Y H (China)* 

Sleep disorders in patients with epilepsy *KING, P (Australia)* 

#### Function Room 3

# SCIENTIFIC PROGRAMME - FRIDAY 23RD MARCH

# 13:30 - 15:00 Satellite Symposium **Function Room 4** Satellite Symposium – UCB Epilepsy: from diagnosis to management challenges Chair: CASANOVA-GUTIERREZ, J (Philippines) Diagnosing epilepsy · what are the key ingredients? LEE, BI (South Korea) Challenges in the treatment of uncontrolled epilepsy SOMERVILLE, E (Australia) First becoming last: selecting an optimal therapeutic choice KWAN, P (Hong Kong) A critical comparative review of AEDs BENBADIS, S (USA) 15:00 - 16:00 **Platform Session Function Room 3** Clinical epileptology Chairs: SANCHEZ, B (Philippines) & DUNNE, J (Australia) 15:00 - 16:00 **Platform Session** Function Room 2 Basic science Chairs: QIN, J (China) & O'BRIEN, T (Australia) 15:00 - 16:00 Platform Session Function Room 1 Clinical neurophysiology and neuroimaging Chairs: IKEDA, A (Japan) & HONG, S B (South Korea) **Function Room 3** 16:30 - 17:30 **Practical Session** Video: Seizures: focal or generalized? Presenters:

Presenters: BLEASEL, A (Australia) HONG, S B (South Korea)

# SCIENTIFIC PROGRAMME - FRIDAY 23<sup>RD</sup> MARCH

# 16:30 - 17:30 Practical Session

## Debate: Add-on or substitute after the first drug failure

Moderator: LEE, B I (South Korea)

Add-ons KWAN, P (Hong Kong)

Substitutes PERUCCA, E (Italy)

### 16:30 - 17:30 Practical Session

Function Room 1

**Function Room 4** 

Workshop: Global Campaign Against Epilepsy Chairs: SOMERVILLE, E (Australia) & TSENG, Y F (Taiwan)

The Epilepsy Manager Project in the Philippines *SOTO, M F (Philippines)* 

A national epilepsy programme for India GOURIE-DEVI, M (India)

Are our efforts effective? Evaluating the outcomes SOMERVILLE, E (Australia)

#### 17:30 - 19:00 Satellite Symposium

#### Satellite Symposium - Elekta An updated view on the clinical uses of magnetoencephalography in epilepsy

Moderator: ENWALL, M (Sweden)

An updated view on the clinical uses of magnetoencephalography in epilepsy LIN, Y Y (Taiwan)

Role and necessity of MEG for epilepsy management in developing countries – a perspective from India *TRIPATI, M (India)* 

#### Function Room 2

# SCIENTIFIC PROGRAMME - SATURDAY 24TH MARCH

Function Room 5	Function Room 4	Function Room 3	
		ASEPA Didactic Lecture Brain stimulation 07:30-08:15	
		ASEPA Didactic Lecture The role of EEG monitoring in ICU 08:15-09:00	
	Main Session The impact of epilepsy <i>09:00-10:30</i>		
	Coffee Break - I	Exhibition Area	
	Post Main Session Anxiety and depression 11:00-12:30	Parallel Session Pharmacogenomics and antiepileptic therapy 11:00-12:30	
Posters on Display 09:00-17:00	Lunch - Exhibition Area		
	Satellite Symposium GSK Key controversies in epilepsy management 13:30-15:00		
		Platform Session Epidemiology and genetics 15:00-16:00	
	Coffee Break - Exhibition Area		
Poster Removal 17:00-18:00		Video Psychogenic non-epileptic seizures 16:30-17:30	

# SCIENTIFIC PROGRAMME - SATURDAY 24<sup>TH</sup> MARCH

Function Room 2	Function Room 1	Meeting Rooms 7-9
		Epilepsy & Society Symposium Empowering people with epilepsy 08:30-09:45
		Coffee Break - Foyer Area
Coffee Break -	Exhibition Area	Epilepsy & Society
Parallel Session Epilepsy and pregnancy 11:00-12:30	Parallel Session Novel surgical approaches 11:00-12:30	Symposium The impact of epilepsy 10:00-12:30
	Lunch - Exhibition Area	
		Epilepsy & Society Symposium Anxiety and depression in epilepsy 13:30-15:10
Platform Session Neuropsychology and social	Platform Session Surgery	Coffee Break - Foyer Area
15:00-16:00	15:00-16:00	Entlance & October
Coffee Break -	Symposium	
Debate Source localization: EEG versus MEG	Workshop Epilepsy in adolescence	The mom with epilepsy 15:30-17:15
16:30-17:30	16:30-17:30	

# SCIENTIFIC PROGRAMME - SATURDAY 24TH MARCH

07:30 - 08:15 ASEPA Didactic Lecture Chair: NEOPANE, A (Nepal)

> Brain stimulation IKEDA, A (Japan)

08:15 - 09:00 ASEPA Didactic Lecture Chair: TOVUUDORJ, A (Mongolia)

> **The role of EEG monitoring in ICU** DUNNE, J (Australia)

#### 08:45 - 09:45 Epilepsy & Society Symposium

Welcome Remarks CALILUNG, M I (Philippines)

Opening Remarks GLYNN, M (Ireland)

#### Empowering people with epilepsy

Chair: PARAGUA, H (Philippines)

Increasing your market value in the workplace *PALMA, A E R (Philippines)* 

Seahorse club: supporting creativity *DING*, *D*(*China*)

The Guagua Epilepsy Project: a livelihood model CALILUNG, M I (Philippines)

#### 09:00 - 10:30 Main Session

**The impact of epilepsy** *Chair: CABRAL-LIM, L (Philippines)* 

The impact of losing control *LEDESMA*, *L*(*Philippines*)

Death in epilepsy: is it preventable? ALI, R A (Malaysia)

The burden and future of epilepsy care in the region *LEE*, *B I* (South Korea)

#### **Function Room 4**

**Function Room 3** 

Function Room 3

Meeting Rooms 7-9

Meeting Rooms 7-9

#### 10:00 - 12:30 Epilepsy & Society Symposium

**The impact of epilepsy** *Chair: LAI, S (Taiwan)* 

What is SUDEP? ALAVA, R (Philippines)

SUDEP: global thrusts CHAPMAN, D (Australia)

Epilepsy: my personal journey MAGBITANG, R (Philippines)

The burden of epilepsy in the Asian-Oceanian region *LEE, B1 (South Korea)* 

Open Forum

#### 11:00 - 12:30 Post Main Session

#### **Function Room 4**

#### **Anxiety and depression in epilepsy** *Chair: CASANOVA-GUTIERREZ, J (Philippines)*

Neurobiology of depression in epilepsy MARASIGAN. S (Philippines)

Anxiety and depression in children with epilepsy ONG, L C (Malaysia)

Depression after epilepsy surgery WILSON, S (Australia)

#### 11:00 - 12:30 Parallel Session

#### **Function Room 3**

#### Pharmacogenomics and antiepileptic therapy

Chair: LIAO, W (China)

lon channels: genetics and as targets for antiepileptic drugs YAMAKAWA, K (Japan)

Genetic polymorphisms and metabolizing enzymes related to antiepileptic drugs KWAN, P (Hong Kong)

Seizure aggravation by antiepileptic drugs: a view from genetics *BERKOVIC*, *S*(*Australia*)

#### 11:00 - 12:30 Parallel Session

#### **Function Room 2**

#### Epilepsy and pregnancy

Chair: JAIN, S (India)

Preconception counselling, management and care of pregnant women with epilepsy; the role of pregnancy registries *THOMAS, S V (India)* 

Teratogenic effects of AEDs: what do we advise women? *KANEKO*, *S* (*Japan*)

Delivery, breastfeeding and child rearing in women with epilepsy CABRAL-LIM, L (Philippines)

#### 11:00 - 12:30 Parallel Session

#### Function Room 1

#### Novel surgical approaches

Chair: RADHAKRISHNAN, K (India)

Surgical disconnections of the epileptic zone as an alternative to lobectomy and hemispherectomy *LUAN*, *G*(*China*)

Surgical technique and outcome of transsylvian selective amygdalohippocampectomy *MORINO, M (Japan)* 

Update of novel surgical approaches for intractable epilepsy CHANG, J W (South Korea)

#### 13:30 - 15:00 Satellite Symposium

#### **Function Room 4**

#### Satellite Symposium – GlaxoSmithKline Key controversies in epilepsy management Chair: BENBADIS, S (USA)

Management of the uncontrolled patient: switch or add in? BRODIE, MJ (United Kingdom)

Response BENBADIS, S (USA)

Counter-response LABINER, D (USA)

Anti-epileptic drugs: are there different benefits between first and second generation agents? *LABINER, D (USA)* 

Response BRODIE, M J (United Kingdom)

Counter-response BENBADIS, S (USA)
13:30 - 15:30	Epilepsy & Society Symposium	Meeting Rooms 7-9
	Anxiety and depression in epilepsy Chair: GUNADHARMA, S (Indonesia)	
	The impact of losing control LEDESMA, L (Philippines)	
	Understanding depression in epilepsy <i>TRONCO, A (Philippines)</i>	
	Anxiety and depression in children with epilepsy ONG, L C (Malaysia)	
	Living with post traumatic epilepsy BELLON, M (Australia)	
	Personal story: parenting a child with epilepsy GENUINO, J (Philippines) & GENUINO, M (Philippines)	
15:00 - 16:00	Platform Session	Function Room 3
	<b>Epidemiology and genetics</b> Chairs: AZIZ, H (Pakistan) & NADKARNI, V V (India)	
15:00 - 16:00	Platform Session	Function Room 2
	<b>Neuropsychology and social issues</b> Chairs: SAXENA, V (India) & BAEL, V (Philippines)	
15:00 - 16:00	Platform Session	Function Room 1
	<b>Surgery</b> Chairs: KAMEYAMA, S (Japan) & LI, S (China)	
15:30 - 17:15	Epilepsy & Society Symposium	Meeting Rooms 7-9
	<b>The mom with epilepsy</b> Chair: KUSUMASTUTI, K (Indonesia)	
	Coping with baby care and other post partum issues for w <i>THOMAS</i> , S V (India)	omen with epilepsy
	Delivery and breastfeeding CABRAL-LIM, L (Philippines)	
	Teratogenic effects of antiepileptic drugs KANEKO, S (Japan)	
	l'm a mom with epilepsy KHONGHUN, J D (Philippines)	

**Closing remarks** COLE, R (Australia)

#### 16:30 - 17:30 Practical Session

#### Video: Psychogenic non-epileptic seizures

Presenters: PAN, A (Singapore) BERGIN, P (New Zealand)

#### 16:30 - 17:30 Practical Session

**Debate: Source localization: EEG versus MEG** Moderator: LIN, Y Y (Taiwan)

EEG GOTMAN, J (Canada)

MEG NAKASATO, N (Japan)

#### 16:30 - 17:30 Practical Session

#### Function Room 1

#### Workshop: Epilepsy in adolescence

Chair: ORTIZ, M (Philippines)

Epilepsy in adolescence: medical and health issues *VISUDTIBHAN, A (Thailand)* 

The adolescent epilepsy clinic: addressing special needs *LEE*, *M*(*South Korea*)

Transitioning from paediatric to adult health services: principles and applications *MEHNDIRATTA, M M (India)* 

#### **Function Room 3**

Function Room 2



## SCIENTIFIC PROGRAMME - SUNDAY 25<sup>TH</sup> MARCH

Function Room 4	Meeting Rooms 4-6	Meeting Rooms 7-9
ASEPA Didactic Lecture: Autoimmune seizure disorders 08:00-08:45		
Award Ceremony 08:45-09:00		
Main Session Epilepsy and the developing brain 09:00-10:30		
Coi	ffee Break - Exhibition A	rea
<b>Post Main Session</b> Epilepsy and autism <i>11:00-12:30</i>	Parallel Session Challenges in the diagnosis and treatment of status epilepticus 11:00-12:30	Parallel Session Sex and epilepsy 11:00-12:30

22<sup>nd</sup> - 25<sup>th</sup> March 2012

Meeting Rooms 4-6

### SCIENTIFIC PROGRAMME - SUNDAY 25TH MARCH

08:00 - 08:45 ASEPA Didactic Lecture Chair: AZIZ, H (Pakistan)

> **Autoimmune seizure disorders** *TAN, C T (Malaysia)*

#### 09:00 - 10:30 Main Session

### Function Room 4

**Epilepsy and the developing brain** *Chair: HARVEY, S (Australia)* 

The effects of seizures on the developing brain ONO, *T* (Japan)

The effects of foetal exposure to AEDs on pre and post natal brain development *O'BRIEN*, *T* (*Australia*)

The impact of treatment on developmental outcome in catastrophic epilepsies *KIM, H D (South Korea)* 

#### 11:00 - 12:30 Post Main Session

### Function Room 4

#### Epilepsy and autism

Chair: BERKOVIC, S (Australia)

Epilepsy and autism - when and how do they keep company? *BERROYA, A (Philippines)* 

Genes for epilepsy and autism spectrum disorders: are they shared? BERKOVIC, S (Australia)

Neuropsychiatric comorbidities in autism spectrum disorders without intellectual disability *KAMIO*, *Y* (*Japan*)

#### 11:00 – 12:30 Parallel Session

#### Meeting Rooms 4-6

**Challenges in the diagnosis and treatment of status epilepticus** *Chair: LIM, S H (Singapore)* 

Diagnosis of Status Epilepticus (SE): what are the EEG criteria? DAVID, I (Philippines)

Initial treatment of Generalized Convulsive SE (GCSE) and Non-Convulsive SE (NCSE): evidence-based AED selection *KIM, J M* (South Korea)

Refractory GCSE & NCSE: how to stop them? *TRIPATHI*, *M* (*India*)

#### 11:00 – 12:30 Parallel Session

Meeting Rooms 7-9

**Sex and epilepsy** *Chair: COLE, R (Australia)* 

Sexual dysfunction of people with epilepsy in China *DING*, *D*(*China*)

Beyond the clinical: counselling someone with epilepsy to deal with their sexual problems *WALKER*, *C* (*Australia*)

Sex after surgery WILSON, S (Australia)



# 7º Congreso Latinoamericano de Epilepsia

# 14 – 17 de Noviembre 2012 14<sup>th</sup> – 17<sup>th</sup> November 2012 Quito, Ecuador 🛲



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Si desea recibir mas informacion sobre el 7º Congreso Latinoamericano de Epilepsia, por favor póngase en contacto con **quito@epilepsycongress.org** If you would like to receive more information on the 7<sup>th</sup> Latin American Congress on Epilepsy, please contact **quito@epilepsycongress.org** 

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NAME	DATE	TIME	ROOM	SESSION TYPE C	HAIR/SPEAKER
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BERKOVIC S (AUSTRALIA)	25/03/2012	11:00	Function Room 4	Post Main Session	Speaker
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NAME	DATE	TIME	ROOM	SESSION TYPE CH	AIR/SPEAKER
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KIM H D (SOUTH KOREA)	25/03/2012	09:00	Function Room 4	Main Session	Speaker
KIM J M (KOREA)	25/03/2012	11:00	Meeting Rooms 4-6	Parallel Session	Speaker
KING P (AUSTRALIA)	23/03/2012	11:00	Function Room 1	Parallel Session	Speaker
KOO D L (SOUTH KOREA)	23/03/2012	15:00	Function Room 1	Platform Session	Speaker
KUSUMASTUTI K (INDONESIA)	24/03/2012	15:30	Meeting Rooms 7.9	Enilensy & Society Symposium	Chair
KWAN P (HONG KONG)	22/03/2012	08:00	Meeting Room 5	ASEPA Workshop: Clinical Epilepsy	Chair
KWAN P (HONG KONG)	22/03/2012	08:00	Meeting Room 5	ASEPA Workshop: Clinical Epilepsy	Speaker
KWAN P (HONG KONG)	23/03/2012	13:30	Function Room 4	Satellite Symposium	Speaker
KWAN P (HONG KONG)	23/03/2012	16:30	Function Room 2	Practical Session: Debate	Speaker
KWAN P (HONG KONG)	24/03/2012	11.00	Function Room 3	Parallel Session	Speaker
LABINER D (USA)	24/03/2012	13:30	Function Room 4	Satellite Symposium	Speaker
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NAME	DATE	TIME	ROOM	SESSION TYPE CH	AIR/SPEAKER
LAI S (TAIWAN)	24/03/2012	10:00	Meeting Rooms 7.9	Epilepsy & Society Symposium	Chair
LEDESMA L (PHILIPPINES)	24/03/2012	09:00	Function Room 4	Main Session	Speaker
LEDESMA L (PHILIPPINES)	24/03/2012	13:30	Meeting Rooms 7.9	Epilepsy & Society Symposium	Speaker
LEE B I (SOUTH KOREA)	22/03/2012	14:00	Function Room 4	Main Session	Chair
LEE B I (SOUTH KOREA)	22/03/2012	16:30	Meeting Rooms 8.9	Special Session: Advocacy	Chair
LEE B I (SOUTH KOREA)	23/03/2012	07:30	Function Room 3	ASEPA Didactic Lecture	Speaker
LEE B I (SOUTH KOREA)	23/03/2012	13:30	Function Room 4	Satellite Symposium	Speaker
LEE B I (SOUTH KOREA)	23/03/2012	16:30	Function Room 2	Practical Session: Debate	Moderator
LEE B I (SOUTH KOREA)	24/03/2012	09:00	Function Room 4	Main Session	Speaker
LEE B I (SOUTH KOREA)	24/03/2012	10:00	Meeting Rooms 7.9	Epilepsy & Society Symposium	Speaker
LEE M (SOUTH KOREA)	24/03/2012	16:30	Function Room 1	Practical Session	Speaker
LEE M K (SOUTH KOREA)	23/03/2012	15:00	Function Room 3	Platform Session	Speaker
LEE S K (SOUTH KOREA)	23/03/2012	11:00	Function Room 2	Parallel Session	Speaker
LI S (CHINA)	22/03/2012	16:30	Meeting Rooms 8-9	Special Session: Advocacy	Speaker
LI S (CHINA)	24/03/2012	15:00	Function Room 1	Platform Session	Chair
LIAO W (CHINA)	22/03/2012	08:30	Meeting Room 4	ASEPA Workshop: Basic Sciece	Chair
LIAO W (CHINA)	22/03/2012	08:30	Meeting Room 4	ASEPA Workshop: Basic Sciece	Speaker
LIAO W (CHINA)	23/03/2012	09:00	Function Room 4	Main Session	Speaker
LIAO W (CHINA)	24/03/2012	11:00	Function Room 3	Parallel Session	Chair
LIM S H (SINGAPORE)	22/03/2012	15:30	Function Room 4	The Masakazu Seino Memorial Lecture	Chair
LIM S H (SINGAPORE)	25/03/2012	11:00	Meeting Rooms 4-6	Parallel Session	Chair
	23/03/2012	17:30	Function Room 4	Satellite Symposium	Speaker
	24/03/2012	16:30	Function Room 2	Practical Session: Debate	Moderator
LUAN G (CHINA)	24/03/2012	11.00	Function Room 1	Parallel Session	Sneaker
MAGBITANG R (PHILIPPINES)	24/03/2012	10.00	Meeting Rooms 7.9	Enilensy & Society Symposium	Sneaker
MANNAN M (BANGLADESH)	23/03/2012	07:30	Function Boom 3	ASEPA Didactic Lecture	Chair
MARASIGAN S (PHILIPPINES)	24/03/2012	11.00	Function Room 4	Post Main Session	Speaker
MATHERN G (USA)	22/03/2012	14.00	Function Room 4	Main Session	Speaker
MATHERN G (USA)	22/03/2012	16.30	Meeting Rooms 8.9	Special Session: Advocacy	Speaker
MEHNDIRATTA M M (INDIA)	24/03/2012	16:30	Function Room 1	Practical Session	Speaker
	22/03/2012	16:30	Function Room 4	Special Session: Research	Speaker
	22/03/2012	16:30	Function Room 4	Special Session: Research	Speaker
	24/03/2012	15.00	Function Room 2	Platform Sossion	Speaker
	24/03/2012	11:00	Function Room 1	Parallal Session	Speaker
	22/03/2012	09.20	Monting Poom 4	ASEPA Workshop: Pasic Scioco	Speaker
	22/03/2012	16.20	Monting Rooms 8 0	Special Session: Advecacy	Chair
	22/03/2012	15.00	Eurotion Poom 1	Platform Sossion	Spoakor
	24/03/2012	16.20	Function Room 4	Special Session Research	Chair
	22/03/2012	15.00	Function Room 2	Distform Session	Chair
	24/03/2012	15.00	Function Room 2	Platform Cossion	Craalian
	24/03/2012	15:00	Function Room 2	Platform Session	Speaker
	24/03/2012	16:30	Function Room 2	Practical Session: Debate	Speaker
NEOPANE, A (NEPAL)	24/03/2012	07:30	Function Room 3	ASEPA Didactic Lecture	Chair
	22/03/2012	08:30	Meeting Room 4	ASEPA Workshop: Basic Sciece	Speaker
	22/03/2012	15.00	Meeting Room 4	ASEPA Workshop: Basic Sciece	Chair
O'BRIEN T (AUSTRALIA)	23/03/2012	15:00	Function Room 2	Platform Session	Chair
U'BRIEN I (AUSTRALIA)	25/03/2012	09:00	Function Room 4	Main Session	Speaker
	23/03/2012	09:00	Function Room 4	Iviain Session	Speaker
UNG L C (MALAYSIA)	24/03/2012	11:00	Function Room 4	Post Main Session	Speaker
UNG L C (MALAYSIA)	24/03/2012	13:30	Meeting Rooms 7-9	Epilepsy & Society Symposium	Speaker
UNU T (JAPAN)	25/03/2012	09:00	Function Room 4	Main Session	Speaker
ORTIZ M (PHILIPPINES)	24/03/2012	16:30	Function Room 1	Practical Session	Chair

PALMA & E R (PHILIPPINES)     24/03/2012     08/45     Meeting Rooms 7.9     Epilepsy & Society Symposium     Speaker       PARA (G.INGAPORE)     24/03/2012     16:30     Function Rooms 7.9     Practical Session: Video     Chair       PARUS H (PHILIPPINES)     24/03/2012     11:00     Function Rooms 7.9     Practical Session: Video     Chair       PRARUS H (PHILIPPINES)     22/03/2012     16:30     Meeting Rooms 7.9     Parallel Session: Chair     Speaker       PERUCCA E (ITALY)     22/03/2012     16:30     Function Room 3     Practical Session: Chair     Speaker       OLI L (CHINA)     23/03/2012     15:00     Function Room 1     Practical Session: Chair     RADHARKISHNAK K (NDIA)     24/03/2012     15:00     Function Room 3     Platform Session     Chair       RNEY K J (AUSTRALIA)     23/03/2012     15:00     Function Room 4     Post Main Session     Speaker       SATEIGHANDAR P (NDIA)     23/03/2012     15:00     Function Room 4     Main Session     Speaker       SOMERVILLE E (AUSTRALIA)     23/03/2012     15:00     Function Room 1     Platform Session     Speaker       SOMERVILLE E	NAME	DATE	TIME	ROOM	SESSION TYPE CI	HAIR/SPEAKER
PNA K (SINGAPORE)     24/03/2012     16:30     Function Room 3     Practical Session: Video     Presenter       PARAQUA (PHULEPINES)     24/03/2012     11:00     Function Room 3     Parallel Session     Speaker       PERUCCA E (ITALY)     22/03/2012     11:00     Meeting Room 5     Special Session: Advocacy     Speaker       PERUCCA E (ITALY)     22/03/2012     16:30     Meeting Room 5     Special Session: Advocacy     Speaker       OIN L (CHINA)     23/03/2012     16:00     Function Room 3     Practical Session     Chair       RADMAKRISHNAN K (INDIA)     24/03/2012     15:00     Function Room 3     Platform Session     Chair       RADMAKRISHNAN K (INDIA)     24/03/2012     15:00     Function Room 4     Platform Session     Chair       SATEHCHANDRA P (INDIA)     24/03/2012     15:00     Function Room 4     Main Session     Speaker       SATERIA V (INDIA)     23/03/2012     15:00     Function Room 1     Platform Session     Speaker       SATERIA V (INDIA)     23/03/2012     15:00     Function Room 1     Platform Session     Speaker       SOMERVILLE E (AUSTRA	PALMA A E R (PHILIPPINES)	24/03/2012	08:45	Meeting Rooms 7.9	Epilepsy & Society Symposium	Speaker
PARABUA H (PHILIPPINES)     24/03/2012     08.48     Meeting Rooms 7     Epilepsy & Society Symposium     Chair       PRRX S P (GOUTK KOREA)     22/03/2012     11:00     Meeting Rooms 5     ASEPA Workshop: Clinical Epilepsy     Speaker       PERUCCA E (ITALY)     22/03/2012     16:30     Meeting Rooms 5     ASEPA Workshop: Clinical Epilepsy     Speaker       PERUCCA E (ITALY)     22/03/2012     16:30     Function Room 1     Pratical Session: Chair       RADHAMKRISHNAN K (INDIA)     24/03/2012     15:00     Function Room 3     Platform Session     Speaker       SANCHEZ B (PHILIPPINES)     23/03/2012     15:00     Function Room 3     Platform Session     Speaker       SANCHEZ B (PHILIPPINES)     23/03/2012     15:00     Function Room 2     Platform Session     Speaker       SANCHEZ B (PHILIPPINES)     23/03/2012     15:00     Function Room 3     Platform Session     Speaker       SANCHEZ B (PHILIPPINES)     23/03/2012     15:00     Function Room 1     Practical Session:     Speaker       SOMERVILLE E (AUSTRALIA)     23/03/2012     15:00     Function Room 1     Practical Session:     Speaker <	PAN A (SINGAPORE)	24/03/2012	16:30	Function Room 3	Practical Session: Video	Presenter
PARK S P (SOUTH KOREA)     23/03/2012     11:00     Function Room 3     Parallel Session     Speaker       PERUCCA E (ITALY)     22/03/2012     16:30     Meeting Rooms 5     Speakal Session: Clainical Epilepsy     Speaker       PERUCCA E (ITALY)     23/03/2012     16:30     Function Room 2     Practical Session: Clainical Epilepsy     Speaker       ON IL (CHINA)     23/03/2012     11:00     Function Room 3     Platform Session     Chair       RADHAKRISHNAN K (INDIA)     24/03/2012     11:00     Function Room 3     Platform Session     Chair       SATISHCHANDRA P (INDIA)     23/03/2012     11:00     Function Room 4     Post Main Session     Speaker       SATISHCHANDRA P (INDIA)     24/03/2012     15:00     Function Room 4     Main Session     Speaker       SATISHCHANDRA P (INDIA)     23/03/2012     15:00     Function Room 1     Platform Session     Speaker       SOMERVILLE E (AUSTRALIA)     23/03/2012     15:00     Function Room 1     Platform Session     Speaker       SOMERVILLE E (AUSTRALIA)     23/03/2012     15:00     Function Room 1     Platform Session     Speaker	PARAGUA H (PHILIPPINES)	24/03/2012	08:45	Meeting Rooms 7.9	Epilepsy & Society Symposium	Chair
PERUCCA E (TALY)     22/03/2012     10:30     Meeting Rooms 5     SEEPA Workshop: Clinical Epliphey     Speaker       PERUCCA E (TALY)     22/03/2012     16:30     Meeting Rooms 8/9     Special Session: Advocacy     Speaker       QIN L (CHINA)     23/03/2012     15:30     Function Room 2     Platform Session     Chair       RINEY KJ (AUSTRALIA)     24/03/2012     15:00     Function Room 3     Platform Session     Speaker       SANCHEZ B (PHILPPINES)     23/03/2012     15:00     Function Room 3     Platform Session     Speaker       SATISHCHANDRAP (INDIA)     24/03/2012     15:00     Function Room 4     Post Main Session     Speaker       SATISHCHANDRAP (INDIA)     24/03/2012     15:00     Function Room 4     Main Session     Speaker       SOMERVILLE E (AUSTRALIA)     23/03/2012     15:00     Function Room 1     Practical Session: Workshop     Speaker       SOMERVILLE E (AUSTRALIA)     23/03/2012     15:00     Function Room 1     Practical Session: Workshop     Speaker       SOMERVILLE E (AUSTRALIA)     23/03/2012     15:00     Function Room 1     Practical Session: Workshop     Speaker<	PARK S P (SOUTH KOREA)	23/03/2012	11:00	Function Room 3	Parallel Session	Speaker
PERUCCA E (ITALY)22/03/201215:30Meeting Rooms 8-9Special Session: AdvocanySpeakerPERUCCA E (ITALY)23/03/201215:00Function Room 1Practical Session: DebateSpeakerRADHAR(SINNAN K (INDIA)24/03/201215:00Function Room 1Parallel SessionChairRADHAR(SINNAN K (INDIA)24/03/201215:00Function Room 3Platform SessionSpeakerSANCHEZ B (PHILIPPINES)23/03/201215:00Function Room 4Post Main SessionSpeakerSANCHEZ B (PHILIPPINES)23/03/201215:00Function Room 4Post Main SessionSpeakerSANCHAR V (INDIA)24/03/201215:00Function Room 4Platform SessionSpeakerSANCHAR V (INDIA)23/03/201215:00Function Room 4Statellite SymposiumSpeakerSOMERVILLE E (AUSTRALIA)23/03/201215:00Function Room 1Practical Session: WorkshopSpeakerSOMERVILLE E (AUSTRALIA)23/03/201215:00Function Room 1Practical Session: WorkshopSpeakerSOMERVILLE E (AUSTRALIA)23/03/201215:00Function Room 1Practical Session: WorkshopSpeakerSOTO M F (PHILIPPINES)23/03/201215:00Function Room 1Practical Session: WorkshopSpeakerSOTO M F (PHILIPPINES)23/03/201215:00Function Room 1Practical Session: WorkshopSpeakerTAN T (MALAYAN)24/03/201215:00Function Room 1Platform SessionSpeakerTAN T (MALAYAN)24/03/2012 </td <td>PERUCCA E (ITALY)</td> <td>22/03/2012</td> <td>10:30</td> <td>Meeting Room 5</td> <td>ASEPA Workshop: Clinical Epileps</td> <td>y Speaker</td>	PERUCCA E (ITALY)	22/03/2012	10:30	Meeting Room 5	ASEPA Workshop: Clinical Epileps	y Speaker
PERUCAE (TALY)     23/03/2012     15:30     Function Room 2     Practical Session: Debate     Speaker       QIN L (CHINA)     23/03/2012     15:00     Function Room 3     Platform Session     Chair       RINEY L (AUSTRALIA)     24/03/2012     15:00     Function Room 3     Platform Session     Speaker       SANCHEZ B (PHILIPPINES)     23/03/2012     15:00     Function Room 3     Platform Session     Speaker       SANCHAR DR (MDIA)     24/03/2012     15:00     Function Room 4     Post Main Session     Speaker       SANENA V(INDIA)     24/03/2012     15:00     Function Room 4     Main Session     Speaker       SOMERVILE E (AUSTRALIA)     23/03/2012     15:00     Function Room 1     Practical Session: Workshop     Speaker       SOMERVILLE E (AUSTRALIA)     23/03/2012     16:30     Function Room 1     Practical Session: Workshop     Speaker       SOMER SOUTH KOREA)     24/03/2012     15:00     Function Room 1     Practical Session: Workshop     Speaker       SOMERVILLE E (AUSTRALIA)     23/03/2012     15:00     Function Room 1     Practical Session: Workshop     Speaker <t< td=""><td>PERUCCA E (ITALY)</td><td>22/03/2012</td><td>16:30</td><td>Meeting Rooms 8-9</td><td>Special Session: Advocacy</td><td>Speaker</td></t<>	PERUCCA E (ITALY)	22/03/2012	16:30	Meeting Rooms 8-9	Special Session: Advocacy	Speaker
QIN L (CHINA)23/03/2012IS:00Function Room 1Platform SessionChairRADHAKRISHNAN K (INDIA)24/03/201211:00Function Room 3Platform SessionSpeakerSANCHEZ B (PHILIPPINES)23/03/201215:00Function Room 3Platform SessionSpeakerSANCHEZ B (PHILIPPINES)23/03/201211:00Function Room 2Platform SessionSpeakerSAVENCHARDEN K (THAILAND)24/03/201215:00Function Room 2Platform SessionSpeakerSAVENKA V (INDIA)24/03/201215:00Function Room 2Platform SessionSpeakerSIM S H (AUSTRALIA)23/03/201215:30Function Room 3Platform SessionSpeakerSOMERVILLE E (AUSTRALIA)23/03/201215:30Function Room 1Practical Session: WorkshopSpeakerSOMERVILLE E (AUSTRALIA)23/03/201215:30Function Room 1Practical Session: WorkshopSpeakerSOTOM F (PHILIPPINES)23/03/201215:00Function Room 1Practical Session: WorkshopSpeakerSUGAN H (JAPAN)24/03/201215:00Function Room 1Platform SessionSpeakerTAN C (MALAYSIA)25/03/201215:00Function Room 1Platform SessionSpeakerTAN KA A (JAPAN)22/03/201215:00Function Room 1Platform SessionSpeakerTANKA A (JAPAN)22/03/201215:00Function Room 1Platform SessionSpeakerTANKA A (JAPAN)22/03/201215:00Function Room 3ASEPA Workshop: Basi	PERUCCA E (ITALY)	23/03/2012	16:30	Function Room 2	Practical Session: Debate	Speaker
RADHARIJSHNAN K (NDIA)     24/03/2012     11:00     Function Room 1     Parallel Session     Chair       RINEY K J (AUSTRALIA)     24/03/2012     15:00     Function Room 3     Platform Session     Chair       SATISHCHANDRA P (INDIA)     23/03/2012     15:00     Function Room 4     Post Main Session     Speaker       SATISHCHANDRA P (INDIA)     24/03/2012     15:00     Function Room 2     Platform Session     Speaker       SATISH (INDIA)     24/03/2012     15:00     Function Room 4     Main Session     Speaker       SIM S H (AUSTRALIA)     23/03/2012     13:30     Function Room 4     Satellite Symposium     Speaker       SOMERVILLE E (AUSTRALIA)     23/03/2012     16:30     Function Room 1     Practical Session: Workshop     Chair       SOMERVILLE E (AUSTRALIA)     23/03/2012     16:30     Function Room 1     Practical Session: Workshop     Chair       SOTO M F (PHILPPINES)     23/03/2012     16:30     Function Room 1     Platform Session     Speaker       SUGMA H (APAN)     24/03/2012     16:30     Function Room 3     ASEPA Didatcit Lecture     Speaker <t< td=""><td>QIN L (CHINA)</td><td>23/03/2012</td><td>15:00</td><td>Function Room 2</td><td>Platform Session</td><td>Chair</td></t<>	QIN L (CHINA)	23/03/2012	15:00	Function Room 2	Platform Session	Chair
RINEY KJ (AUSTRALIA)     24/03/2012     IS:00     Function Room 3     Platform Session     Speaker       SANCHEZ B (PHILIPPINES)     23/03/2012     IS:00     Function Room 4     Post Main Session     Speaker       SANKOHAREON K (THAILAND)     24/03/2012     IS:00     Function Room 2     Platform Session     Speaker       SAVENAV (INDIA)     24/03/2012     IS:00     Function Room 4     Main Session     Speaker       SOMERVILLE E (AUSTRALIA)     23/03/2012     IS:00     Function Room 4     Main Session     Speaker       SOMERVILLE E (AUSTRALIA)     23/03/2012     IS:00     Function Room 1     Praterial Session: Workshop     Speaker       SOMERVILLE E (AUSTRALIA)     23/03/2012     IS:00     Function Room 1     Praterial Session: Workshop     Speaker       SOTM F (PHILIPPINES)     23/03/2012     IS:00     Function Room 1     Praterial Session: Workshop     Speaker       SOTM F (PHILIPPINES)     23/03/2012     IS:00     Function Room 3     ASEPA Workshop: Basic Seice Speaker       TAN C (MALAYSIA)     23/03/2012     IS:00     Function Room 3     ASEPA Workshop: Basic Seice Speaker <td< td=""><td>RADHAKRISHNAN K (INDIA)</td><td>24/03/2012</td><td>11:00</td><td>Function Room 1</td><td>Parallel Session</td><td>Chair</td></td<>	RADHAKRISHNAN K (INDIA)	24/03/2012	11:00	Function Room 1	Parallel Session	Chair
SANCHEZ B (PHILIPPINES)     23/03/2012     15:00     Function Room 4     Platform Session     Chair       SATISHCHANDRA P (INDIA)     23/03/2012     11:00     Function Room 4     Platform Session     Speaker       SAXENA V (INDIA)     24/03/2012     15:00     Function Room 2     Platform Session     Speaker       SAXENA V (INDIA)     23/03/2012     15:00     Function Room 3     Platform Session     Speaker       SIM S H (AUSTRALIA)     23/03/2012     16:30     Function Room 4     Satellite Symposium     Speaker       SOMERVILLE E (AUSTRALIA)     23/03/2012     16:30     Function Room 1     Practical Session: Workshop     Speaker       SOMERVILLE E (AUSTRALIA)     23/03/2012     16:30     Function Room 1     Pratical Session: Workshop     Speaker       SOTO M F (PHILIPPINES)     23/03/2012     16:30     Function Room 1     Platform Session     Speaker       SOGAN H (JARNN)     22/03/2012     16:30     Function Room 1     Platform Session     Speaker       SOGAN H (PHILIPPINES)     23/03/2012     15:00     Function Room 3     ASEPA Didaciti Lecture     Speaker	RINEY K J (AUSTRALIA)	24/03/2012	15:00	Function Room 3	Platform Session	Speaker
SATISHCHANDRA P (INDIA)     23/03/2012     11:00     Function Room 2     Pest Main Session     Speaker       SAWANCHAREON K (THAILAND)     24/03/2012     15:00     Function Room 2     Platform Session     Chair       SCHEFFER I (AUSTRALIA)     23/03/2012     15:00     Function Room 4     Main Session     Speaker       SOMERVILLE C (AUSTRALIA)     23/03/2012     15:00     Function Room 4     Satellite Symposium     Speaker       SOMERVILLE C (AUSTRALIA)     23/03/2012     16:30     Function Room 1     Practical Session: Workshop     Speaker       SOMERVILLE C (AUSTRALIA)     23/03/2012     16:30     Function Room 1     Pratical Session: Workshop     Speaker       SOMER P(SUNT KOREA)     24/03/2012     16:30     Function Room 1     Platform Session     Speaker       SOTO M F (PHILIPPINES)     23/03/2012     16:30     Function Room 1     Platform Session     Speaker       SOLGANO H (APANN)     22/03/2012     10:30     Function Room 3     ASEPA Didactic Lecture     Speaker       TAN K C (IMALANN)     22/03/2012     10:30     Function Room 4     ASEPA Workshop: Basic Sciece     Speaker	SANCHEZ B (PHILIPPINES)	23/03/2012	15:00	Function Room 3	Platform Session	Chair
SAWANCHAREON K (THAILAND)24/03/201215:00Function Room 2Platform SessionSpeakerSAXENA V (INDIA)23/03/201215:00Function Room 4Main SessionSpeakerSIM S H (AUSTRALIA)23/03/201215:00Function Room 4Satellite SymposiumSpeakerSOMERVILLE E (AUSTRALIA)23/03/201216:30Function Room 1Practical Session: WorkshopSpeakerSOMERVILLE E (AUSTRALIA)23/03/201216:30Function Room 1Practical Session: WorkshopSpeakerSOMERVILLE E (AUSTRALIA)23/03/201216:30Function Room 1Practical Session: WorkshopSpeakerSOMERVILLE E (AUSTRALIA)23/03/201216:30Function Room 1Practical Session: WorkshopSpeakerSOTD M F (PHILIPPINES)23/03/201215:00Function Room 1Platform SessionSpeakerSUGANO H (APAN)24/03/201215:00Function Room 1Platform SessionSpeakerTAN N C (SINGAPORE)23/03/201215:00Function Room 1Platform SessionSpeakerTANKAT (JAPAN)22/03/201215:00Function Room 2Platform SessionSpeakerTANKAT (JAPAN)22/03/201216:00Function Room 3ASEPA Vorkshop: BeakerSpeakerTOMAKAT (JAPAN)22/03/201217:00Function Room 4ASIEPA Vorkshop: BeakerSpeakerTOMAKA T (JAPAN)22/03/201215:00Function Room 3ASEPA Vorkshop: BeakerSpeakerTOMAKA T (JAPAN)22/03/201215:00Function Room 3	SATISHCHANDRA P (INDIA)	23/03/2012	11:00	Function Room 4	Post Main Session	Speaker
SAXENA V (INDIA)     24/03/2012     15:00     Function Room 2     Platform Session     Speaker       SCHEFFER I (AUSTRALIA)     23/03/2012     10:00     Function Room 4     Main Session     Speaker       SOMERVILLE E (AUSTRALIA)     23/03/2012     15:00     Function Room 1     Practical Session: Workshop     Speaker       SOMERVILLE E (AUSTRALIA)     23/03/2012     16:30     Function Room 1     Practical Session: Workshop     Speaker       SOMERVILLE E (AUSTRALIA)     23/03/2012     16:30     Function Room 1     Practical Session: Workshop     Speaker       SOTG M F (PHILIPPINES)     23/03/2012     16:30     Function Room 1     Pratidorm Session     Speaker       SOTG M F (PHILIPPINES)     23/03/2012     16:30     Function Room 1     Pratidorm Session     Speaker       TAN C T (MALAYSIA)     25/03/2012     16:30     Function Room 3     ASEPA Didactic Lecture     Speaker       TANAK T (JAPAN)     23/03/2012     15:00     Function Room 3     Platform Session     Speaker       TANAK T (JAPAN)     22/03/2012     15:30     Meeting Room 7     Platform Session     Speaker <t< td=""><td>SAWANCHAREON K (THAILAND)</td><td>24/03/2012</td><td>15:00</td><td>Function Room 2</td><td>Platform Session</td><td>Speaker</td></t<>	SAWANCHAREON K (THAILAND)	24/03/2012	15:00	Function Room 2	Platform Session	Speaker
SCHEFFER I (AUSTRALIA)23/03/201209:00Function Room 4Main SessionSpeakerSIM S H (AUSTRALIA)23/03/201215:00Function Room 4Stallitle SymposiumSpeakerSOMERVILLE E (AUSTRALIA)23/03/201216:30Function Room 1Practical Session: WorkshopSpeakerSOMERVILLE E (AUSTRALIA)23/03/201216:30Function Room 1Practical Session: WorkshopSpeakerSOME VILLE E (AUSTRALIA)23/03/201216:30Function Room 1Practical Session: WorkshopSpeakerSOTO M F (PHILIPPINES)23/03/201216:30Function Room 1Practical Session: WorkshopSpeakerSOTO M F (PHILIPPINES)23/03/201216:30Function Room 1Practical Session: WorkshopSpeakerTAN C T (MALAYSIA)25/03/201216:30Function Room 3ASEPA bidactic LectureSpeakerTAN K C (SINGAPORE)23/03/201215:00Function Room 3Platform SessionSpeakerTANAKA T (JAPAN)22/03/201216:00Function Room 4ASEPA Workshop: Basic SciceeSpeakerTANAKA T (JAPAN)22/03/201216:30Meeting Room 7-9Platform SessionSpeakerTHOMAS S V (INDIA)24/03/201216:30Meeting Room 7-9Parallel SessionSpeakerTRINATH M (INDIA)23/03/201216:30Meeting Room 7-9Parallel SessionSpeakerTRINATH M (INDIA)23/03/201216:30Meeting Room 7-9Parallel SessionSpeakerTRINATH M (INDIA)23/03/201216:30 </td <td>SAXENA V (INDIA)</td> <td>24/03/2012</td> <td>15:00</td> <td>Function Room 2</td> <td>Platform Session</td> <td>Chair</td>	SAXENA V (INDIA)	24/03/2012	15:00	Function Room 2	Platform Session	Chair
SIM S H (AUSTRALIA)     23/03/2012     15:00     Function Room 3     Platform Session     Speaker       SOMERVILLE E (AUSTRALIA)     23/03/2012     16:30     Function Room 1     Practical Session: Workshop     Speaker       SOMERVILLE E (AUSTRALIA)     23/03/2012     16:30     Function Room 1     Practical Session: Workshop     Chair       SOME FVILLE E (AUSTRALIA)     23/03/2012     16:30     Function Room 1     Parallel Session: Workshop     Speaker       SOTO M F (PHILIPPINES)     23/03/2012     16:30     Function Room 1     Parallel Session: Workshop     Speaker       SUGANO H (JAPAN)     24/03/2012     15:00     Function Room 1     Platform Session     Speaker       TAN C (SINGAPORE)     23/03/2012     15:00     Function Room 3     ASEPA Didactic Lecture     Speaker       TANKA A (JAPAN)     22/03/2012     15:00     Function Room 4     ASEPA Didactic Lecture     Speaker       THOMAS S V (INDIA)     24/03/2012     11:00     Function Room 4     ASEPA Didactic Lecture     Chair       TRIPATH M (NDIA)     24/03/2012     11:00     Function Room 4     Satellite Symposium     Speaker	SCHEFFER I (AUSTRALIA)	23/03/2012	09:00	Function Room 4	Main Session	Speaker
SOMERVILLE E (AUSTRALIA)23/03/201213:30Function Room 1Satellite SymposiumSpeakerSOMERVILLE E (AUSTRALIA)23/03/201216:30Function Room 1Practical Session: WorkshopChairSOMERVILLE E (AUSTRALIA)23/03/201216:30Function Room 1Practical Session: WorkshopChairSOTO M F (PHILIPPINES)23/03/201216:30Function Room 1Parallel SessionSpeakerSOTO M F (PHILIPPINES)23/03/201216:30Function Room 1Practical Session: WorkshopSpeakerSUGANO H (JAPAN)24/03/201215:00Function Room 1Platform SessionSpeakerTAN C T (MALAYSIA)25/03/201215:00Function Room 3ASEPA Didactic LectureSpeakerTANAKA T (JAPAN)22/03/201215:00Function Room 3Platform SessionSpeakerTANAKA T (JAPAN)22/03/201216:00Function Room 4ASEPA Didactic LectureSpeakerTHOMAS S V (INDIA)24/03/201211:00Function Room 7Platform SessionSpeakerTOVUUDORJ A (MONGOLIA)24/03/201217:30Function Room 4ASEPA Didactic LectureChairTRIPATHI M (INDIA)22/03/201217:30Function Room 3ASEPA Didactic LectureChairTRIPATHI M (INDIA)22/03/201217:30Function Room 4Satellite SymposiumSpeakerTRIPATHI M (INDIA)22/03/201216:30Meeting Rooms 7.9Speatical SessionSpeakerTRIPATHI M (INDIA)22/03/201216:30Function Roo	SIM S H (AUSTRALIA)	23/03/2012	15:00	Function Room 3	Platform Session	Speaker
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TRIPATHI M (INDIA)23/03/201217:30Function Room 4Satellite SymposiumSpeakerTRIPATHI M (INDIA)25/03/201211:00Meeting Rooms 4-6Parallel SessionSpeakerTRONCO A (PHILIPPINES)24/03/201213:30Meeting Rooms 8-9Special Session: AdvocacySpeakerTSAI J J (TAIWAN)22/03/201216:30Meeting Rooms 8-9Special Session: AdvocacySpeakerTSENG Y F (TAIWAN)23/03/201216:30Function Room 1Practical Session: WorkshopChairVILANILAM G C (INDIA)24/03/201216:30Function Room 1Platform SessionSpeakerWAHAB A (PAKISTAN)23/03/201215:00Function Room 2Platform SessionSpeakerWALKER C (AUSTRALIA)24/03/201215:00Function Room 3Platform SessionSpeakerWALKER C (AUSTRALIA)25/03/201215:00Function Room 3Platform SessionSpeakerWAINGASINGHE J (SRI LANKA)23/03/201215:00Function Room 3Platform SessionSpeakerWILSON S (AUSTRALIA)23/03/201211:00Function Room 3Parallel SessionSpeakerWILSON S (AUSTRALIA)22/03/201209:00Meeting Rooms 7-9Parallel SessionSpeakerWILSON S (AUSTRALIA)22/03/201211:00Function Room 3ASEPA Didactic LectureSpeakerWILSON S (AUSTRALIA)22/03/201211:00Function Room 4Post Main SessionSpeakerWILSON S (AUSTRALIA)22/03/201211:00Function Room 3<	TOVUUDORJ A (MONGOLIA)	24/03/2012	08:15	Function Room 3	ASEPA Didactic Lecture	Chair
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WALKER C (AUSTRALIA)24/03/201215:00Function Room 2Platform SessionSpeakerWALKER C (AUSTRALIA)25/03/201211:00Meeting Rooms 7:9Parallel SessionSpeakerWANIGASINGHE J (SRI LANKA)23/03/201215:00Function Room 3Platform SessionSpeakerWATANABE M (JAPAN)23/03/201211:00Function Room 3Parallel SessionChairWIEBE S (CANADA)23/03/201208:15Function Room 3ASEPA Didactic LectureSpeakerWILSON S (AUSTRALIA)22/03/201209:00Meeting Room 5ASEPA Workshop: Clinical EpilepsySpeakerWILSON S (AUSTRALIA)24/03/201211:00Function Room 4Post Main SessionSpeakerWILSON S (AUSTRALIA)25/03/201211:00Meeting Rooms 7:9Parallel SessionSpeakerYAMAKAWA K (JAPAN)22/03/201208:30Meeting Rooms 7:9Parallel SessionSpeakerYAMAKAWA K (JAPAN)22/03/201211:00Function Room 1Parallel SessionSpeakerYANG T (CHINA)23/03/201215:00Function Room 1Parallel SessionSpeakerYANG T (CHINA)23/03/201215:00Function Room 1Parallel SessionSpeakerYANG T (CHINA)23/03/201211:00Function Room 1Parallel SessionSpeaker	WAHAB A (PAKISTAN)	23/03/2012	15:00	Function Room 2	Platform Session	Speaker
WALKER C (AUSTRALIA)   25/03/2012   11:00   Meeting Rooms 7.9   Parallel Session   Speaker     WANIGASINGHE J (SRI LANKA)   23/03/2012   15:00   Function Room 3   Platform Session   Speaker     WATANABE M (JAPAN)   23/03/2012   11:00   Function Room 3   Parallel Session   Chair     WIEBE S (CANADA)   23/03/2012   08:15   Function Room 3   ASEPA Didactic Lecture   Speaker     WILSON S (AUSTRALIA)   22/03/2012   09:00   Meeting Room 5   ASEPA Workshop: Clinical Epilepsy   Speaker     WILSON S (AUSTRALIA)   24/03/2012   11:00   Function Room 4   Post Main Session   Speaker     WILSON S (AUSTRALIA)   25/03/2012   11:00   Meeting Rooms 7.9   Parallel Session   Speaker     YAMAKAWA K (JAPAN)   22/03/2012   08:30   Meeting Room 7.9   Parallel Session   Speaker     YAMAKAWA K (JAPAN)   22/03/2012   08:30   Meeting Room 3   ASEPA Workshop: Basic Sciece   Speaker     YAMAKAWA K (JAPAN)   22/03/2012   11:00   Function Room 1   Parallel Session   Speaker     YAMAKAWA K (JAPAN)   23/03/2012   11:00   Function Room 1	WALKER C (AUSTRALIA)	24/03/2012	15:00	Function Room 2	Platform Session	Speaker
WANIGASINGHE J (SRI LANKA)   23/03/2012   15:00   Function Room 3   Platform Session   Speaker     WATANABE M (JAPAN)   23/03/2012   11:00   Function Room 3   Parallel Session   Chair     WIEBE S (CANADA)   23/03/2012   08:15   Function Room 3   ASEPA Didactic Lecture   Speaker     WILSON S (AUSTRALIA)   22/03/2012   09:00   Meeting Room 5   ASEPA Workshop: Clinical Epilepsy   Speaker     WILSON S (AUSTRALIA)   24/03/2012   11:00   Function Room 4   Post Main Session   Speaker     WILSON S (AUSTRALIA)   25/03/2012   11:00   Meeting Rooms 7·9   Parallel Session   Speaker     YAMAKAWA K (JAPAN)   22/03/2012   08:30   Meeting Rooms 7·9   Parallel Session   Speaker     YAMAKAWA K (JAPAN)   22/03/2012   08:30   Meeting Room 3   Parallel Session   Speaker     YAMAKAWA K (JAPAN)   22/03/2012   11:00   Function Room 1   Parallel Session   Speaker     YAMAKAWA K (JAPAN)   23/03/2012   11:00   Function Room 1   Parallel Session   Speaker     YANG T (CHINA)   23/03/2012   15:00   Function Room 1   Parallel Se	WALKER C (AUSTRALIA)	25/03/2012	11:00	Meeting Rooms 7.9	Parallel Session	Speaker
WATANABE M (JAPAN)   23/03/2012   11:00   Function Room 3   Parallel Session   Chair     WIEBE S (CANADA)   23/03/2012   08:15   Function Room 3   ASEPA Didactic Lecture   Speaker     WILSON S (AUSTRALIA)   22/03/2012   09:00   Meeting Room 5   ASEPA Workshop: Clinical Epilepsy   Speaker     WILSON S (AUSTRALIA)   24/03/2012   11:00   Function Room 4   Post Main Session   Speaker     WILSON S (AUSTRALIA)   25/03/2012   11:00   Meeting Rooms 7-9   Parallel Session   Speaker     YAMAKAWA K (JAPAN)   22/03/2012   08:30   Meeting Rooms 7-9   Parallel Session   Speaker     YAMAKAWA K (JAPAN)   22/03/2012   08:30   Meeting Room 3   Parallel Session   Speaker     YAMAKAWA K (JAPAN)   22/03/2012   11:00   Function Room 3   Parallel Session   Speaker     YAMAK (JAPAN)   23/03/2012   11:00   Function Room 1   Parallel Session   Speaker     YANG T (CHINA)   23/03/2012   15:00   Function Room 1   Parallel Session   Speaker     ZHANG Y H (CHINA)   23/03/2012   11:00   Function Room 1   Parallel Session	WANIGASINGHE J (SRI LANKA)	23/03/2012	15:00	Function Room 3	Platform Session	Speaker
WIEBE S (CANADA)   23/03/2012   08:15   Function Room 3   ASEPA Didactic Lecture   Speaker     WILSON S (AUSTRALIA)   22/03/2012   09:00   Meeting Room 5   ASEPA Workshop: Clinical Epilepsy   Speaker     WILSON S (AUSTRALIA)   24/03/2012   11:00   Function Room 4   Post Main Session   Speaker     WILSON S (AUSTRALIA)   25/03/2012   11:00   Function Room 4   Post Main Session   Speaker     WILSON S (AUSTRALIA)   25/03/2012   11:00   Meeting Room 7-9   Parallel Session   Speaker     YAMAKAWA K (JAPAN)   22/03/2012   08:30   Meeting Room 3   ASEPA Workshop: Basic Sciece   Speaker     YAMAKAWA K (JAPAN)   22/03/2012   11:00   Function Room 3   Parallel Session   Speaker     YANG T (CHINA)   23/03/2012   11:00   Function Room 1   Platform Session   Speaker     ZHANG Y H (CHINA)   23/03/2012   11:00   Function Room 1   Parallel Session   Speaker	WATANABE M (JAPAN)	23/03/2012	11:00	Function Room 3	Parallel Session	Chair
WILSON S (AUSTRALIA)   22/03/2012   09:00   Meeting Room 5   ASEPA Workshop: Clinical Epilepsy   Speaker     WILSON S (AUSTRALIA)   24/03/2012   11:00   Function Room 4   Post Main Session   Speaker     WILSON S (AUSTRALIA)   25/03/2012   11:00   Function Room 4   Post Main Session   Speaker     YAMAKAWA K (JAPAN)   22/03/2012   11:00   Meeting Room 7:9   Parallel Session   Speaker     YAMAKAWA K (JAPAN)   22/03/2012   11:00   Function Room 3   Parallel Session   Speaker     YANG T (CHINA)   23/03/2012   11:00   Function Room 1   Platform Session   Speaker     ZHANG Y H (CHINA)   23/03/2012   15:00   Function Room 1   Parallel Session   Speaker	WIEBE S (CANADA)	23/03/2012	08:15	Function Room 3	ASEPA Didactic Lecture	Speaker
WILSON S (AUSTRALIA)   24/03/2012   11:00   Function Room 4   Post Main Session   Speaker     WILSON S (AUSTRALIA)   25/03/2012   11:00   Function Room 4   Post Main Session   Speaker     YAMAKAWA K (JAPAN)   22/03/2012   10:00   Meeting Room 4   ASEPA Workshop: Basic Sciece   Speaker     YAMAKAWA K (JAPAN)   24/03/2012   11:00   Function Room 3   Parallel Session   Speaker     YAMAKAWA K (JAPAN)   24/03/2012   11:00   Function Room 3   Parallel Session   Speaker     YANG T (CHINA)   23/03/2012   15:00   Function Room 1   Platform Session   Speaker     ZHANG Y H (CHINA)   23/03/2012   11:00   Function Room 1   Parallel Session   Speaker	WILSON S (AUSTRALIA)	22/03/2012	09:00	Meeting Room 5	ASEPA Workshop: Clinical Epileos	v Speaker
WILSON S (AUSTRALIA) 25/03/2012 11:00 Meeting Rooms 7-9 Parallel Session Speaker   YAMAKAWA K (JAPAN) 22/03/2012 08:30 Meeting Room 4 ASEPA Workshop: Basic Sciece Speaker   YAMAKAWA K (JAPAN) 24/03/2012 11:00 Function Room 3 Parallel Session Speaker   YAMAKAWA K (JAPAN) 23/03/2012 15:00 Function Room 1 Platform Session Speaker   ZHANG Y (CHINA) 23/03/2012 15:00 Function Room 1 Parallel Session Speaker	WILSON S (AUSTRALIA)	24/03/2012	11:00	Function Room 4	Post Main Session	Speaker
YAMAKAWA K (JAPAN) 22/03/2012 08:30 Meeting Room 4 ASEPA Workshop: Basic Sciece Speaker   YAMAKAWA K (JAPAN) 24/03/2012 11:00 Function Room 3 Parallel Session Speaker   YANG T (CHINA) 23/03/2012 15:00 Function Room 1 Platform Session Speaker   ZHANG Y H (CHINA) 23/03/2012 11:00 Function Room 1 Parallel Session Speaker	WILSON S (AUSTRALIA)	25/03/2012	11:00	Meeting Rooms 7.9	Parallel Session	Speaker
YAMAKAWA K (JAPAN)     24/03/2012     11:00     Function Room 3     Parallel Session     Speaker       YANG T (CHINA)     23/03/2012     15:00     Function Room 1     Platform Session     Speaker       ZHANG Y H (CHINA)     23/03/2012     15:00     Function Room 1     Platform Session     Speaker	YAMAKAWA K (JAPAN)	22/03/2012	08:30	Meeting Room 4	ASEPA Workshop: Basic Sciece	Speaker
ZHANG T (CHINA)     23/03/2012     15:00     Function Room 1     Platform Session     Speaker       ZHANG Y H (CHINA)     23/03/2012     11:00     Function Room 1     Parallel Session     Speaker	YAMAKAWA K (JAPAN)	24/03/2012	11:00	Function Room 3	Parallel Session	Speaker
ZHANG Y H (CHINA) 23/03/2012 11:00 Function Room 1 Parallel Session Speaker	YANG T (CHINA)	23/03/2012	15.00	Function Room 1	Platform Session	Speaker
, , , operation	ZHANG Y H (CHINA)	23/03/2012	11:00	Function Room 1	Parallel Session	Speaker

# **EXHIBITION INFORMATION**

### **EXHIBITION OPENING HOURS**

Friday, 23 <sup>rd</sup> March	09:00 - 17:00
Saturday, 24 <sup>th</sup> March	09:00 - 17:00
Sunday, 25 <sup>th</sup> March	09:00 - 12:00

Exhibitor	Stand no.
Abbott Laboratories	7
Ad-Tech Medical Instrument Corporation	13
Cadwell	11
CareFusion	17
Compumedics	14
Cyberonics	12
EB Neuro	6
Electrical Geodesics Inc	10
Elekta	9
GlaxoSmithKline	25
Hi-Eisai	22
International Bureau for Epilepsy	23
International League Against Epilepsy	23
John Libbey Eurotext	16
Novartis Healthcare Philippines, Inc.	21
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## **EXHIBITION FLOOR PLAN**



### **PLATFORM SESSIONS**

Clinical epileptology Friday 23rd March 15:00 - 16:00 Function Room 3 Please refer to pages 62 - 64 for abstracts

Chairs: SANCHEZ, B (Philippines) & DUNNE, J (Australia)

- 001 Characteristic of the new-onset epilepsy in the elderly TANAKA A, TOYOTA T, SHOZAKI T, AKAMATSU N, TSUJI S (Japan)
- 002 Prognosis of first-ever remote symptomatic seizure <u>SIM SH</u><sup>1,2</sup>, DUNNE J<sup>1</sup>, LEE J<sup>1</sup>, GAITATZIS A<sup>1</sup>, CHAN J<sup>1</sup>, LAWN N<sup>1</sup> (<sup>1</sup>Australia, <sup>2</sup>Malaysia)
- 003 Classification of generalized tonic-clonic seizures: is it focal or generalized? LEE MK, KIM EH, CHO Y-J, LEE BI, HEO K (South Korea)
- 004 Syndromic classification of epilepsy and seizure control among patients with epilepsy in a metropolitan tertiary neurology referral center in Penang, Malaysia HOR\_JY, LIM TT, EOW GB, EASAW PES, RAFIA MH (Malaysia)
- 005 Role of steroids in reversing hypsarrhythmia in previously untreated infants with epileptic spasms WANIGASINGHE J, ATTANAPOLA GM, ARAMBEPOLA C, LIYANAGE C, SUMANASENA S, SRI RANGANATHAN S (Sri Lanka)

Basic science Friday 23<sup>rd</sup> March 15:00 - 16:00 Function Room 2 Please refer to pages 64 - 67 for abstracts

Chairs: QIN, J (China) & O'BRIEN, T (Australia)

- 006 Effects of neuroprotective drugs against experimentally induced status epilepticus in young rats <u>AHMAD M</u>, WADAAN MAM (Saudi Arabia)
- 007 Antipsychotic drug related seizures may result from inhibition of potassium currents in central neurons - a voltage-clamp and computational modelling study FRENCH CR, CHAN D, LIN WC (Australia)
- 008 Pharmacoresistant seizure and its mechanism in immature brain WAHAB A<sup>1,2</sup>, ALBUS K<sup>2</sup>, HEINEMANN U<sup>2</sup> (<sup>1</sup>Pakistan, <sup>2</sup>Germany)
- 009 Monocyte chemoattractant protein-1 affects ectopic migration of neural progenitors within the hippocampal dentate gyrus of rats with status epilepticus HUNG Y-W, CHOU C-C, LAI M-T, TSENG Y-J, LIN Y-Y (Taiwan)
- 010 In vitro transport profiles of lacosamide, rufinamide, pregabalin and zonisamide by human P-glycoprotein BAUM L, ZHANG C, ZUO J, KWAN P (Hong Kong)

Clinical neurophysiology and neuroimaging Friday 23rd March 15:00 - 16:00 Function Room 1 Please refer to pages 67 - 70 for abstracts

Chairs: IKEDA, A (Japan) & HONG, S B (South Korea)

- 011 Changes of cortical silent period in patients with drug-naïve epilepsy KOO DL, UHM DO, JOO EY, HONG SB (South Korea)
- 012 Chronotopology of neurophysiologic indicators for early and late epileptogenesis and drug-resistant focal and multifocal epilepsy surgery <u>KASUMOV V</u>, STEPANOVA T, KASUMOV R, BERSNEV V, KRAVTSOVA S, SEBELEV K (Russian Federation)
- 013 Testing times: Test-Enhanced Learning as an instructional method for EEG courses TAN NC, CHIN HX, LOH NK, CHAN DW, RATHAKRISHNAN R, LIM SH (Singapore)
- 014 A rational surgery for hypothalamic hamartoma: ictogenesis and symptomatogenesis of gelastic seizures KAMEYAMA S, MURAKAMI H, MASUDA H, SHIROZU H (Japan)
- 015 White matter impairment in the basal ganglia-thalamocortical circuit of drug-naïve childhood absence epilepsy YANG T, LUO C, LIU L, GONG Q, ZHOU D (China)

#### Epidemiology and genetics Saturday 24<sup>th</sup> March 15:00 - 16:00 Function Room 3 Please refer to pages 70 - 72 for abstracts

Chairs: AZIZ, H (Pakistan) & NADKARNI, V V (India)

- 016 Sleep disordered breathing in patients with epilepsy BHUMA V, MOHAN A, VIJAYA BHASKARA RAO J (India)
- 017 A novel digital animation seizure survey may be an effective screening instrument for seizures in population-based research <u>D'SOUZA W</u>, FREEMAN J, HARVEY S, COOK M (Australia)
- 018 Vitamin D deficiency in a hospital based population of children with epilepsy on longterm antiepileptic drugs FONG C-Y, <u>RINEY KJ</u> (Australia)
- 019 Antiepileptic drug and risk of stroke in patients with epilepsy: a population-based cohort study HSIEH C-Y, LAI EC-C, KAO YANG Y-H (Taiwan)
- 020 Pharmacogenetic evaluation of fetal malformations in women with epilepsy receiving anti-epileptic drugs JOSE M, MATHEW A, VIJAYAN N, BANERJEE M, THOMAS SV (India)

Neuropsychology and social issues Saturday 24<sup>th</sup> March 15:00 - 16:00 Function Room 2 Please refer to pages 72 - 74 for abstracts

Chairs: SAXENA, V (India) & BAEL, V (Philippines)

- 021 The effects of support group on self-esteem in people with epilepsy <u>SAWANCHAREON K</u>, PRANBOON S (Thailand)
- 022 Prediction of memory change in mesial temporal lobe epilepsy after anterior temporal lobectomy with amygdalohippocampectomy CHOI SJ, <u>SONG P</u>, CHOI DS, JOO EY, HONG SB (South Korea)
- 023 Longitudinal surveys into the social impact of epilepsy: an Australian 'first' WALKER C, EPILEPSY FOUNDATION VICTORIA RESEARCH WORKING GROUP (Australia)
- 024 Beyond the biomedical: the experience of epilepsy among women in Kerala, India NAIR A, THOMAS SV (India)
- 025 Patterns of psychological adjustment following paediatric epilepsy surgery MICALLEF S, HARVEY AS, WRENNALL JA, WILSON SJ (Australia)

Surgery Saturday 24th March 15:00 - 16:00 Function Room 1 Please refer to pages 75 - 77 for abstracts

Chairs: KAMEYAMA, S (Japan) & LI, S (China)

- 026 MRI features and surgical outcome of focal cortical dysplasia according to the new ILAE classification criteria KIM DW, LEE SK (South Korea)
- 027 MRI-guided stereotactic radiofrequency thermocoagulation for 53 intractable epilepsy patients with hypothalamic hamartoma: analysis of patients with residual seizures <u>MURAKAMI H</u>, KAMEYAMA S, MASUDA H, SHIROZU H (Japan)
- 028 Resection frequency map after awake resective surgery for non-lesional neocortical epilepsy involving eloquent area CHUNG CK, KIM Y-H, KIM CH, KIM JS, LEE SK (South Korea)
- 029 Unresolved enigmata about hypothalamic hamartomas: an institutional inquest VILANILAM GC, ABRAHAM M, MENON G, NAIR S, RADHAKRISHNAN K (India)
- 030 Posterior quadrantectomy for patient with Sturge-Weber syndrome <u>SUGANO H</u>, NAKAJIMA M, NAKANISHI H, HIGO T, IIMURA Y, ARAI H (Japan)

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### **POSTER SESSIONS**

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- p031 Effect of antiepileptic drugs on memory in elderly patients with cognitive impairment and epileptic seizures KINOSHITA M, ARAKI Y, SUDOH S (Japan)
- p032 Cardiac pacemaker implantation in a patient with ictal asystole associated with multi drug-resistant temporal lobe epilepsy SHIN J-W, MOON H-J, KANG BS, LEE S-T, JUNG K-H, CHU K, LEE SK (South Korea)
- p033 Comparative analysis of quality life between patients with temporal lobe epilepsy with and without seizure BAYARMAA D (Mongolia)
- p034 Amygdalar or amygdalohippocampal enlargement in patients with temporal lobe epilepsy: a hidden cause <u>HEO K</u>, LEE S-K, LEE BI, CHO Y-J, LEE MK (South Korea)
- p035 A 20 year-old aged encephalitis patient with bilateral ovarian teratoma SHIN DJ (South Korea)
- p036 Study of factors which may influence body weight in overweight patients of epilepsy: a cross-sectional study from a tertiary care centre in New Delhi SINGH M, LAMBA A, BHATIA R, PADMA MV, TRIPATHI M, PRASAD K (India)

#### **AED** issues

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- p037 Pharmacokinetics of carbamazepine in Mongolia for people with partial epilepsy MANDAKH B, <u>MANDSHIR U</u>, ULZIIBAYAR D (Mongolia)
- p038 The efficacy, safety, and pharmacokinetics of intravenously administered Fosphenytoin Sodium in Japanese patients with status epilepticus and neurosurgery NAKAGAWA E (Japan)
- p039 Vitamin D deficiency in children with epilepsy who are taking anticonvulsants  $\underline{YU}$  J, KOH JW (South Korea)
- p040 Duration is associated with responsiveness to AED in lesional epilepsy KIM SE (South Korea)
- p041 Metabolic syndrome among obese Chinese epileptic patients treated with valproate FANG J, ZHOU D (China)
- p042 Evaluation of different antiepileptic drug strategies in medically refractory epilepsy patients following epilepsy surgery ZHOU D, ZENG T.F, AN D-M, LEI D (China)
- p043 Comparison of dried blood spot and plasma measured valproic acid levels in patients with epilepsy KONG ST, WANG HY, LIM WH, NG YL, HO PC, LIM S-H (Singapore)
- p044 A survey of the use of antiepileptic drugs in stroke patients LEE J, JANG W, KIM HY, KIM H-T, KIM J (South Korea)

p045 The EpiNet project in Asia-Oceania; a means to undertake collaborative clinical research in epilepsy

BERGIN P1, D'SOUZA W2, SADLEIR L1, TRIPATHI M3, DANG N3, MOGAL Z4, MAHMUD H4, CHO Y-J5, SRIVASTAVA K3, TAN H-J6, RAYMOND AA6, WANIGASINGHE J7, EPINET STUDY GROUP (<sup>1</sup>New Zealand, <sup>2</sup>Australia, <sup>3</sup>India, <sup>4</sup>Pakistan, <sup>5</sup>South Korea, 6Malaysia, 7Sri Lanka)

p046 Low occurrence of skin rash with antiepileptic drugs in Pakistan: do ethnic / racial variations exist?

MOGAL Z, MAHMUD H, AZIZ H (Pakistan)

- p047 A phenytoin-induced neutropenia case KIM W-J, KIM OJ, KWAK Y-T (South Korea)
- p048 The evaluation of implementing HLA-B\*1502 genotyping test at an epilepsy clinic of a university hospital TSAI C-F, FANG C-W, TSAI J-J (Taiwan)

#### Basic science

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- p049 Intervention of seizure-induced neuronal death by inhibition of NADPH oxidase activation CHOI HC, SUH SW, SONG HK (South Korea)
- p050 Anticonvulsant actions of CyPPA in pharmacoresistant human epileptic tissue RAZA ML, HEINEMANN U (Germany)
- p051 Experimental study on the neuroprotection role of Baclofen in hippocampus of epilepsy rats induced by kainic acid WANG Z (China)
- p052 Hippocampal dentate granule cell neurogenesis and phenotypic differentiation in mice after pilocarpine-induced seizures KIM DW. PARK J (South Korea)
- p053 Effect of esomeprazole on pharmacokinetics of phenytoin MEDHI B, PRAKASH A, RUPA J, PRASAD BYRAV DS (India)

#### Clinical neurophysiology

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- p054 Transient increase of serum ammonia level in differential diagnosis of seizure and syncope CHOI YH, CHO YN, LEE S-Y, KIM W-J (South Korea)
- p055 Vertiginous seizure and postural control LEE G-H (South Korea)

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- p056 Amplitude integrated electroencephalography in the neonatal intensive care unit for diagnosis of neonatal seizure KIM WS, KIM WK (South Korea)
- Automatic abnormal waves detection from the electroencephalograms of epilepsy p057 cases to sort out the spikes, the sharps, the polyphase based on wavelet transformationautocorelation

NOERTJAHJANI S, SUSANTO A, HIDAYAT R, WIBOWO S, MUTAQIN Z, UNDARIS (Indonesia)

- p058 Ictal high-gamma oscillation (60-99 Hz) in intracranial electroencephalography and postoperative seizure outcome in neocortical epilepsy <u>PARK S-C</u>, LEE SK, CHE H, CHUNG CK (South Korea)
- p059 Clinical characteristics and prognosis of patients showing periodic lateralized epileptiform discharges LEE H, LEE S (South Korea)
- p060 Six year old children of women with epilepsy have higher prevalence of epileptiform abnormalities in EEG SYAM UK, SUCHARITHADEVI S, SABARINATHAN S, SREEVIDYA D, THOMAS SV (India)
- p061 Discrepancy of simultaneous ictal recordings with scalp and subdural electrode arrays in patients with intractable temporal lobe epilepsy KOO DL, JOO EY, SEO D-W, HONG SB (South Korea)
- p062 Mapping interictal high-frequency oscillations in neocortical epilepsy CHO JR, JOO EY, HONG SC, HONG SB (South Korea)
- p063 Occipital paroxysms in dizygotic twins JOCIC-JAKUBI B<sup>1,2</sup>, JOVANOVIC M<sup>1</sup>, STANKOVIC JANKOVIC D<sup>1</sup> (<sup>1</sup>Serbia, <sup>2</sup>Kuwait)
- p064 The role of sleep deprivation on EEG recordings in possible epilepsy patients THAYEB RN, OCTAVIANA F, YAMANIE N (Indonesia)
- p065 Temporal intermittent rhythmic delta activity: prevalence and significance CHAN J, DUNUWILLE J, DUNNE J, LAWN N (Australia)
- p066 EEG pattern in seizure free epilepsy patients at Cipto Mangunkusumo Hospital INDRASARI U, <u>OCTAVIANI D</u>, BUDIKAYANTI A, OCTAVIANA F (Indonesia)

#### Epidemiology

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- p068 Toxocara and neurocysticercosis risk factors for epilepsy: a community based study <u>KAUSHAL S</u><sup>1</sup>, SINGH G<sup>1</sup>, CHHINA DK<sup>1</sup>, CHAUDHARY A<sup>1</sup>, MODI M<sup>1</sup>, BAWA J<sup>1</sup>, SANDER JW<sup>2</sup> (<sup>1</sup>India, <sup>2</sup>United Kingdom)
- p069 Seizure free in temporal lobe epilepsy patient Cipto Mangunkusumo Hospital June 2010 - May 2011 WIRATMAN W, OCTAVIANA F, BUDIKAYANTI A (Indonesia)

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- p071 Pharmacogenetic testing of antiepileptic therapy: a pilot study <u>PILYUGINA M</u>, SHNAYDER N, DMITRENKO D (Russian Federation)

- p072 Advances in the molecular genetics of the benign epilepsies of infancy <u>HERON SE<sup>1</sup></u>, GRINTON BE<sup>1</sup>, KIVITY S<sup>2</sup>, AFAWI Z<sup>2</sup>, ZUBERI SM<sup>3</sup>, PRIDMORE C<sup>1</sup>, HODGSON BL<sup>1</sup>, IONA X<sup>1</sup>, SADLEIR LG<sup>4</sup>, PELEKANOS J<sup>1</sup>, HERLENIUS E<sup>5</sup>, GOLDBERG-STERN H<sup>2</sup>, BASSAN H<sup>2</sup>, HAAN E<sup>1</sup>, KORCZYN AD<sup>2</sup>, GARDNER AE<sup>1</sup>, CORBETT M<sup>1</sup>, GECZ J<sup>1</sup>, MULLEY JC<sup>1</sup>, BERKOVIC SF<sup>1</sup>, SCHEFFER IE<sup>1</sup>, DIBBENS LM<sup>1</sup> (<sup>1</sup>Australia, <sup>2</sup>Israel, <sup>3</sup>United Kingdom, <sup>4</sup>New Zealand, <sup>5</sup>Sweden)
- p073 CAMSAP1L1 is a potential epilepsy gene in the Chinese population ZHANG S, KWAN P, GUO Y, SHAM PC, CHERNY SS, BAUM L (Hong Kong)

#### Neurobiology

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- p074 System administration of neuregulin-β1 inhibits epileptogenesis in pilocarpine induced epilepsy rat model DU P, TANG H, LI X, LIN H, LIU J, MA Y, FAN W, WANG X (China)
- p075 Attenuation of kainic acid-induced neurotoxicity by an antioxidant in organotypic hippocampal slice culture LEE KH, KIM UJ, LEE BH (South Korea)

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- p077 Crossed cerebellar abnormality in three patients with refractory partial seizures LL X, JIAO J, CHEN J, WU D, HONG W, REN L (China)
- p078 Hypertrophic amygdala in three patients with medial temporal lobe epilepsy WU D, LEE X, CHEN J, YU Y, LEE R, HONG W, REN L, JIAO J (China)
- p079 Resting-state fMRI study of catamenial epilepsy patients with correlated changes of E/P serum ratio concentration <u>MU J</u>, CHEN Q, CHEN S, YU X, ZHOU D (China)
- p080 Longitudinal assessment of brain function on patients with temporal lobe epilepsy after anterior temporal lobectomy: a resting state fMRI study YU X, ZHOU B, CHEN Q, CHEN S, WANG Y, TANG H, GONG Q, ZHOU D (China)
- p081 Inter-ictal FDG PET in temporal lobe epilepsy. Correlation with ictal EEG, MR imaging, neuropsychology and outcome SINGA PK, SUDHAKAR P, JAYALAKSHMI S, PANIGRAHI M (India)
- p082 Characteristics of altered brain function in epilepsy patients with different seizure type: evidence from resting-state fMRI CHEN Q, ZHOU B, LI Q, LUI S, YAO Z, YU X, HUANG X, GONG Q, ZHOU D (China)
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- p084 Is SISCOM (Subtraction Ictal SPECT Co-registered to MRI) helpful to the successful surgery in nonlesional extratemporal epilepsy? SONG P, JOO EY, SEO DW, HONG SB (South Korea)

- p085 Cerebral activations during Chinese verbal memory processing in temporal lobe epilepsy: a functional magnetic resonance imaging study LIN Y-Y, CHEN T-C, YIU C-H, YEN D-J, KWAN S-Y, YU H-Y, CHEN C, SU T-P, SHIH Y-H (Taiwan)
- p086 Verbal memory function in temporal lobe epilepsy: a MEG study KO HW, CHEN T.C, LIN Y.Y (Taiwan)

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- p089 Parenting stress, attitude and quality of life in children with epilepsy EOM S, KIM H, KANG H-C, LEE JS, KIM HD (South Korea)
- p090 Korean version of the neurological disorders depression inventory for epilepsy (NDDI-E) KO PW, LIM HW, KIM SH, PARK SP (South Korea)
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- p092 Analyses of personality profiles in patients with psychogenic non epileptic seizures, epileptic seizures, or combined type KANG MJ, JOO EY, HONG SB (South Korea)

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- p094 Verbal memory disorder and auditory cognitive event related potentials in epilepsy patients with secondary generalized seizure OCTAVIANA F (Indonesia)
- p095 Therapeutic drug monitoring, Epilepsy Clinic, Srinagarind Hospital, Thailand LERTSINUDOM S, CHAIYAKUM A, TUNTAPAKUL S, TIAMKAO S, EPILEPSY RESEARCH GROUP, KHON KAEN UNIVERSITY (Thailand)
- p096 A case of sleep-induced cortical myoclonus JUNG B-W, KIM J-E (South Korea)
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- p102 Experience of epilepsy treatment in Angelman syndrome in Korea KANG JW, YOON J.R, KIM SH, RHIE SK, LEE JS, LEE Y.M, KIM HD, KANG H-C (South Korea)
- p103 Remote effect of spike discharges in patients with atypical benign partial epilepsy: a SPECT study <u>HAGINOYA K</u>, UEMATSU M, FUKUYO N, MATSUMOTO Y, NAKAYAMA T, KOBAYASHI T, KAKISAKA Y, NAKASATO N (Japan)
- p104 Analysis of interictal epileptiform discharges in benign Rolandic epilepsy: predict to prognosis of seizures BYEON JH, KIM MK, PARK C, KIM MS, KIM GH, EUN S-H, EUN B-L (South Korea)
- p105 Retrospective review of the etiologies of acute symptomatic seizures in children with acute lymphoblastic leukemia (ALL) TAPAWAN SJC, ONG HT, TAY SKH, ALCASABAS AP, LOW PC, LIM JOO LI K (Singapore)
- p106 Outcome of antiepileptic drugs withdrawal in childhood onset nonidiopathic focal epilepsies <u>PAVLOVIC M<sup>1</sup></u>, JOVIC N<sup>2</sup>, PEKMEZOVIC T<sup>2</sup> (<sup>1</sup>Kuwait, <sup>2</sup>Serbia)
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- p108 Hypsarrhythmia severity; correlation with infants age, duration from onset of spasms and spasms load WANIGASINGHE J, ATTANAPOLA GM, ARAMBEPOLA C, LIYANAGE LSD, SUMANASENA S, SRIRANGANATHAN S (Sri Lanka)
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- p112 Seizures in children with demyelinating changes of the brain MENDIGALIYEVA N, LEPESSOVA M, NUKEBAYEVA Z (Kazakhstan)
- p113 Congenital vascular malformation in the genesis of focal epilepsy in children. Clinical case NUKEBAYEVA Z, LEPESSOVA M, MENDIGALIYEVA N (Kazakhstan)
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- p120 Seizure semiology in temporal lobe epilepsy NOFRIANSYAH A, CAHYANI D, BUDIKAYANTI A, OCTAVIANA F (Indonesia)

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- p123 How to improve quality of life people with epilepsy in I-San of Thailand <u>TIAMKAO S</u>, INTEGRATED EPILEPSY RESEARCH GROUP (Thailand)
- p124 Characteristics of patients with epilepsy who use a web-based health providing program of epilepsy, 'Epilia' KOO YS, SEOK HY, LEE SK, CHO YW, YANG K-S, JUNG K-Y (South Korea)
- p125 Epilepsy Action clinics within the acute hospital setting YOUNG M (Australia)

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- p127 Resective surgery in status epilepticus: report of three cases <u>MURTHY JMK</u>, MURTHY TVRK, AMEER BASHA P, KIRAN ESS, RAVI N (India)

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- Vagus Nerve Stimulation (VNS) implant surgery: operative technique assessment in a p132 single surgeon implant series ROOSEN N<sup>1</sup>, MCINTOSH KA<sup>1</sup>, RIZK T<sup>2</sup>, AWAAD Y<sup>2</sup> (<sup>1</sup>USA, <sup>2</sup>Saudi Arabia)
- p133 Surgical treatment for epilepsy induced by angiocentric glioma and literature review (analysis of 2 cases) LIU C, LUAN G (China)
- p134 Definitive surgical treatment of hypothalamic hamartoma based on 23 consecutive surgical experiences of hamartomas HORI T (Japan)
- p135 Long-term seizure and cognitive outcomes after surgical treatment of therapy-resistant temporal lobe epilepsy KHUSAINOV T, KWON HE, EOM S, KANG H-C, LEE JS, KIM HD (Uzbekistan)
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### PLATFORM SESSION ABSTRACTS

Clinical epileptology Friday 23<sup>rd</sup> March 15:00 - 16:00 Function Room 3

001

#### Characteristic of the new-onset epilepsy in the elderly

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**Purpose:** The elderly population is increasing. Epilepsy in the elderly will be an important issue around the world. To reveal the characteristics of the epilepsies in the senior citizens, we reviewed our experience at the tertiary referral center in Japan.

**Method:** We searched all the electric medical record at the epilepsy clinic in our University affiliated hospital between March 1<sup>st</sup>, 2005 and June 30<sup>th</sup>, 2011. Elderly person was defined as older than 65 years old. All the patients underwent history and physical, 3 Tesla MRI and/or CT, and electroencephalography. Diagnosis of epilepsy, age of onset, etiology and antiepileptic medication were recorded.

**Results:** We identified 80 patients who developed epilepsy after the age of 65 years. Epilepsy diagnosis were temporal lobe epilepsy (n=53,66.3%), frontal lobe epilepsy (N=11, 13.8%), other localization-related epilepsy (N=7, 8.8%), generalized epilepsy (n=5, 6.3%) and unclassified (n=3, 3.8%). Etiological diagnosis were post-stroke (n=13, 16.4%), inflammation (n=7, 8.8%), dementia (n=6, 7.5%), brain tumor (n=6, 7.5%), others (n=8, 10%), and non-lesional, MRI-negative (n=40, 50%). Interictal spikes were seen in 55 patients (68.8%). Fifty eight patients (72.5%) were on monotherapy, 12 patients (15.0%) were on polytherapy and 4 patients were not on medication. **Conclusion:** In our cohort of elderly persons with new onset epilepsy, temporal lobe epilepsy was most frequent followed by frontal lobe epilepsy. Non-lesional temporal lobe epilepsy is not uncommon. Epileptogenecity in the elderly patients is relatively low and responded well to anti-epileptic medication.

#### 002

#### Prognosis of first-ever remote symptomatic seizure

<u>SIM SH</u><sup>1,2</sup>, DUNNE J<sup>1</sup>, LEE J<sup>1</sup>, GAITATZIS A<sup>1</sup>, CHAN J<sup>1</sup>, LAWN N<sup>1</sup> <sup>1</sup>Royal Perth Hospital, Neurology Department, Perth, Australia, <sup>2</sup>Sarawak General Hospital, Neurology Department, Kuching, Malaysia

**Background and Purpose:** Anti-epileptic drug (AED) treatment is generally recommended in patients presenting with first-ever remote symptomatic seizure and with prior central nervous system (CNS) insult or epileptogenic lesion on neuroimaging due to perceived higher risk of seizure recurrence. It remains unclear whether the nature of the pre-existent lesion determines the risk of recurrence. The aims are to study the (1) risk of seizure recurrence in patients with a first-ever remote symptomatic seizure compared to those without a definable cause (idiopathic), (2) prognosis of first-ever remote symptomatic seizure due to various etiology, and (3) clinical factors that predict the recurrence following first-ever remote symptomatic seizure.

**Method:** Adults attending hospital-based clinics in the major hospitals in Western Australia from year 2000 to 2010, with first-ever seizure (except febrile convulsions) were prospectively studied. Seizure was defined as remote symptomatic in patients with history of prior CNS insults or potentially epileptogenic lesion on neuroimaging.

**Results:** 308 patients with first-ever unprovoked remote symptomatic seizure were identified (68% male, median age 47 years, range 14·91 years). Specific etiologies were stroke(n=94), head injury(n=67), tumour(n=54) and others(n=93). The 1-year risk of seizure recurrence was 58% for all unprovoked remote symptomatic seizure compared to 42% for idiopathic group(relative risk(RR) 1.4[95% CI 1.2·1.6]). The 1-year risk of seizure recurrence for remote symptomatic subgroups RR [95% CI] were stroke 64%, RR 1.5[1.3·1.8]; head injury 41%, RR 0.9[0.6·1.2]; tumour 69%, RR 1.7[1.4·2.0] and others 57%, RR 1.4[1.1·1.7]. Risk of recurrence was significantly lower with head

injury compared to other etiologies(p=0.003) including those with head injury and with clear post-traumatic changes on neuroimaging 23/15(45%),(p=0.03).

**Conclusion:** The prognosis of remote symptomatic first-ever seizure is not uniform. First-ever seizures related to prior significant head injury have lower risk of recurrence that is no different to first-ever seizure in the idiopathic group. Clinical factors predictive of recurrence following first-ever remote symptomatic seizure were stroke or head injury, Rankin score and simple partial seizure. Routinely treating these patients may be unnecessary.

#### 003

#### Classification of generalized tonic-clonic seizures: is it focal or generalized?

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**Purpose:** The purpose of the study was classifying the generalized tonic clonic seizure (GTCS) and evaluating the treatment outcome. Rapidly evolving secondarily GTCS are hardly distinguishable from primarily GTCS by semiology.

**Method:** We retrospectively analyzed clinical findings of patients who have only GTCS. Patients with at least two attacks of GTCS from our database. We excluded patients with 1) significant etiological factors except for febrile seizures and family history of epilepsy. 2) mental retardation or focal neurological deficits, 3) focal ictal features strongly suggesting focal onset, such as aura, conscious head version, and conscious or unconscious focal convulsive movements involving unilateral face or extremities, 4) GTCS only during sleep, 5) occurrence of focal features on ictal semiology during the follow-up.

**Results:** Total 67 patients were selected. The patients were divided into three groups according to EEG and MRI findings. Group A with generalized epileptiform discharges and normal MRI was 14 (20.9 %), and their mean age of onset was 18.5 years (range 11.5 to 29.5). Group B with normal EEG and MRI was 35 (52.2 %), and their mean age of onset was 25.7 (range 8 to 65). Group C with focal epileptiform discharges or abnormal MRI was 18 (26.9 %), and their mean age of onset was 33.4. All patients of group A were under age of 30 years of onset. The onset age of group A was significantly younger than group C (p=0.031). Treatment outcome was available in 49 patients. 44 patients were treated with one antiepileptic drug (AED). 45 of 49 patients (91.8%) achieved remission (group A: 10/12 (83.3%), group B: 22/24 (91.7 %), group C: 13/13 (100%)) with AED treatment.

**Conclusion:** This study suggests that patients with only GTCS had either focal or generalized origin, as expected. If strict criteria of generalized epilepsy are considered, epilepsy classification may be hard in the majority of patients with only GTCS. The age of onset may be an useful information for differentiating only GTCS.

#### 004

# Syndromic classification of epilepsy and seizure control among patients with epilepsy in a metropolitan tertiary neurology referral center in Penang, Malaysia

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**Purpose:** Epilepsy is a disease with varied etiologies, each with different prognosis and treatment response. We studied the syndromic classification of epilepsy and seizure control in a patient population in Penang, Malaysia.

**Method:** We reviewed data about demography, syndromic classification, anti-epileptic drug (AED) usage, and seizure control among patients with epilepsy that were followed up for > 1 year at the adult neurology clinic in Penang General Hospital, Penang, Malaysia.

**Results:** A total of 596 patients were studied. Among them, 124 patients (20.8%) have idiopathic epilepsy, 343 (57.6%) have symptomatic epilepsy, and 129 (21.6%) have cryptogenic epilepsy. Mean age of onset for idiopathic, symptomatic and cryptogenic epilepsies was 16.4 years, 20.4 years, and 21.3 years, respectively. Family history of epilepsy was elicited in 57 patients (9.6%). Among patients with idiopathic epilepsy, only 18.5% (23 patients) have family history, while 27.4% of idiopathic cases (34 patients) were adult onset (≥ 20 years old). Mean number of AED usage

was higher for symptomatic epilepsy (1.72 AED) than idiopathic (1.24) and cryptogenic epilepsies (1.39) (p< 0.05). Refractory epilepsy (> 2 AEDs correct for the seizure type) was commoner in symptomatic epilepsy (19.0%) than in idiopathic (4.8%) and cryptogenic epilepsies (7.0%) (p< 0.05).

**Conclusion:** Refractory epilepsy is more likely in symptomatic epilepsy and could be difficult to treat despite multiple AEDs. There were difficulties to distinguish between idiopathic and cryptogenic epilepsies, especially those of late adult onset. The availability of genetic testing for potential epilepsy genes may be useful in such cases, and we would be glad to participate in such international genetic collaborations.

#### 005

Role of steroids in reversing hypsarrhythmia in previously untreated infants with epileptic spasms WANIGASINGHE J<sup>1</sup>, ATTANAPOLA GM<sup>1</sup>, ARAMBEPOLA C<sup>1</sup>, LIYANAGE C<sup>2</sup>, SUMANASENA S<sup>1</sup>, SRI RANGANATHAN S<sup>1</sup>

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**Purpose:** Hypsarrhythmia in epileptic spasms is characterised by multi focal, high-amplitude spike wave discharges in a disorganised background with diffuse delta slowing. Though steroids are known to improve spasm outcome, role of steroids in reversing hypsarrhythmia has not been systematically studied before.

**Method:** Thirty previously untreated infantile spasms patients with hypsarrhythmia were prospectively followed up. Hypsarrhythmia severity was scored according to a 16 point scale described by Kramer et al by a paediatric neurologist blinded to the clinical information and the treatment given. All infants were treated with steroids (oral prednisolone or Intramuscular ACTH) for fourteen days according to the United Kingdom Infantile Spasms Study protocol. Repeat electroencephalogram was performed on 14<sup>th</sup>/15<sup>th</sup> day after commencement of therapy. Using a Paired T test the pre-and post treatment severity scores were compared. Improvement of each independent EEG abnormality {background disorganisation, diffuse delta activity, voltage of epileptic discharges, number of spikes/sharp waves and other items(4)} was also evaluated. **Results:** Twenty six children completed the therapy. Fourteen received ACTH while twelve received prednisolone. The mean pretreatment hypsarrhythmia severity score was 10.8+ 2.3. The post treatment severity score of 3.92+3.23 indicating a highly significant improvement (p=0.00). All components of the score i.e. Background disorganisation, diffuse delta slowing, voltage of epileptic discharges, number of spike and sharp waves, electro-decremental pattern, burst suppression in sleep, absence of normal sleep patterns and relative normalisation in wakefulness showed a highly significant (p=0.00) improvement following treatment. Clinically the post treatment hypsarrhythmia score was highest in those who showed no cessation of spasms while the score was lowest in those who completely responded. This difference was significant (F=1.58; df=2; p=0.22).

**Conclusion:** Steroids (ACTH and Prednisolone) resulted in a statistically significant improvement in all EEG characteristics of Hypsarrhtyhmia. The improvement of the hypsarrhythmia occurred even in those infants with no spasm control. These findings indicate that steroids play an important role in the reversal of "electrographic encephalopathy" seen in West syndrome.

#### Basic science Friday 23<sup>rd</sup> March 15:00 - 16:00 Function Room 2

#### 006

Effects of neuroprotective drugs against experimentally induced status epilepticus in young rats  $\underline{AHMAD}\ M^1,\ WADAAN\ MAM^2$ 

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**Purpose:** All currently available antiepileptic drugs do not provide satisfactory protection against seizures and associated neurodegenerative changes. Based on our pilot study of 10 drugs, the following short listed drugs including quinacrine (QCN), pentoxifylline (PTX) and proglumide (PGM)

have been tested for their neuroprotective activity against experimentally induced status epilepticus (SE) in young rats.

**Method:** SE was induced in 19 days old rats by lithium chloride (Li) (3mEq/kg, i.p.) followed (24 h later) by pilocarpine (Pc) in a dose of 20 mg/kg, subcutaneously. The animals received varying doses of QCN (5, 15 and 30 mg/kg), PTX (20, 40 and 60 mg/kg) and PGM (0.25, 0.5 and 0.75 g/ kg), i.p., one hour before induction of seizure. The control group received same volume of saline. Animals were subjected to a battery of behavioral tests to record seizures, tremors, anxiety, cognitive functions and locomotor activity. Thereafter, the animals were sacrificed and their brain was isolated for histopathological studies in the hippocampus area.

**Results:** Li-Pc produced significantly all symptoms of seizures leading to SE in animals along with significant motor deficit, anxiety and impairment of memory. QCN, PTX and PGM attenuated dose-dependently the latencies and incidence of seizures, motor deficits, loss of memory and cognitive dysfunction. These drugs also ameliorated the Li-Pc induced neuronal degeneration in the hippocampus.

**Conclusion:** The comparative significant and dose-dependent efficacy of the tested drugs were in the order PTX>PGM>QCN. Although all the three drugs were effective in protecting young animals against status epilepticus induced seizures and associated neurodegenerative changes, PTX was most effective causing no mortality.

#### 007

# Antipsychotic drug related seizures may result from inhibition of potassium currents in central neurons - a voltage-clamp and computational modelling study

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**Purpose:** Antipsychotic drug treatment is associated with seizures in up to 10% of patients. This is thought to be a result of seizure threshold reduction, but the actual mechanism is unknown. This study investigated the effects of three commonly prescribed antipsychotic drugs on "outward" or membrane stabilising currents that are critical in setting neuronal firing thresholds.

**Method:** Dissociated pyramidal neurons (n=30) were obtained from the hippocampal CA1 region of 2-3 week old Wistar rats. Whole-cell patch voltage clamp recordings measured the effects of 10 nM to 50  $\mu$ M haloperidol, clozapine and chlorpromazine on outward currents. Calcium and non-calcium sensitive, as well as inactivating ("IA") and non-inactivating ("IKDR") subtypes were studied. Neuron and Matlab programming environments were used to simulate the drug effects on single neurons as well as networks.

**Results:** The evoked currents were mainly carried by potassium ions, as confirmed by tail current and ion-substitution experiments. Current amplitudes with antipsychotic application and subsequent washouts were normalized against the largest current amplitude in the control solution. Current-voltage (I-V) curves of the control, drug and washout were plotted at each concentration, for both the peak and steady -state currents. We found that haloperidol and clozapine reduced steady state current amplitudes ranging from a  $\sim 10\%$  at 10 nM to 100% at 50 µM, with peak currents showing similar results. Blocade of calcium-sensitive currents with 0.5mM extracellular cadmium displayed essentially the same reductions in peak and steady state currents. IC50 for haloperidol was  $\sim$ 5uM with a qualitatively lower value for clozapine. Subtraction of traces before and after neurolept exposure at 10 nM concentration revealed a "delayed rectifier" non-inactivating outward current that may correspond to a HERG-class channel. Computational modelling of these effects produced reduced action potential threshold and network activation stimulus for single neuron and network simulations respectively.

**Conclusion:** We hypothesise that suppression of repolarising potassium currents by antipsychotic drugs is likely to reduce action potential threshold, leading to network hyperexcitability and an elevated risk of seizure generation.

#### 800

#### Pharmacoresistant seizure and its mechanism in immature brain

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**Purpose:** The incidence of seizures is high in childhood and pharmacoresistance is more intense in this age group. In this study we investigated the age specific effects of anticonvulsants using hippocampal-entorhinal cortex slices prepared from different age groups of rats. We also evaluated the role of NKCC1 co-transporter in pharmacoresistance.

**Method:** Frequently recurring SLEs presumably representing status epilepticus were induced by 4-aminopyridine in acute rat hippocampal-entorhinal cortex slices obtained from postnatal day 3-19 (P3-P19), and the effects of carbamazepine, phenytoin, valproic acid and phenobarbital were examined. In addition, bumetanide was tested, which blocks the Na+-K+-2Cl- (NKCC1) co-transporter, and also acetazolamide, which blocks the carbonic anhydrase and thereby the accumulation of bicarbonate inside neurons.

**Results:** The efficacy of all antiepileptic drugs in blocking SLEs was dependent on postnatal age, with low efficacy in P3-P5 slices and high efficacy in older age groups. Antiepileptic drugs suppressed SLEs more readily in the medial entorhinal cortex (ECm) than in the CA3. In P3-P5 slices, valproic acid and phenobarbital increased both tonic and clonic seizure-like activities in the CA3, whereas phenytoin and carbamazepine blocked tonic-like but prolonged clonic-like activity. Similar to the effects of other antiepileptic drugs, the seizure-suppressing effects of acetazolamide increased with postnatal age. In contrast to standard antiepileptic drugs bumetanide showed high efficacy in younger age group and low efficacy in older age group. In P3-P5 slices, bumetanide often blocked SLEs in the CA3, but was not as effective in the ECm. In slices prepared from the older age groups bumetanide failed to block SLEs in both CA3 and ECm.

**Conclusion:** We conclude that pharmacoresistance may be inherent to very immature tissue and suggest that expression of the NKCC1 co-transporter might contribute to pharmacoresistance.

#### 009

# Monocyte chemoattractant protein-1 affects ectopic migration of neural progenitors within the hippocampal dentate gyrus of rats with status epilepticus

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**Purpose:** Previous studies suggest a role of neuroinflammation in pathogenesis of mesial temporal lobe epilepsy. In the present study, we further investigated the involvement of monocyte chemoattractant protein-1 (MCP-1) in seizure-induced aberrant migration of neuronal progenitors in the hippocampus.

**Method:** Status epilepticus (SE) was induced by pilocarpine in Sprague-Dawley rats. Transcriptional expression of MCP-1 in dentate gyri (DG) was measured by quantitative real-time PCR. From 1 to 28 days after SE, the temporal profiles of MCP-1 protein expression in DG was evaluated by enzyme-linked immunosorbent assay. Chemokine (C-C motify receptor 2 (CCR2) in doublecortin-positive neuronal progenitors was examined by double-labeling immunohistochemistry. The involvement of MCP-1/CCR2 signaling in the aberrant neuronal progenitor migration in epileptic hippocampus was assessed in the SE rats treated with a CCR2 antagonist, RS102895, and then the formation of hilar ectopic granule cells was determined by Prox-1 immunostaining.

**Results:** There was a significant increase in gene expression of MCP-1 in response to seizure insults. MCP-1 protein expression in DG was significantly increased on day 1 and day 3 post-SE. Notably, some hilar ectopic progenitor cells expressing MCP-1 receptor were found in SE rats. A blockade of MCP-1/CCR2 interaction with a selective CCR2 inhibitor, RS102895, attenuated the formation of seizure-induced hilar ectopic granule cells.

**Conclusion:** These findings suggest that an increase in dentate MCP-1 is associated with seizureinduced aberrant migration of neuronal progenitors through interacting with CCR2 and may contribute to subsequent epileptogenesis.

#### 010

# In vitro transport profiles of lacosamide, rufinamide, pregabalin, and zonisamide by human P-glycoprotein

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**Purpose:** Epilepsy is resistant to treatment in about 30-40% of patients. Efflux of antiepileptic drugs from the brain by P-glycoprotein (Pgp) is one of the potential mechanisms of drug resistance. To determine whether antiepileptic drugs may be substrates for Pgp transport, we examined transport of four drugs that had not previously been tested for Pgp substrate status. **Method:** Madin-Darby Canine Kidney (MDCK) or porcine kidney endothelial LLC-PK1 cell lines stably expressing human Pgp were grown as monolayers in Transwells. We used the concentration equilibrium transport assay, which is a sensitive method for detecting directional efflux of drugs that exhibit high diffusion across cell membranes. An equal concentration of drug was initially loaded in both the apical and basal chambers, and the concentration in both chambers was measured up to 4 hours. Zonisamide, pregabalin, rufinamide, and lacosamide were studied, using concentrations within the range found in plasma during drug treatment: 5 µg/ml for all except zonisamide, for which we used 10 µg/ml. The experiments were transported.

**Results:** Lacosamide displayed basal-to-apical transport in Pgp-expressing MDCK and LLC cells. The transport was blocked in the presence of tariquidar. Zonisamide, pregabalin, and rufinamide did not exhibit directional transport.

**Conclusion:** Lacosamide is a Pgp substrate, while zonisamide, pregabalin, and rufinamide are not. Animal experiments may help confirm these results. Comparing resistance among patients treated with Pgp substrates vs. non-substrates may reveal the contribution of Pgp efflux to resistance.

Clinical neurophysiology and neuroimaging Friday 23<sup>rd</sup> March 15:00 - 16:00 Function Room 1

011

Changes of cortical silent period in patients with drug-naïve epilepsy

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**Purpose:** We used transcranial magnetic stimulation (TMS) to investigate the difference of cortical excitability between patients with IGE or FE and controls.

**Method:** We consecutively recruited 219 drug-naïve patients with epilepsy (IGE 100, PE 119, mean age 28.5 yrs) and 65 age- and sex- matched normal subjects with no drug history for CNS. Based on the seizure semiology, EEG, and brain MRI, epileptic focus was lateralized into right (N=45) or left hemisphere (N=74) in FE patients. We measured TMS parameters including resting motor threshold (RMT), motor evoked potential (MEP) amplitudes, cortical silent period (CSP), intracortical inhibition (ICI, interstimulus interval 2, 3, 5ms) and facilitation (ICF, interstimulus interval 10, 15, 20 ms). The TMS parameters were measured during seizure free state more than 24 hours in patients.

**Results:** In epilepsy patients and controls, the CSP was lengthened approximately linearly with increases of stimulus intensity in both hemispheres. The mean CSP was longer in IGE patients compared with controls at all stimulus intensities (P < 0.05). In FE patients, TMS parameters were compared between 1) ipsilateral hemisphere to epileptic focus (IH) vs. contralateral one (CH), or 2) IH or CH vs. normal controls, and 3) IH or CH vs. IGE patients. The mean CSP was significantly shorter in IH than that in CH at 120% (129.5±34.0 in CH vs. 114.3±24.2 ms in IH, p=0.028) and 140% of RMT (170.4±34.0 in CH vs. 149.4±28.4 ms in IH, p=0.003). Mean CSP duration in CH was significantly longer at the stimulus intensities 120 - 150% of RMT and that in IH was longer only at 120% of RMT than that of controls (114.3±24.2 ms in FE vs. 88.1±31.3 in controls, p=0.005).

**Conclusion:** These findings suggest that the CSP may have a lateralizing value in FE by shorter CSP in the epileptic hemisphere. Prolonged CSP in epilepsy may indicate the abnormally increased interictal cortical inhibition in human epileptic brains.

#### 012

# Chronotopology of neurophysiologic indicators for early and late epileptogenesis and drug-resistant focal and multifocal epilepsy surgery

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**Purpose:** To investigate neurophysiologic mechanisms of epileptogenesis in resistant temporal epilepsy and to optimize adequate surgical treatment.

**Method:** Electroclinical examination and surgical treatment results in over 200 drug-resistant temporal epilepsy patients aged 19-45 were analyzed. Algorithm including neurophysiologic (monitoring EEG, ECoG, SEEG with topographic brain map via deep and subdural electrodes) and visualizing (functional MRI) technologies to study dynamics of epileptogenesis in the process of epileptic syndrome forming has been elaborated.

**Results:** Based on chronotopology of EEG-SEEG trait-markers particularities of preclinical (initial), early (monotemporal) and late (extratemporal) epileptogenesis were depicted reflecting clinical-neurophysiologic forms of focal and multifocal temporal epilepsy. Early (monotemporal) epileptogenesis is characterized by peculiar localization and extent of the epileptic focus (zone): ECoG-SEEG studies revealed a combined neocortical and limbic (hyppocampus, amygdala) damage in 79% of patients. This optimizes technology of open surgical treatment (anterior temporal lobectomy) with additional use of multiple subpial transection in eloquent (frontal and temporal) cortex. Morphofunctional basis of extratemporal links of epileptogenesis is amounted by integral brain systems (ensuring cerebral homeostasis in normal conditions): according to EEG-SEEG data limbical-brain-stem pathways of epileptizaton were followed-up - hippocampus and amygdala via thalamic anterior nuclei are involved in cyclic Papez and Livingston-Escobar-Yakovlev systems, forming multifocal forms, which is of importance in neuromodulating interventions.

**Conclusion:** Our results show that neurophysiologic indicators appear a reliable method to evaluate regularities and chronotopology of epileptogenesis at the cortical, limbic and brainstem structure levels. The dynamics of epileptogenesis optimizes strategy of differentiated surgical treatment of drug-resistant focal and multifocal temporal epilepsy.

#### 013

#### Testing times: Test-Enhanced Learning as an instructional method for EEG courses

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**Purpose:** EEG courses often use lectures to teach EEG interpretation, but retention after lectures is poor, and there are few data regarding effective EEG instructional methods. In medical education, tests are usually used for assessment. However, cognitive psychology studies demonstrate that repeated testing can enhance retention of information; this is termed Test-Enhanced Learning (TEL). We aimed to determine if TEL would be useful in learning EEG interpretation in an EEG course.

**Methods:** We conducted a half-day course on EEG and video EEG interpretation. Participants were given material for pre-reading two weeks before the course; this included (a) key features and sample EEG recordings of normal and abnormal EEG patterns (b) pointers on seizure semiology. No formal teaching was done during the course. Participants instead underwent TEL, doing 5 sets of 10 multiple choice questions (MCQ). During each 20-minute set, an EEG page or video of a seizure was shown on screen with 4-5 answer choices; participants were required to select the correct answer using an audience response system clicker within 1 minute. The instructor then provided the correct answer and a very brief explanation why. Participants were given a 3-minute break between sets. We measured effectiveness of TEL using a 16-item MCQ pre-post test done before and after TEL.

**Results:** We included 106 participants; 58.5% were neurologists, 21.7% were EEG technicians. The 16-item pre-post test showed acceptable reliability (Cronbach's alpha=0.72) and good discriminating ability; all discriminant indices were >0.35. Mean pre- and post-test score were 57.2% and 69.8%; mean improvement in score was 12.6% (95% CI 9.6  $\cdot$  15.5%, p< 0.001). The improvement in scores was not significantly different in neurologists or EEG technicians (p=0.83). The effect size of the TEL intervention was moderate (Cohen's D = 0.62).

**Conclusion:** TEL is an effective EEG instructional method for EEG courses with a moderate effect on learning. TEL can be employed for EEG courses, instead of relying on transmissive methods such as lectures.

#### 014

# A rational surgery for hypothalamic hamartoma: ictogenesis and symptomatogenesis of gelastic seizures

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**Purpose:** To elucidate the rationale of stereotactic radiofrequency thermocoagulation (SRT) for treating hypothalamic hamartoma (HH), we topographically localize the ictogenic zone and the symptomatogenic zone for gelastic seizure (GS) using single photon emission computed tomography (SPECT).

**Method:** Fifty-three consecutive patients (aged 2 to 50, mean: 15 years) with HH underwent SRT in our hospital between 1997 and 2011. Mean maximum diameter of the HH was 18 mm (range, 5-80 mm). Ictal SPECT data were statistically analyzed by means of subtraction ictal SPECT coregistered to MRI (SISCOM) and statistical parametric mapping (SPM). Topographical localization of ictal hyperperfusion areas was evaluated. SRT (74 °C, 60 s) was used to make multiple 5-mm spherical lesions within the HH.

**Results:** SISCOM demonstrated ictal hyperperfusion in the HH interface zone. 70 SRT surgeries made 1-36 lesions (mean, 8) in the interface zone, with 1-9 tracks (mean, 4). SRT was successfully performed for 3 giant HHs (over 5 cm in diameter). No permanent complications persisted after SRT. GS disappeared in 50 patients (96%). Complete freedom from seizures was achieved in 44 patients (83%). SPM group analysis revealed significantly (p< 0.001) hyperperfused areas in the ipsilateral hypothalamus, mediodorsal (MD) nucleus of the thalamus, and putamen, bilateral pontine tegmentum, and contralateral inferior semi-lunar lobule of the cerebellum.

**Conclusion:** The present study confirmed that SRT is minimally invasive and appears effective and rational surgery without adaptive limitations regardless of the size or subtype. Hypothalamus and MD nucleus of the thalamus play an important role for symptomatogensis of GS.

#### 015

# White matter impairment in the basal ganglia-thalamocortical circuit of drug-naïve childhood absence epilepsy

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**Purpose:** It is unknown whether white matter abnormalities exist in childhood absence epilepsy (CAE), a syndrome of idiopathic epilepsy (IGE). Diffusion tensor imaging (DTI) can noninvasively quantify white matter integrity. This study used DTI to investigate abnormal changes in white matter of untreated CAE patients.

**Method:** Subjects included nine patients with untreated CAE and nine age-and sex-matched healthy controls. Diffusion tensor imaging parameters were voxel based and statistically compared between patients and controls. The correlations between DTI parameters in regions of interest (ROIs) and age of seizure onset or duration of epilepsy were analyzed.

**Results:** Untreated CAE patients had a significantly higher fractional anisotropy (FA) value in the bilateral thalamus, anterior corpus callosum and upper brainstem, while also displaying a lower FA value in prefrontal white matter, anterior cingulate, and bilateral posterior limbs of the internal

capsule compared to control subjects. An increase in mean diffusivity (MD) value was observed in parietal lobe white matter, prefrontal white matter, and posterior cerebellar hemispheres, in addition to subcortical structures including bilateral putamen and posterior limb of internal capsule. MD significant correlations between ROI diffusion parameters and the duration of the disease or the age of onset.

**Conclusion:** The results showed white matter integrity impairment in the basal gangliathalamocortical circuit of drug-naïve CAE patients. These abnormalities in white matter may be related to increased cortical excitability and cause cognitive, linguistic, and behavioral/emotional deficits both during and between seizures.

Epidemiology and genetics Saturday 24<sup>th</sup> March 15:00 - 16:00 Function Room 3

#### 016

#### Sleep disordered breathing in patients with epilepsy

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**Purpose:** The present study was designed to assess the prevalence of sleep disordered breathing (SDB) in patients with epilepsy.

**Method:** In the first stage of this cross-sectional study, 426 consecutive patients with epilepsy (cases) and 500 normal age- and gender-matched healthy controls were screened for excessive daytime-sleepiness (EDS) using Epworth Sleepiness Score (ESS). In comparison with controls, patients with epilepsy had an almost 6-times higher prevalence of EDS (ESS >10) (3.6% Vs 23%; p=0.0000). Of the 98 (23%) cases with EDS, 30 were taken up for second stage of study and underwent overnight in-hospital polysomnography (PSG).

**Results:** The mean age of the patients who underwent PSG (n=30) was 35.8±13.1 years; there were 26 males. Median symptom epilepsy duration was 72 [interquartile range (IQR) 72·180] months; and majority had idiopathic epilepsy (n=22; 73%). On PSG (n=30), an apnea-hypopnea index (AHI) ≥5 was observed in 13 (43.3%) patients, suggestive of obstructive sleep apnea syndrome (OSAS). A higher proportion of patients with nocturnal seizures had OSAS compared with those with diurnal and combined periodicity (7/8 Vs 6/16; p=0.009). Patients with uncontrolled seizures had a higher prevalence of OSAS (7/10 Vs 6/20; p=0.045). A statistically-significant positive correlation was observed between ESS and body-mass index (BMI) (r=0.681; p=0.000); mid-arm circumference (MAC) (r=0.575; p=0.001); waist circumference (WC) (r=0.576; p=001); and waist-hip ratio (WHR) (r=0.398; p=0.029).

**Conclusion:** Our observations suggest that EDS is an important symptom in patients with epilepsy. OSAS (present in 43% cases) appears to be an underdiagnosed disease in patients with epilepsy from India.

#### 017

# A novel digital animation seizure survey may be an effective screening instrument for seizures in population-based research

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**Purpose:** The conventional research method to screen for epilepsy is with a written questionnaire administered door-to-door (WQ). Although this enables comprehensive capture of undiagnosed and untreated cases, it is resource intense, insensitive to non-convulsive seizures and inefficient for large-scale case ascertainment. Our aim was to 'field test' a novel digital animation seizure-screening questionnaire that may be more suitable for community-based recruitment.

**Method:** We developed a series of five high resolution digital animations depicting visual sequences of young people with seizures (AQ) consisting of: tonic clonic, simple partial motor, complex partial
temporal lobe, absence and myoclonic. We administered AQ to parents of primary and secondary school students and secondary school students by a specially constructed website www.sparks. org.au. All students underwent epilepsy specialist assessment (ESA) including EEG, to confirm the diagnosis of epilepsy. AQ was repeated after first completion, to estimate AQ repeatability.

**Results:** 161 AQ internet surveys were conducted with all undergoing ESA: 7 screened positive with 3 confirmed cases of epilepsy by ESA. Both parental and student's surveys combined: sensitivity 1.00 (1.00-1.00), specificity 0.96 (0.93-0.99), Youden's Index - a summary measure of sensitivity and specificity - (0.93-0.99). Surveying only parents improved specificity slightly without affecting sensitivity. Repeat survey demonstrated 100% concordance.

**Conclusion:** Although numbers are relatively small, early results suggest that AQ may be a more effective population-screening instrument than WQ. In addition, it should have improved efficiency as it can readily access individuals and households through the internet rather than door-to-door.

### 018

# Vitamin D deficiency in a hospital based population of children with epilepsy on long-term antiepileptic drugs

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**Purpose:** Long-term AED use is an important risk factor for impaired bone health in children, with a subsequent increased risk of premature osteoporotic fractures. Latest evidence highlights the importance of periodic bone health monitoring in these children. Vitamin D replacement is indicated with vitamin D deficiency (< 50nmol/L) and supplementation should be considered with vitamin D insufficiency (50-75nmol/L). The aim of this study was to to evaluate Vitamin D levels in a hospital-based population of children on long-term antiepileptic drugs (AEDs).

**Method:** A cross-sectional cohort study of a hospital-based population of children on long-term AEDs was undertaken through contacting parent/carers of all children with epilepsy, requesting bone health blood tests during the winter of 2011. Children already on vitamin D supplements or with existing vitamin D monitoring were excluded. Bone health risk factors were assessed through review of the medical record.

**Results:** 130 letters were sent to parents/carers with 111/130 (85%) children subsequently having blood tests performed. Frank vitamin D deficiency was identified in 24(22%) children and a further 45(41%) had Vitamin D insufficiency. Multiple logistic regression analysis identified children on multiple >2 AEDs or with underlying genetic aetiologies were more likely to have Vitamin D deficiency.

**Conclusion:** This first study of an Australian hospital-based population of children with epilepsy has demonstrated that a high proportion of children on long-term AED(s) risk Vitamin D insufficiency/ deficiency despite such children living in the subtropics (latitude 27°S) with greater exposure to sunlight all year round. Bone health monitoring and vitamin D supplementation is essential in the management of all children on long-term AEDS.

# 019

# Antiepileptic drug and risk of stroke in patients with epilepsy: a population-based cohort study $\underline{HSIEH \ C\cdot Y^{1,2}}$ , LAI EC-C<sup>1</sup>, KAO YANG Y-H<sup>1</sup>

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**Purpose:** We aimed to evaluate the risk of stroke in adult patients with epilepsy using older antiepileptic drugs (AEDs), i.e., phenytoin (PHT), valproic acid (VPA), and carbamazepine (CBZ). **Method:** We conducted a retrospective cohort study using a 2-million population randomly sampled from Taiwan National Health Insurance Research Database. Epileptic patients without prior stroke history and new to monotherapy with PHT, CBZ, or VPA were included and followed for 5 years. Our event of interest was defined as hospitalization or emergency room visit due to stroke. Coxproportional hazard models were used to estimate the stroke risk of each AED. Moreover, sub-analyses and several sensitivity analyses, including propensity score technique and intention-to-treat (ITT) approach, were performed to test the robustness of this study.

**Results:** Patients receiving PHT had significantly higher stroke risk (adjusted hazard ratio, AHR, 1.718; 95% CIs, 1.197-2.466), followed by VPA (AHR, 1.269; 0.778-2.069), compared with CBZ. All the results of sub-analyses and sensitivity analyses showed concordance trend for higher risk toward PHT, even in the PS matched approach and ITT analysis. In addition, the risk tended to increase in those who used longer duration and higher dose of PHT.

**Conclusion:** It's important for health care providers to consider the risk of stroke in patients under older AEDs treatment, especially PHT. Further studies are needed to compare vascular risks between older and newer generation of AEDs.

# 020

### Pharmacogenetic evaluation of fetal malformations in women with epilepsy receiving antiepileptic drugs

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**Purpose:** Pharmacogenetic variability in drug transporters (MDR1) and metabolizing enzymes (Cyp2C9, Cyp2C19 and MTHFR) and induction or inhibition of these enzymes by antiepileptic drug (AED) therapy can be determinants for teratogenic potential.

**Method:** The MDR1, Cyp2C9, Cyp2C19 and MTHFR polymorphisms were genotyped in 264 Women with Epilepsy (WWE) comprising of 141 with malformations and 123 with normal offsprings. The allelic, genotypic and haplotypic distributions were tested for association between malformation group and epileptic controls and further between the type of malformation, seizure frequency and therapy within the malformation group itself.

**Results:** A strong allelic, genotypic and haplotypic association of MDR1 polymorphism Ex07 +139C/T is reported in the mothers with malformations in their infants. We observed that the patients with infants having malformations were more likely to have the C allele and CC genotype of Ex07 +139C/T when compared to women with normal offspring. There was a significant association with Cyp2C19 (p=0.005) with malformation at both allelic and genotypic combination for poor metaboliser phenotype.

**Conclusion:** Our study indicates that MDR1 and Cyp2C19 can play a pivotal role in the AED induced teratogenesis.

Neuropsychology and social issues Saturday 24<sup>th</sup> March 15:00 - 16:00 Function Room 2

### 021

The effects of support group on self-esteem in people with epilepsy SAWANCHAREON  $K^1$ , PRANBOON S<sup>2</sup>

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**Purpose:** The purpose of this study was to examine the effects of a support group on self-esteem in PWE.

**Method:** A Quasi-experimental study was performed of 120 PWE in an Epilepsy Clinic at Srinagarind Hospital. An experimental group (N=60) attended in the support group before receiving regular healthcare services. The control group (N=60) received only regular healthcare services. Data was collected by using the Rosenberg self -esteem scale scoring before and after the experiment. The score was analyzed by using a paired t-test and an independent t-test.

**Results:** The study showed that before the experiment, the self -esteem score of the control group was significantly higher than the experimental group(= 35.06, 32.80, t = 2.38 P < .05). After the experiment, the score of the control group and the experimental group were showed a significant statistical difference (= 3.06, 34.30, t = -6.61 P < .05). The score in the control group was significantly lower than the experimental group (= 35.01, 30.60, t = 9.97 P < .05), while the score

in the experimental group was significantly higher than before the experiment ( = 32.80, 34.30, t = 2.19 P < .05)

**Conclusion:** The support group improves in self- esteem in PWE. Medical personnel should set up a support group for PWE to enhance their self-esteem.

### 022

# Prediction of memory change in mesial temporal lobe epilepsy after anterior temporal lobectomy with amygdalohippocampectomy

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**Purpose:** Purpose of our study is to investigate Wada test as a predictor of memory outcome after anterior temporal lobectomy in patients with mesial temporal lobe epilepsy (TLE).

**Method:** Patients were included in the study if they met the following criteria: (1) unilateral TLE based on a long-term video EEG monitoring, (2) unilateral hippocampal sclerosis or atrophy without other structural abnormality on brain MRI, (3) left hemisphere dominance for language on IAT, (4) seizure free-state after the surgery. Total of 74 patients (38 males, mean age 29.7 years) met the inclusion criteria for the study. Patients had undergone anterior temporal lobectomy with amygdalohippocampectomy (ATL with AH) for removal of the epileptic focus. All patients underwent presurgical and postsurgical neuropsychological test (NP test). We divided the patients for two groups by epileptic focus (resection side, Right or Left). Each postoperative memory changes (PMC) in the two groups were investigated. We calculated Wada asymmetry index (WAI) and NP asymmetry index (NPAI).

**Results:** Right ATL group showed significant postoperative improvement in verbal memory [California verbal learning task (CVLT) 1-5 total raw score (TRS), p < 0.001; CVLTSD, P < 0.05; CVLTLD, p < 0.001; CVLTRH, p < 0.01; logical memory test immediate recall (LMTIR), p < 0.01; LM delayed recall, p < 0.01]. Left ATL group revealed significant decrease in verbal memory score [CVLT 1-5TRS (p < 0.05); CVLTSD (P < 0.05) and CVLTLD (p < 0.01)]. In whole patients, NPAIs of verbal memory were correlated with WAI. CVLT15TRS, CVLTSD, LMTIR and LMTDR of NPAIs were weakly correlated with WAI. CVLT1D of NPAIs is moderately correlated with WAI. The more WAI is increased, the greater improvement of NPAIs is expected.

**Conclusion:** Our data shows that the information acquired by the Wada test, specifically language dominance and memory ability of the hemisphere, is highly important for preoperative patient counseling before elective epilepsy surgery. WAI could predict memory change in mesial TLE after ATL with AH.

### 023

# Longitudinal surveys into the social impact of epilepsy: an Australian 'first'

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**Purpose:** To report previously unpublished results from Wave 2 of longitudinal research conducted with 343 people with epilepsy in a community setting. These results relate to education, income and employment. These are followed by results on seizure activity, injury and hospitalisation. **Method:** In 2006 the Epilepsy Foundation Victoria established a Research Participant Register (RPR) to conduct applied social and behavioural research. Now the Australian Epilepsy Research Register with research participants from all Australian states, this register provides the basis for a longitudinal study to demonstrate the psycho-social impact of epilepsy across the whole of people's lives in Australia. Two surveys or 'waves' have now been conducted in 2007 and 2010. Extensive data have been collected on income, housing, marital status, costs of care and quality of life. **Results:** Using SPSS 17, researchers have cross-tabulated the relationship between age, employment, incomes and costs of epilepsy care. This has been followed by an analysis of seizure activity, rates of injury and hospitalisations.

Analysis shows this group is relatively well-educated but generally unemployed. Many live under the Australian Poverty Line. This group also has poor seizure control and high rates of injury and hospitalisations.

**Conclusion:** Surveys will be undertaken every two years exploring people's socio-economic status related to the impact of having epilepsy. While giving a rare picture of how people live with epilepsy in the community and how epilepsy impacts on quality of life over time, the results will also provide evidence to argue for better government policies and assistance to people with epilepsy.

# 024

# Beyond the biomedical: the experience of epilepsy among women in Kerala, India $\underline{\sf NAIR}\, A^1,\, {\sf THOMAS}\,\,{\sf SV}^2$

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**Purpose:** To study the stigma of epilepsy and its impact on various domains of life for women with epilepsy in Kerala, India.

**Method:** We interviewed fifty women with epilepsy (18 - 45 years) attending the Kerala Registry of Epilepsy and Pregnancy. Respondents were requested to recall their first brush with epilepsy and recount if and how epilepsy had impacted their lives; these narratives were analysed using sociological theories of stigma and insights from the newer field of disability studies.

**Results:** Vivid descriptions of the experience of illness emerged from the interviews that went beyond biomedical perspectives. The diagnosis of epilepsy had impacted their dynamics of identity formation, perceptions of self, family interactions, education, employment and marital life. The course of education changed after the diagnosis of epilepsy for 24% of the respondents. Stigma—both actual and perceived—was a very real presence in the lives of all participants. The ability to cope with stigma was conditional upon the 'resources' available to the individual, with the associated stigma in families proving critical. An almost-ubiquitous pattern of concealment of epilepsy was practiced by our respondents, irrespective of the type of epilepsy, and the consequences of concealment were evident in conjugal relationships, familial conflicts and social interactions.

**Conclusion:** This study demonstrated that stigma linked with epilepsy is a serious force to contend with and has the potential to influence multiple domains of life and therefore must be taken into consideration by practitioners.

### 025

### Patterns of psychological adjustment following paediatric epilepsy surgery

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**Purpose:** We explored the process of psychological adjustment following paediatric epilepsy surgery given the limited knowledge base in this area.

**Method:** Semi-structured interviews (modified Austin Comprehensive Epilepsy Program Interview) canvassing a broad range of pre/post-surgical issues were administered to 46 children who underwent resective epilepsy surgery (temporal and extratemporal) between ages 6-18 years. Interviewed were 33 patients (mean age, 19.4 years, 53% female) and 44 parents (80% mothers) with a mean time to interview of 6 years.

**Results:** Analysis of patient and parent perceptions revealed four distinct groups: (1) patients with few seizures prior to surgery who were rendered seizure free and reported minimal adjustment issues after surgery (11%); (2) chronic patients who were rendered predominantly seizure free and reported adjustment difficulties associated with learning to become "well", known as the 'burden of normality' (39%); (3) patients who experienced little change in seizure status and retained a sense of being "sick" (20%); and (4) patients who, despite being rendered seizure free, failed to achieve a sense of normality and reported psychiatric and psychosocial difficulties after surgery (30%). Comparison of patient and parent perceptions in 31 dyads revealed 80% concordance across the groups.

**Conclusion:** There are distinct patterns of psychological change following paediatric epilepsy surgery. Similar to adults, paediatric patients report changes in their sense of self, including some who describe the burden of normality following efficacious epilepsy surgery. These findings have significant implications for the provision of preoperative counselling and postoperative follow-up and rehabilitation of paediatric patients and families.

#### Surgery Saturday 24<sup>th</sup> March 15:00 - 16:00 Function Room 1

# 026

# MRI features and surgical outcome of focal cortical dysplasia according to the new ILAE classification criteria

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**Purpose:** Focal cortical dysplasia (FCD) is the most common pathological diagnosis in patients who underwent surgical treatment for intractable neocortical epilepsy. However, presurgical identification of MRI abnormalities in FCD patients remains difficult, and there are no highly sensitive imaging parameters available that can reliably differentiate among FCD subtypes.

**Method:** We retrospectively recruited epilepsy patients who underwent surgical treatment for refractory epilepsy with focal MRI abnormalities and the pathological diagnosis of FCD. We evaluated the surgical outcome according to the pathological subtypes, and studied the prognostic roles of various MRI features. We used recently proposed three-tiered FCD classification system which defined type III when FCD occurs in association with other epileptogenic pathologies. **Results:** A total of 69 patients were included, and 68.1% of patients became seizure free. Patients with FCD type III had a lower chance for seizure free (7/15) than in patients with FCD type I or II (40/54, p=0.044). Cortical thickness, blurring of gray-white matter, and transmantle sign was more common in FCD types I or II than in FCD type III, but most MRI features failed to differentiate between FCD types I and II and only transmantle sign was specific for FCD type II.

**Conclusion:** Our study shows that patients with FCD III have poorer surgical outcome. Typical MRI features of FCD types I and II such as cortical thickness, blurring of gray-white matter, and transmantle sign were not common in FCD type III and only transmantle sign is helpful in differentiating between FCD types I and II. Recruitment of more patients and further MRI analysis is needed to further characterize the MRI features in patients with FCD type III.

### 027

# MRI-guided stereotactic radiofrequency thermocoagulation for 53 intractable epilepsy patients with hypothalamic hamartoma: analysis of patients with residual seizures

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Purpose: To investigate the epileptogenesis of hypothalamic hamartoma (HH).

**Method:** Fifty-three HH patients underwent stereotactic radiofrequency thermocoagulation (SRT) and were followed for  $\geq 6$  months; all but one displayed gelastic seizures (GSs), with non-gelastic seizures (NGSs) in 47 (89%), behavioral disorder (BD) in 22 (41%), and mental retardation (MR) in 28 (53%). Mean maximum diameter of HH was 18 mm (range, 5-80 mm). Patients were classified into three types by magnetic resonance imaging (MRI): intra-hypothalamic type (I-HH), 16 patients (30%); para-type, 11 (21%); and mixed-type (M-HH), 26 (49%). Patients were also classified into two groups by severity: severe group, with  $\geq 2$  of tonic seizures, BD or MR, 27 (51%); and mild group,  $\leq 1$  of these disorders, 26 (49%).

**Results:** Age of seizure onset was younger in GS (mean, 2 years; range, 0.10 years) than in NGS (mean, 6 years; range, 0.17 years). Both BD and MR were improved in all patients. GS disappeared in 50 patients (96%), and both GS and NGS disappeared in 43 (81%), whereas GS or NGS remained in 10 (19%). Seizure-free rate was higher in the mild group (92%) than in the severe group (70%; p< 0.05). I-HH rate was higher in the mild group (50%) than in the severe group (11%; p< 0.002). M-HH rate was higher in the severe group (63%) than in the mild group (35%; p< 0.002).

**Conclusion:** SRT can almost completely resolve GS. Another focus of epileptogenesis besides HH could be present in cases of M·HH with tonic seizures. NGS may represent intractable secondary epileptogenesis.

### 028

# Resection frequency map after awake resective surgery for non-lesional neocortical epilepsy involving eloquent area

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**Purpose:** The resection of eloquent area is challenging due to postoperative neurological deficits. The purpose of this study was to assess the efficacy and risk of awake brain surgery for non-lesional epilepsy involving the eloquent areas or their adjacent areas and to advocate the generation of a resection frequency map.

**Method:** We enrolled 55 patients who underwent awake surgery between 1994 and 2007 for nonlesional epilepsy involving the primary sensori-motor or language areas. All patients underwent two-staged operations including subdural electrode monitoring and awake resective surgery. For each case, the preoperative and postoperative images were spatially normalized and compared on a standard atlas, and the resection map was then computed by summing up each resected area on the atlas.

**Results:** The postoperative seizure outcome was Engel class I in 27 patients (49.1%), II in 9 (16.4%), III in 14 (25.5%) and IV in 5 (9.1%). Ten patients (18.2%) experienced postoperative neurological deficits including 7 transient (12.7%) and 3 permanent, but mild ones (5.5%). The neurological complication rate of purely eloquent area resection was 36.8% (7/19). The resection frequency map computed in this study showed that the resection of eloquent areas was tolerable, with the exception of the Broca's area.

**Conclusion:** Awake resective surgery with intraoperative brain mapping is an effective and safe treatment option for non-lesional epilepsy involving eloquent areas. The resection frequency map can show the resected area of a group as well as individuals obviously and provide an objective measure of neurological risk.

# 029

Unresolved enigmata about hypothalamic hamartomas: an institutional inquest

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**Purpose:** Though a rare entity, a good volume of scientific evidence (predominantly levels IV,III and II) exists in published literature about hypothalamic hamartomas (HH). However, several unsolved enigmata still remain and we chose to address them through a retrospective review of our institutional experience. Some of these issues include correlation of size/location/radiological subtype with seizure semiology/symptoms, choice of optimum surgical approach, seizure outcome benefits of partial resection and role of reoperations/staged resections.

**Method:** We retrospectively reviewed surgically treated, consecutive patients with histologically proven hypothalamic hamartomas (1995-2011) treated at our centre. Fifteen patients were included in the study with mean age of 12.8 years (range 2-28,SD 10.1).

**Results:** Eight patients (53.3%) had intractable seizures (group 1), 5 (33.3%) had refractory precocious puberty (PP) (group 2) and 2 (13.3%)had both (group 3). Mean maximum dimension of the HH in groups 1,2 and 3 were 11.3 mm, 9.2mm and 17.5 respectively (p=0.34) and the median Delalande Fohlen radiological subtype was II in groups 1 and 2.Seizure types included purely single semiology-gelastic (2), CPS (1), both gelastic and CPS (3),both gelastic and generalized tonic-clonic (2) and all three types in one patient. Nineteen surgical procedures were done on 15 patients (mean 1.2/patient). Primary procedures were transsylvian approach in 10 patients (66.7%), transcallosal interforniceal in 3 (20%) and endoscopic excision in 2 patients (13.3%). Reoperations were by the transylvian route in two patients and endoscopic in one patient. Complete resection was achieved in 9 patients after the primary procedure (4,5 and none in groups 1,2,3 respectively). At a mean 3.7 years follow up the seizure outcome was noted in 4 patients with complete resection and 3 patients with partial resection (p=0.28) in group 1.Complete excision was achieved in 4 patients (50%) in group 1, 5 patients (100%) in group 2(p=0.057) and none in group 3.

Conclusion: In surgically treated hypothalamic hamartomas, the extent of excision correlates with

better symptom relief both for refractory seizures and precocious puberty. More than one surgical approach may be necessary and the optimum approach is decided by the size and radiological subtype of the lesion. Partial resection may also have a symptom relief benefit, particularly for seizures.

### 030

# Posterior quadrantectomy for patients with Sturge-Weber syndrome

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**Purpose:** Some patients with Sturge-Weber syndrome (SWS) need epilepsy surgery for good seizure control and prevention of psychomotor deterioration. Most frequent type of SWS has leptomeningeal angioma distributed temporal, parietal and occipital lobe. We indicate the posterior quadrant disconnection surgery for those patients. In this paper, we report the surgical procedure of the posterior quadrantectomy and the seizure and developmental outcome in patients with SWS. **Method:** We surgically treated 9 patients with SWS whose leptomeningeal angioma located in temporal, parietal and occipital lobe using the posterior quadrantectomy. After the craniotomy, we dissect the sylvian fissure to observe the inferior insular sulcus from the limen insulae to the distal end. Next step is disconnecting the temporal stem to the inferior horn of lateral ventricle. We can see hippocampus from the head to tail at this point. Corticotomy of parietal lobe is made to the trigon of lateral ventricle and the falx. Final step is the posterior corpus callosotomy and the disconnection of the fornix. Using this method, we can reduce blood loss and shorten surgical time.

**Results:** Seven in 9 patients with SWS who were operated using this method resulted seizure free and improved psychomotor development. We carried out the second surgery for the resting 2 patients with residual seizures, and finally resulted seizure free. Early surgery is better to seizure control and psychomotor development.

**Conclusion:** The posterior quadrantectomy for patients with SWS is suitable for good seizure control and psychomotor development. This method can reduce surgical insult in infant.

# **POSTER ABSTRACTS**

# Adult epileptology

#### p031

Effect of antiepileptic drugs on memory in elderly patients with cognitive impairment and epileptic seizures

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**Purpose:** To evaluate the effect of antiepileptic drugs on memory in elderly patients with cognitive impairment and epileptic seizures.

**Method:** Reported are two elderly patients with cognitive impairment and epileptic seizures, who were treated with antiepileptic drugs but never with anti-dementia drugs. Their memory was chronologically evaluated by Wechsler Memory Scale - Revised (WMS-R). Patient 1: A 73-year-old male who developed generalized tonic-clonic seizures at the age of 56. Valproate treatment was not effective and seizures worsened. He had gradually progressive forgetfulness and indifference for the past 3 years. His EEG showed sharp waves regional in left temporo-parietal area. His seizures were controlled after administration of carbamazepine as add-on therapy. Patient 2: A 79-year-old female who developed dyscognitive seizures with retrograde amnesia and forgetfulness for the past 1 year. Her EEG showed spikes regional in bilateral frontotemporal areas. Carbamazepine treatment suppressed her seizures.

**Results:** In Patient 1, WMS-R standardized scores of all five subcategories increased by more than 10 over 1 year. In Patient 2, a standardized score of attention/concentration increased by 7, and scores of other subcategories increased by more than 10, over 1 year and 8 months. **Conclusion:** Antiepileptic drug treatment can improve memory in some elderly patients with cognitive impairment and epileptic seizures.

### p032

#### Cardiac pacemaker implantation in a patient with ictal asystole associated with multi drugresistant temporal lobe epilepsy

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**Background:** Ictal asystole during seizure attacks in temporal lobe epilepsy (TLE) is very rare. Ictal asystole is potentially life-threatening, as sudden loss of consciousness may lead to falls, traumatic injuries and sudden unexpected death in epilepsy. So early diagnosis and effective intervention is very important. We describe a patient with left TLE who was confirmed to have ictal asystole by video-electroencephalography monitoring (VEM).

Case report: A 45-year-old man was admitted to our epilepsy center for recurrent episodes of drop attacks and complex partial seizures over the previous 5 years. He was unresponsive for a few seconds after feeling epigastric uprising sense, followed by losing consciousness with head drop and muscle atonia. He had visited to other hospital for an evaluation of his symptoms. He had been diagnosed as epilepsy and antiepileptic drugs (valproate, oxcarbazepine, levetiracetam, pregabalin, clobazam) were prescribed. Despite he had been treated with multidrugs, the frequency of the symptoms had increased during 2 year-time period. We examined the VEM with ECG. Video-EEG revealed frequent interictal spikes in left temporal areas. Furthermore, it showed one seizure: he was unresponsive with lip smacking, and suddenly lost his muscle tone for 20 seconds. EEG showed rhythmic delta activity in the left temporal (T1) area, which became generalized, and the ECG showed ictal asystole during the loss of consciousness and muscle atonia. Brain MRI showed no abnormalities. In order to rule out the heart problem, we checked echocardiography, holter monitoring, which also revealed no abnormalities. Because the patient had drug-resistant epilepsy. he received a permanent pacemaker implantation with medications (oxcarbazepine, levetiracetam). He continued to have complex partial seizures; however, falls or traumatic injuries were no longer reported after discharge.

**Conclusions:** Implantation of cardiac pacemaker can decrease serious morbidity in a subset of epilepsy patients by preventing seizure-related falls.

# Comparative analysis of quality life between patients with temporal lobe epilepsy with and without seizure

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**Purpose:** To investigate quality life difference between patients with temporal lobe epilepsy with seizure and without seizure

**Method:** From 144 patients with temporal lobe epilepsy we selected 28 patients into 2 groups. In first group we included 15 patients with rare seizure frequency 1-2 times in 3 months and second group consisted from 13 seizure free patients. Seizure free period of this patients were 1.5 · 2 years. QOLIE ·31P was translated into Mongolian was filled up by all patients. Self ·Rating depression Scale were used to assess the degree of depressive symptoms.

**Results:** Good quality life was noticed in patients from 1st group with no or light depression. Poor quality life with moderate depressive symptoms, other psychological distress such as irritability, angry outbursts were common in seizure free patients.

**Conclusion:** In Mongolia patients with temporal lobe epilepsy with dysphoric disorder have often passed by neurologist to the psychiatrist and vise versa. In view of this study we as neurologist should focus not only on seizure control, important to concentrate on psychiatric comorbidities, which is a big part of the patient's life. Early psychiatric evaluation and intervention would improve quality of life patients with epilepsy.

#### p034

# Amygdalar or amygdalohippocampal enlargement in patients with temporal lobe epilepsy: a hidden cause

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**Purpose:** The purpose of the study was to clarify the significance of amygdalar (AE) or amygdalohippocampal (AHE) enlargement in patients with temporal lobe epilepsy (TLE) detected by MRI.

**Method:** Twenty-seven TLE patients with AE (n=23) or AHE (n=4) treated at Severance Hospital were studied; prospectively, we included 13 cases and added 14 cases by retrospective investigation for patients with nonlesional TLE based on semological features and EEG findings from Yonsei Epilepsy Registry. AE or AHE on epilepsy-dedicated MRI was found by definite asymmetry of each side, as determined by the visual assessment and agreement of one neurologist and one neuroradiologist. **Results:** Average age of onset was 49 years (range, 6-81). Twenty-one patients were men. Twentythree patients had complex partial seizures (CPSs), eight patients had seizures exclusively or predominantly occurring during sleep, and seven patients had secondarily generalized seizures exclusively or predominantly occurring during sleep. Generally, the patients had shown high seizure frequency at their initial visits: eight patients had experienced daily occurring seizures. EEG with nasopharyngeal electrodes indicated focal epileptiform discharges exclusively or predominantly in the temporal area ipsilateral to the AE or AHE in all patients except for two patients with normal EEG. Enlarged amygdala or hippocampus showed iso- to slightly high intensity in FLAIR images without enhancement. Excellent response to antiepileptic drug treatment was found. Only two patients had experienced rare CPSs and aura only, respectively, and the other patients were seizurefree for the last 6 months of follow-up, when seven patients were excluded because of too short follow-up period.

**Conclusion:** The results of this study, in conjunction with semiological and EEG findings, indicate that AE or AHE may be an epileptogenic substrate in significant number of TLE patients, especially with late onset of age, who can be regarded as having nonlesional epilepsy although the pathologic nature is still unknown.

# A 20 year-old aged encephalitis patient with bilateral ovarian teratoma $\ensuremath{\mathsf{SHIN}}\xspace$ DJ

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**Purpose:** Paraneoplastic limbic encephalitis is unusual disorder that is characterized by severe neurological and psychiatric manifestations and has rarely been reported in association with ovarian teratoma. These symptoms mostly precede the gynecological diagnosis and cured with resection of ovarian tumor.

**Case:** We present a case of 20-year-old female patient who developed clinical seizures and confusion followed by psychiatric abnormal behavior and insomnia through 2 weeks. She was previously healthy. Among abnormal laboratory findings, CK-MB, LDH/CPK was increased by 10.15ng/ml, 652/5365 IU/L and CFS studies revealed that RBC 1/mm2 and WBC 18/mm2were increased. EEG showed continuous slow waves of low amplitude and brain MRI demonstrated the leptomeningeal enhancement of bilateral frontal lobe. The patient was treated with acyclovir, IV phenytoin and levetiracetam. The clinical symptoms did not improved and continuous slowing on EEG monitoring. Additional studies for paraneoplastic etiology demonstrated bilateral mature teratoma on abdominal CT. After ovarian cystectomy the patient showed rapid progressive neurological improvement with complete regression of the symptoms during follow-up. **Conclusion:** Female patients who present with symptoms of limbic encephalitis should be thoroughly screened for gynecological malignancy. The prompt recognition and surgical resection of ovarian teratoma is of great importance especially considering the fact that paraneoplastic limbic encephalitis can carry the risk of permanent disability or even death.

# p036

### Study of factors which may influence body weight in overweight patients of epilepsy: a crosssectional study from a tertiary care centre in New Delhi

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**Purpose:** Patients of epilepsy often struggle with abnormal body weight. This study was undertaken with the aim of studying factors associated with overweight in patients of epilepsy. **Method:** In this cross sectional study, consecutive epilepsy patients with disease duration of at least 2 years and history of regular AED use of at least one year were enrolled. Exclusion criteria were co-morbid illnesses which could account for limitation in physical activity or account for obesity. Using a questionnaire based interview, history including age of onset of epilepsy, type and frequency of seizures, any aura, level of consciousness, physical activity before seizures, nocturnal or daytime occurrence and control of seizures was obtained. Assessment of coexisting depression, obesity, physical activity and treatment details regarding use of antiepileptic drugs was also documented.

**Results:** 160 consecutive patients with median age of 21(14-41) years were enrolled. There were104 males. Normal BMI (< 23) was noted in 115 and increased BMI (<23) in 45. The variables significantly associated with obese group were female sex(p=0.01), higher annual seizure frequency, physical inactivity(p=0.03),past history of seizures during physical activity(p< 0.0005), depression, use of antidepressants, more number or AEDs(p=0.03), use of valproate(p< 0.0005) and clobazam(p=0.01). On logistic regression analysis significance was reached only for valproate use and presence of depression (p< 0.0005).

**Conclusion:** It is more likely that instead of any single factor causing obesity in patients with epilepsy there are multiple factors acting together. Many of these factors are modifiable and can be successfully managed with patient education and counseling.

#### **AED** issues

p037 Pharmacokinetics of carbamazepine in Mongolia for people with partial epilepsy MANDAKH B, <u>MANDSHIR U</u>, ULZIIBAYAR D Health Science University of Mongolia, Ulaanbaatar, Mongolia

**Purpose:** The aim of the present study was to determine pharmacokinetic parameters (PP): volume of distribution (Vd), clearance (Cl) and half life (T1/2) of carbamazepine (CBZ).

**Methods:** 30 patients with symptomatic focal epilepsy: 14 males and 16 females included in the study. The average age was  $29.9 \pm 8.7$  (range 16.45 years). The mean duration of partial seizure was  $11.4 \pm 5.8$  years. All patients were using not appropriate doses of CBZ and most of patients were taking it irregularly. Patients were follow-uped during 3 months. Initial dose of treatment was  $9.4 \pm 2.2$  mg/kg. Pharmacokinetic analysis was performed by ABBOTT TDXFLX immunofluorescence autoanalyser. The blood samples were collected from patients in the morning before taking the morning dose and after 2 hours. CBZ pharmacokinetic parameters were measured by the equations (VD, CI and t1/2).

**Results:** Therapeutic effective serum level of CMZ in 18 seizure free patients (60%) was  $8.7\pm1.2 \mu$ g/ml (range 6.89·10.77). There was poor correlation between CBZ daily dose and serum concentration Cmax (r=0.236,p=003). Frequency of seizure was decreased after CBZ treatment (t=8.479; p=0.0001; Cl 95%). Vd was 1.36 l/kg, Cl was 0.66 ml/min/kg, T1/2 was 23.78 hours. No relation between CBZ daily dose (780 mg/day) and CL (0.66ml/min/kg) (r=-0.01). Strong relation between weight and CBZ clearance r=0.69.

**Conclusion:** Pharmacokinetic parameters of CBZ was performed for the first time in Mongolia. Our result clearly demonstrates that is no difference of the Pharmacokinetic parameters of CBZ between Mongolian patients and patients from other Asian and Western country.

The study has been approved at #20/6 meeting of Biomedical Ethics Committee in 16 Jun 2010.

# p038

# The efficacy, safety, and pharmacokinetics of intravenously administered Fosphenytoin Sodium in Japanese patients with status epilepticus and neurosurgery

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**Purpose:** Phenytoin sodium (PHT) for injection is formulated in a hypertonic solution of pH 12 with an osmotic pressure ratio of approximately 29 to physiological saline. Hence, it can cause pain, phlebitis, and purple glove syndrome at injection site. Fosphenytoin sodium (FOS) is a water-soluble formulated at a pH 8.5 to 9.1, and an osmotic pressure ratio of approximately 1.9. We assessed the efficacy, safety and pharmacokinetics of FOS in Japanese patients with status epilepticus, and with need of seizure prevention following neurosurgery or head traumatics.

**Method:** Total 47 patients aged 2-86 years old with status epilepticus (SE), repetitive seizures (RS), brain surgery, or head trauma were enrolled in this open-labelled study. FOS was administered intravenously at a loading dose of 18 or 22.5 mg/kg for seizure treatment and 15 or 18 mg/kg for seizure prevention. The maintenance doses were administered, if needed.

**Results:** 50% or more reduction of seizure activity was observed in 65.4% of 26 patients with SE or RS. Out of 21 patients with post-operative or traumatics, no seizure was observed 7 days before and after FOS treatment in 14 patients, disappearance or reduction of seizures were observed after FOS treatment in 6 patients. One patient of 21 patients showed a seizure after neurosurgery. Plasma total PHT concentrations after a loading dose of FOS increased in the range of 10-20 µg/ml dependent on the dose level, with Cmax rising linearly. The most of adverse events were mild and there was no serious adverse event and discontinuation in this study.

**Conclusion:** FOS, intravenously administered at a loading dose of 22.5 mg/kg, was optimal in Japanese Paediatric and adult patients with SE and RS. In patients with neurosurgery, the loading dose of 15-18 mg/kg is expected to exert preventive effects for early seizures. FOS is considered to be a useful drug with almost no local irritation at injection site and can be substituted for PHT.

# Vitamin D deficiency in children with epilepsy who are taking anticonvulsants YU J. KOH JW

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**Purpose:** Vitamin D plays an important role in calcium metabolism and also has been linked to several brain disorders, including cognitive decline and epilepsy. Many studies have reported the association between anticonvulsants and metabolic bone disease. We evaluated vitamin D levels in children with epilepsy who are taking anticonvulsants to find the prevalence and risk group of vitamin D deficiency.

**Method:** Serum vitamin D levels were checked in 139 children who are taking anticonvulsants from the department of Paediatrics in Dankook University Hospital. Type and duration of medication, and blood chemistry were analyzed using their medical records. Vitamin D deficiency was defined as serum levels less than 20 ng/mL by the Endocrine Society Clinical Practice Guideline 2011. **Results:** Seventy one were males. Their average age at study was 11.3 years. Average duration of medication was 5.32 years. Ninety eight (70.5%) had vitamin D deficiency, with average level of 10.1 ng/mL. Serum calcium levels were significantly lower in the group with vitamin D deficiency, but there was no significant difference in gender as well as the levels of phosphorus and alkaline phosphatase compared to that of non-vitamin D deficient children. Forty seven children (33.8%) were taking more than two anticonvulsants, 35 (74.5%) were vitamin D deficient. Vitamin D deficiency is more frequent in adolescents.

**Conclusion:** Vitamin D deficiency was common in children, especially in adolescents, with epilepsy who are taking anticonvulsant. It was frequently observed in children with more than one anticonvulsant (74.5%). This study suggests to monitor vitamin D status and also to encourage sufficient sunlight exposure and adequate exercise in children with anticonvulsants to prevent vitamin D deficiency.

# p040

# Duration is associated with responsiveness to AED in lesional epilepsy KIM SE

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**Purpose:** Even if the response to antiepileptic drug (AED) in partial epilepsy with structural lesions on MRI (lesional epilepsy) is less favorable than without structural lesions, there has been few study focusing on the responsiveness to AEDs in lesional epilepsy. The aims of this study were to clarify the prognostic factors for responsiveness to AEDs in lesional epilepsy.

Method: On the epilepsy database we found 787 patients with partial epilepsy who had MRI. The inclusion criteria for this study were 1) having structural lesions on MRI, 2) taking AEDs for at least 1 year, 3) age  $\geq$  13 years old. The definition for AED resistance was the failure of adequate trials of two tolerated, appropriately chosen and used AEDs to achieve sustained seizure freedom. We analyzed the risk of AED-resistance in lesional epilepsy with multiple logistic regression. Results: Of 787 patients with partial epilepsy, 234 patients met the inclusion criteria for this study. The median age was 22 years old (range 13-78 years old) and the median duration (interval between AED on and the last follow-up) was 131 months (range 12-516 months). Male was 49% (115/234) and 40% (94/234) were AED-resistance. Of the pathology of lesions on MRI, hippocampal sclerosis (HS) was most frequent. Other pathologies were neuronal migration disorder (NMD), cerebromalatic lesions related with trauma, tumor, vascular malformation and cerebral infarction in order. Duration was only independent variable associated with AED-resistance after multiple logistic regression (OR 4, 95% CI 2.2-7.6, p< 0.0001), whereas the pathology of lesional epilepsy on MRI was not significant. The median duration in the patients with AED-resistance was significantly longer than that without AED resistance 178 months (range 23-516 months) Vs 102 months (range 12-479 months), p< 0.0001 by Mann-Whitney test).

**Conclusion:** In lesional epilepsy, the responsiveness to AEDs may be expected to decrease when the duration is prolonged.

# Metabolic syndrome among obese Chinese epileptic patients treated with valproate $\underline{FANG}\ J,\ ZHOU\ D$

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**Purpose:** This study was conducted to evaluate the incidence of metabolic syndrome (MetS) among adult obese epileptic patients treated with valproate(VPA); special attention was given to the component characteristics of MetS. In addition, the possible relevant factors for the development of MetS among patients were determined.

**Method:** With the aim of evaluating the incidence of MetS, thirty-six patients receiving VPA monotherapy were included, all of them were interviewed, clinically examined, and blood samples after overnight fasting were obtained. An oral glucose-tolerance test (OGTT) was subsequently performed. Twenty-eight subjects who were diagnosed as "simply obesity" were collected as controls.

**Results:** The two study groups were well matched for age, gender and body mass index. Insulin resistance, which was measured by homeostasis model assessment (HOMA) index was more severe among the obese patients group  $(4.91\pm2.91 \text{ vs}. 2.00\pm1.72, P=0.007)$ . The frequency of the MetS was slightly higher in patients compared to controls (47.2% vs 32.1%, respectively), but did not reach statistical significance (P=0.223). In terms of MetS components, the patients group tended toward a higher risk for developing high blood pressure (55.6% vs 28.6%, P = 0.031) whereas lower proportion unexpectedly met the criterion for low HDL-C (36.1% vs 60.7%, P = 0.05).Multivariate analysis with stepwise logistic regression revealed that low positive correlations were observed between MetS developing and both HOMA index (P=0.029; r=0.361) and valproic acid dose (P=0.049; r=0.323), but independent of other clinical parameters.

**Conclusion:** Our preliminary study suggests that obese patients treated with VPA have a higher risk of developing MetS. The development of MetS among obese patients treated with VPA may be different from the general obese population. Rather than monitoring body weight alone, the HOMA index should be monitored during prescribing VPA.

#### p042

# Evaluation of different antiepileptic drug strategies in medically refractory epilepsy patients following epilepsy surgery

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**Purpose:** This study aimed to explore the most appropriate antiepileptic drug strategies after successful epilepsy surgery.

**Method:** A total of 131 refractory epilepsy patients who underwent epilepsy surgery from January 2005 to December 2008 in the Department of Neurosurgery, West China Hospital, were retrospectively reviewed. Patients were divided into three groups (monotherapy, duotherapy, and polytherapy) according to drug combinations used immediately after epilepsy surgery. Seizure outcomes were followed up for more than 2 years. Engel classification was used to evaluate seizure outcomes.

**Results:** The mean postoperative follow-up period was  $3.7\pm1.0$  years. Preoperative baseline data among the three groups were comparable. Seizure recurrence rate in monotherapy was obviously higher than in other groups (34.1% vs. 15.1%, 7.1%) at 6-month follow-up, which showed a statistically significant difference (p=0.02). Seizure outcomes for 2 years were assessed using Engel classification. In the duotherapy group, the rate of Engel class I was definitely higher than in the other two groups (69.9% vs. 47.7%, 57.1%, p=0.02). Seizure relapse rates at the 2-year follow-up, after planned reduction or withdrawal, were 46.4% for monotherapy, 16.9% for duotherapy, and 25.0% for polytherapy (p=0.01).

**Conclusion:** Monotherapy may be not sufficient enough to control seizures completely. It appears to have a higher risk for seizure relapse when considering drug reduction. Duotherapy seems to be more effective than monotherapy. Even after successful epilepsy surgery, duotherpy seems preferable to montherapy or polytherapy for control of residual seizures.

# Comparison of dried blood spot and plasma measured valproic acid levels in patients with epilepsy

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**Purpose:** Therapeutic drug monitoring (TDM) of valproic acid (VPA) plasma levels is commonly performed in patients with epilepsy (PWE) to assess compliance and guide dosage optimization. Currently TDM involves invasive sampling, requirement of a phlebotomist, and cumbersome transportation of specimen. Dried blood spot (DBS) represents an alternative approach that overcomes some of the current issues on using plasma as a monitoring matrix. Since the established target therapeutic drug level of VPA is based on plasma quantifications, we aim to correlate DBS measured VPA levels with its plasma levels, to determine whether it could serve as a good alternative to the traditional TDM.

**Method:** This study was conducted in a tertiary referral hospital in Singapore after obtaining approval from Institutional Review Board A. 23 PWE who visited Neurology Clinic in October 2011, taking VPA for more than 2 weeks and had routine plasma TDM on the visit day were included. DBS levels of VPA were quantified by a previously developed gas-chromatography mass-spectrometry assay while the plasma levels were measured using conventional plasma immunoassay available from the hospital laboratory. Evaluation was performed using Statistical Package for Social Sciences (SPSS) version 19.

**Results:** Levels measured from the DBS were well-correlated to the levels measured from plasma (r > 0.877). The conversion factor for DBS measured levels to plasma levels is as follows: Plasma VPA level = (1.778 x DBS VPA level) + 11.729.

**Conclusion:** DBS measured VPA level appears to be a useful alternative measurement and could be considered for routine estimation of plasma VPA levels.

# p044

### A survey of the use of antiepileptic drugs in stroke patients

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**Purpose:** Seizures occur in about 2~20% of stroke patients. The impact of post-stroke seizures on stroke outcome remains unclear. But recent studies reported that post-stroke seizures are associated with a higher mortality or worse functional outcome. Therefore, it is an important issue to use antiepileptic drugs (AED) in stroke patients. It complicates this issue that some AEDs may adversely affect motor recovery. Nevertheless, there are no official guidelines on how to use AED in stroke-related seizures. In this study we survey neurologists and neurosurgeons and compare the difference between subgroups categorized by department, specialty and workplace discrimination by a survey which contains questions investigating the present propensity for the use of AEDs in stroke patients.

**Method:** 256 neurologists and neurosurgeons participated in this survey. The research instrument was a questionnaire comprising 9 parts including 30 questions. The questions are consisted of the stroke mechanism, the prophylactic use of AED, the choice of AED in early and late onset post-stroke seizure.

**Results:** Propensity for the use of prophylactic AEDs in stroke revealed significantly difference as department and workplace (Neurologist vs. Neurosurgeon; 17.8% vs. 83.1%, p< 0.001, Hospitalists vs. Faculties; 46.2% vs. 28.4%, p=0.05). The most commonly chosen prophylactic AEDs are valproic acid (75%) and levetiracetam (60%). Carbamazepine is the most commonly used AEDs (66%). Phenytoin (1.6~15.2%) and phenobarbital (1.7~2.6%) are used in patients with post-stroke seizures.

**Conclusion:** There are significant differences in applying of prophylactic AED after stroke between neurologists (17.8%) and neurosurgeons (83.1%). Propensity of starting AED in Korean neurosurgeon was higher than that of neurosurgeon in America (24%) and Germany (4%). Valproic acid (75%) and levetiracetam (60%) are considered as first-line prophylactic AEDs by neurosurgeon, although there is no evidence for their effectiveness. Furthermore, phenytoin

 $(1.6 \sim 15.2\%)$  and phenobarbital  $(1.7 \sim 2.6\%)$  are still used in post-stroke seizure although they have been reported to have an adverse influence on motor recovery. It seems that proper guidelines for AED in stroke-related seizure should be established.

# p045

# The EpiNet project in Asia-Oceania; a means to undertake collaborative clinical research in epilepsy

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**Purpose:** The EpiNet study group intends to undertake investigator initiated research into the management of epilepsy. Our aim is to use the Internet to conduct large, simple clinical trials that are investigator initiated and independent of pharmaceutical companies. The EpiNet platform is ideally suited to study epilepsy syndromes in the Asia-Oceania region.

**Method:** A secure epilepsy patient database (the EpiNet database) has been created . The database is held on a server in Auckland, New Zealand. It can be accessed by approved investigators via a secure, password-protected website from anywhere in the world. Information is collected according to multiple axes; epilepsy overview; seizure history; electroclinical syndrome; aetiology; investigations; drug treatment; miscellaneous (including intercurrent illnesses). All data is encrypted. Forms are designed as a series of expandable trees. They can rapidly and efficiently collect very precise information. Drop down lists are presented whenever possible, to ensure uniformity of data collection. Some fields are mandatory, but most are optional.

Neurologists and epileptologists joined the project from late 2010. Participants were required to obtain approval from their ethics committees.

**Results:** As of Nov 20, 2011, 1010 patients had been registered in the EpiNet database by neurologists or epileptologist from 25 centres in 13 countries. 805 of these patients had been registered by 54 investigators or research assistants from 7 countries within the AOEC region (Australia, India, Korea, Malaysia, New Zealand, Pakistan, and Sri Lanka.) Over 50 different aetiologies were identified. Investigators stated that more than half of these patients would be suitable for clinical trials.

**Conclusion:** The EpiNet collaboration has demonstrated that neurologists working from quite different cultural backgrounds and in very different health systems are able to combine resources to undertake clinical research in epilepsy. We are now establishing several registries within the global EpiNet database, and we hope to start running clinical trials within the next year. We invite interested epileptologists from the Asia-Oceania region who are not already involved to join the EpiNet study group.

### p046

# Low occurrence of skin rash with antiepileptic drugs in Pakistan: do ethnic / racial variations exist? MOGAL Z, MAHMUD H, AZIZ H

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**Purpose:** It has been an anecdotal observation, over many decades by the authors, that occurrence of skin rash associated with use of antiepileptic drugs (AEDs) in people with epilepsy in Pakistan is less than that reported in world literature. We hypothesize that ethnic / racial dermatological features may influence the susceptibility.

**Methods:** Retrospective analysis of patients' follow-up records to determine occurrence of skin rash in people with epilepsy and on atleast one AED at a tertiary-care epilepsy centre was done. The results are compared with those reported in published literature.

Results: Eighty-eight of the total 1943 patients (4.5%) reported side-effects. Of these only 11

(0.57%) developed skin rash; non-serious skin rash in 06, Steven Johnson syndrome in 03, Drug Rash with Eosinophilia and Systemic Symptoms (DRESS) in 01 and Toxic Epidermal Necrolysis (TEN) in 01. The patient with TEN expired. Seven of the 11 patients were on monotherapy whilst 04 on polytherapy. Of all the AEDs, carbamazepine was found to be most responsible for skin rash whilst valproate and lamotrigine were also accountable in a small minority.

**Conclusion:** Occurrence of AED-associated skin rash in Pakistan is much less (0.57%) than that reported in other countries like USA (14.3%), Norway (14%), Canada (8.5%) and China (3.6%). We hypothesize that this variability may be due to ethnic / racial dermatological features. Further information from other countries' research is required to substantiate or refute the hypothesis.

# p047

## A phenytoin-induced neutropenia case

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**Purpose:** Phenytoin is conventional anti-epileptic drug (AED) for long time and essential to control status epilepticus pat-ients. Phenytoin has many kinds of adverse drug reaction including dose-related toxicity and hypersensitivity rea-ctions. But adverse effects in hematologic disorders are rare. We report a patients showed severe neutropenia with phenytoin use.

**Case:** A 72-year-old man was admitted for confusional mental state, dysarthria, mild right-sided weakness. We diagnosed as non-convulsive status epilepticus (NCSE) and treated with IV phenytoin and maintained 300mg for oral intake. He developed severe neutropenia after 8 days of pheny-toin therapy and reverse to normal after stop the medication.

**Conclusion:** This case report of severe neutropenia occurring in an adult emphasizes the importance of monitoring complete blood cell counts with phenytoin therapy.

## p048

# The evaluation of implementing HLA-B\*1502 genotyping test at an epilepsy clinic of a university hospital

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**Purpose:** To survey the results of HLA-B\*1502 genotyping and to evaluate the benefits of seizure control after taking carbamazepine (CBZ) among patients at an epilepsy clinic of a university hospital.

**Method:** From July 2008 to October 2011, HLA-B\*1502 genotyping test was arranged when patients agreed to take CBZ. The indications included patients with chronic side effects or inadequate seizure control under monotherapy or polytherapy with phenytoin (PHT), and patients with either newly diagnosed epilepsy or intractable partial epilepsy never treated with CBZ. Exclusion criteria were patients having history of transplantation, flu symptoms with ulcer, blister, or rash and being not Han Chinese descent. The benefit of taking CBZ was evaluated by the comparison of seizure frequency changes between six months (24 weeks) before and six months after taking CBZ. **Results:** Seven (5.47 %) out of 128 patients had positive HLA-B\*1502 genotype. Six out of 121 patients with negative HLA-B\*1502 genotype refused to take CBZ. Eight (6.9%) out of 115 patients taking CBZ developed mild skin eruption and CBZ was then withdrawn in all of these patients and CBZ substituted PHT in 38 patients. Three patients with newly diagnosed epilepsy were treated with CBZ. In add-on group and substituted group patients, 66 (67.5%) out of 98 patients became seizure free.

**Conclusion:** HLA-B\*1502 genotype positive rate was 5.47% in our cohort. 6.9% of 115 HLA-B\*1502 negative patients developed mild skin eruptions after taking CBZ. Among patients with CBZ substitution and CBZ add-on, two-third of them achieved seizure free.

# **Basic science**

# p049

# Intervention of seizure-induced neuronal death by inhibition of NADPH oxidase activation <u>CHOI HC</u><sup>1</sup>, SUH SW<sup>2</sup>, SONG HK<sup>3</sup>

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**Purpose:** to evaluate the neuroprotective effects of apocynin, a NADPH oxidase assembly inhibitor, on seizure-induced neuron death. Here we tested our hypothesis that post-treatment of apocynin may prevent seizure-induced neuronal death by suppression of NADPH oxidase-mediated superoxide production.

**Method:** Epileptic seizure was induced by intraperitoneal injection of pilocarpine (25mg/kg) in male Sprague-Dawley rats. Animals were treated with lithium chloride 1 day before the pilocarpine injection. Diazepam (10 mg/kg) was administered into intraperitoneal space 2 hours after onset of status epilepticus. Apocynin (30mg/kg) was injected into the intraperitoneal space 2 hours after seizure onset and then the second injection was performed 24 hours after seizure. To test whether apocynin inhibits NADPH oxidase activation-induced reactive oxygen species (ROS) production, dehydroethidium (dHEt) was injected into the intraperitoneal space before pilocarpine injection and brains were harvested 3 hours after diazepam injection (5 hours after seizure onset, 5 mg/kg). Neuronal death was evaluated with Fluoro Jade-B (FJB) staining and microglia activation was evaluated with CD11b immunostaining in the hippocampus three days after seizure. **Results:** The present study found that apocynin decreased the intensity of dHEt fluorescent signal. The number of FJB-positive neurons was reduced by treatment of apocynin after seizure.

**Conclusion:** the present results suggest that inhibition of NADPH oxidase by apocynin may have a high therapeutic potential to reduce seizure-induced neuronal death.

### p050

# Anticonvulsant actions of CyPPA in pharmacoresistant human epileptic tissue

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**Purpose:** Pharmocoresistance in epilepsy patients is of one the major treatment problems, over 30% patients are non-responsive to current anti-epileptic drugs. We have previously assessed anticonvulsant actions of CyPPA in acute slices of rats and slice cultures. In the present study we test effects of CyPPA in human tissue obtained from pharmacoresistant epileptic patients. **Method:** Using 4-AP and high potassium+bicuculline, seizure like events (SLEs) were induced in temporal cortex surgically resected from pharmacoresistant epileptic patients. Local field potential recordings were performed using interface setup with glass electrode filled with aCSF. Input out curve is determined before recording to confirm quality of slice.

**Results:** CyPPA at dose of 100 $\mu$ M blocked SLEs in 100% of tissues (n=6) induced by 4-AP (100 $\mu$ M), whereas 60% blockade has been observed with SLEs induced by high potassium+Bicuculline (50mM). Parameters such as frequency, amplitude, duration of SLEs and negative DC shift were also significantly altered.

**Conclusion:** Results suggested that CyPPA (sk-channel enhancer) may be new option for the treatment of pharmacoresistant type of seizures.

#### p051

# Experimental study on the neuroprotection role of Baclofen in hippocampus of epilepsy rats induced by kainic acid

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**Purpose:** Baclofen is the agonist of GABA<sub>a</sub>, the relationship of Baclofen to GABA<sub>a</sub> receptor and NSE is not definated. The aim of this study is to observe the protein expressions of GABA<sub>a</sub> and NSE in

hippocampus of epilepsy rats.

**Method:** Epilepsy rat models were made by injecting kainic acid 1µl into amygdale under stereotactic instrument. 70 male Wistar rats were randomly divided into 7 groups: Group A is the control group; group B the false operation group; group C 0.5h epilepsy group; group D 2h epilepsy group; group E 6h epilepsy group; group F 12h epilepsy group; and group G 24h epilepsy group. The protein expressions of GABAB and NSE in the rat brain were tested by the methods of immunohistochemistry, and the grey values were calculated.

**Results:** The grey value of GABA<sub>a</sub> and NSE positive cells in the group A B was 15.43 $\pm$ 1.32, 15.34 $\pm$ 1.58, 1543357 $\pm$ 1324, and 1543215 $\pm$ 1356 respectively. The protein expression of GABA<sub>a</sub> decreased from 2h (26.34 $\pm$ 2.52) to 24h (23.43 $\pm$ 3.31), attain the lowest point at 6h (27.31 $\pm$ 3.02) after kindling. The protein expression of NSE increased from 0.5h (2634768 $\pm$ 2527)to 2h (5230965 $\pm$ 3021),then returned to normal level at 6h (1643746 $\pm$ 3310).

**Conclusion:** Baclofen increased the protein expression of GABA<sub>B</sub>, and decreased the protein expression of NSE play an neuropotection role in epilepsy brain injury.

#### p052

# Hippocampal dentate granule cell neurogenesis and phenotypic differentiation in mice after pilocarpine-induced seizures

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**Purpose:** Hippocampal dentate granule cells might play an important role in the pathogenesis of human temporal lobe epilepsy. Proliferation, differentiation, and survival of dentate granule cells have been reported to be influenced by epileptic seizures. This study was designed to investigate hippocampal dentate granule cell neurogenesis after pilocarpine-induced seizures in mice. **Method:** Fifteen male ICR mice at postnatal day 21 were divided into two groups: pilocarpine-

treated group (n=7) and control (saline-treated) group (n=8). Seizures were chemically induced by intraperitoneal injection of pilocarpine (300 mg/kg). Bromodeoxyuridine (BrdU, 50 mg/kg) was subsequently administered once a day for 6 consecutive days, starting at 24 hours after pilocarpine or saline treatment. Mice were sacrificed 24 hours after the last BrdU injection (n=4 in each group). We examined BrdU-positive cells in the dentate gyrus by immunohistochemistry. To investigate the phenotypic pattern of newborn cells, the animals were allowed to survive for 28 days after the last injection of BrdU. We examined the long-term fate of BrdU-positive cells after seizures by doublelabeled immunofluorescence with confocal microscopy.

**Results:** After pilocarpine administration, every seizure behavior was grade 3 or more. Quantitative analysis revealed that BrdU-positive cells were significantly increased in the pilocarpine-treated group compared to control (230.55±59.50 vs. 148.67±40.00, p< 0.05). The majority of these mitotic cells (92 %) were differentiated into neurons.

**Conclusion:** Our results indicated that mitotic activity in the dentate gyrus was enhanced after pilocarpine-induced seizures in mice, and the majority of all BrdU-positive cells showed the phenotypic differentiation to neuronal cells.

### p053

#### Effect of esomeprazole on pharmacokinetics of phenytoin

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**Purpose:** Esomeprazole is commonly prescribed proton pump inhibitor for gastritis and peptic ulcer disease. Most of the time in clinical practice, phenytoin and esomeprazole are prescribed for patients of generalized seizures with concomitant peptic ulcer. Hence there are chances of drugdrug interaction because of modulations of isoenzymes CYP2C9/10 and CYP2C19, are involved in metabolism of phenytoin and esomeprazole. But it is important to maintain the therapeutic level of phenytoin in plasma for effective seizures control. So, the aim of the study was to determine the effect of esomeprazole on the pharmacokinetics of phenytoin in rabbits.

**Methods:** In a parallel design study, phenytoin, 30 mg/kg/day per oral was given daily for 14 days. On day 15, blood samples were taken at various time intervals between 0-24 hours. In esomeprazole-phenytoin group, phenytoin was administered for seven days as mentioned earlier

and from day 8<sup>th</sup> onward, esomeprazole 2.8 mg/kg along with phenytoin 30 mg/kg/day was administered till 14<sup>th</sup> days and blood samples were drawn as above on 15<sup>th</sup> day. Plasma phenytoin levels were assayed by HPLC and pharmacokinetic parameters were calculated. In esomeprazole-phenytoin group, there was a significant increase of  $t_{ye}$  than phenytoin alone group and significant increase in AUCO<sub>.24</sub> was also observed in the esomeprazole and phenytoin treated group. **Results:** Esomeprazole significantly prolonged the absorption of phenytoin, and the absorption method. (Constant)

constant (K<sub>a</sub>) was found to be (0.303573 vs 0.56518) whereas  $t_{1/2}a$  was significantly increased (1.226157 hr-1 vs 2.28281 hr-1). The elimination half life of phenytoin, ( $t_{1/2}a$ ) (37.86528 vs 55.7367 hr) and the area under the curve (AUCO<sub>24</sub>) (10.88325 vs 326.951 µg/ml.h) increased significantly when it was combined with the esomeprazole as compared to phenytoin alone. There was 35 fold increased in the AUCO<sub>24</sub> of phenytoin in the combination group as compared to phenytoin alone group.

**Conclusion:** Esomeprazole alters the pharmacokinetics of phenytoin. Confirmation of these results in further clinical studies will warrant changes in phenytoin dose or frequency when esomeprazole is co-administered.

# **Clinical neurophysiology**

# p054

# Transient increase of serum ammonia level in differential diagnosis of seizure and syncope <u>CHOI YH</u>, CHO YN, LEE S-Y, KIM W-J

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**Purpose:** The purpose of this study is to review the use of serum ammonia level in the differential diagnosis of seizure and syncope.

Differential diagnosis between a generalized tonic-clonic seizure and syncope may be difficult due to similar clinical features. The need for a biological marker to distinguish a seizure from syncope has been emphasized from past studies. Transient hyperammonemia could be an indicator of recent convulsive seizure.

**Method:** Adult patients who were admitted to the Department of Neurology at Gangnam Severance Hospital with final diagnosis of a generalized tonic-clonic seizure or syncope were eligible for this study. Serum ammonia levels were checked within 24h after a loss of consciousness.

**Results:** Among the patients with a loss of consciousness who underwent analysis of serum ammonia level, diagnoses were made with a seizure (n = 68) and syncope (n = 40). The seizure group had  $68.78 \pm 69.615$  umol/L and the syncope group had  $28.33 \pm 10.014$  umol/L of ammonia level, respectively. The seizure group presented with a significantly increased serum ammonia (p< 0.05) compared to the syncope group. The cut-off value with the reliable diagnostic level was defined as 36 umol/L with a sensitivity of 0.65 and specificity of 0.80 by receiver operating characteristic (ROC) curve analysis.

**Conclusion:** Serum ammonia measurement may be a useful test for the identification and differential diagnosis of seizure and syncope. Therefore, it is useful to checking the serum ammonia level during acute post-ictal status for the differential diagnosis of seizure and syncope.

#### p055

### Vertiginous seizure and postural control

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**Purpose:** Vertiginous seizure is one of the most common symptoms of complex partial seizure. They can be brought on- or triggered by stress and muscle strain. The purpose of this study was to investigate whether patients with stress-induced vertiginous seizure(SVS) have abnormalities of postural control, and to disclose any frequency shift of the centre of gravity while standing upright. **Method:** Thirty five SVS patients with tenderness in pericranial muscles were compared with age and sex matched controls. The questionnaires for stress amount were asked to both groups. All the subjects underwent balance measurements that included vestibular evoked myogenic potential (VEMP) testing, static and computerized dynamic posturography. **Results:** Stress amount were significantly increased in the patient group. SVS patients had reduced VEMP amplitudes compared to the controls. The spectral frequency analysis of body sway while standing upright was investigated. The sway distance and the sway area of center of foot pressure significantly increased in the patients with SVS. Spectral frequency analysis showed a significant increase in spectral power below 1.00Hz frequency band. Computerized dynamic posturography showed decreased somatosensory ratio in the patients with SVS.

**Conclusion:** These findings suggest that stress may induce abnormal vestibulo collic reflex and a significant proprioceptive disturbance in vertigious seizure patients.

# EEG

## p056

# Amplitude integrated electroencephalography in the neonatal intensive care unit for diagnosis of neonatal seizure

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**Purpose:** To assessment the usefulness of amplitude integrated electroencephalography (aEEG): screening tool for seizure detection. And We compared a aEEG with EEG (electroencephalography) that is one of the most useful tool for assessment of neonatal seizure detection in the neonatal intensive care unit.

**Method:** We retrospectively studied 24 neonates who have been suspected seizure on neonatal intensive care unit from January 2009 to february 2010. The 24 neonates had seizures or apnea that did not respond to aminophylline or a continuous positive airway pressure. We compared the efficacy of diagnosis among aEEG, EEG and neurosonography.

**Results:** We analyzed 24 aEEGs in neonatal intensive care unit. The aEEGs showed that seizure pattern in 15 (62.6%), burst suppression pattern in 2(8.3%), flat pattern in 4 (8.3%), and normal 5(20.8%). The result of EEGs were neonatal seizure 15(62.5%), cerebral dysfunction 2 (8.3%), normal 7 (29.2%). The result of aEEG and EEG showed the correlation (P < 0.05). But result of aEEG and echoencephalography did not show correlation.

**Conclusion:** There is correlation between aEEG and EEG for seizure detection in neonatal intensive care unit. We think that aEEG is a useful screening tool for seizure detection.

# p057

# Automatic abnormal waves detection from the electroencephalograms of epilepsy cases to sort out the spikes, the sharps, the polyphase based on wavelet transformation-autocorelation <u>NOERTJAHJANI S</u><sup>1,2</sup>, SUSANTO A<sup>3</sup>, HIDAYAT R<sup>3</sup>, WIBOWO S<sup>4</sup>, MUTAQIN Z<sup>5</sup>, UNDARIS

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**Purpose:** Wavelet transformation is applied to electroencephalogram records from epileptic patients. The temporal sharpness associated with interictal spikes at different resolutions is observed and two ways for representing the multiresolution sharpness of the spikes. **Method:** Patient consist of 349M,234F, ages 3-68 years. Clinical status: epileptic (136M,80F: EEG with spike;47M, 40F without); clinical status: nonepileptic: (98M, 65F EEG with spike; 68M, 49F without). Numerical data were acquired with EEG dump diagnostic Biologic system at Sarjito hospital Yogyakarta etc, 2002-2011. The wave components were sorted out according to their amplitudes, time span between zerocrossings, and different frequencies wavelet.

**Results:** The experimental results the spikes show consistent large outputs throughout the wavelet set, they have sharpness at several different resolutions. Utilizing the hardware and software facilities at hand, marking the starts and ends of abnormal waves could be done with +100µV threshold. The zerocrossings and autocorelation detection could automatically distinguished according to the 20ms-70ms time period for the "spikes" (109M,68F),70ms-120ms for "sharps" (147M, 98F), and the existence of multiple peaks for "polyphase"(93M,68F).

**Conclusion:** The research carried out so far was to find the prospect of this digital signal processing on EEG waves to support the doctors' work in this field. Keywords: EEG. spike, wavelet.

# p058

# Ictal high-gamma oscillation (60-99 Hz) in intracranial electroencephalography and postoperative seizure outcome in neocortical epilepsy

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**Purpose:** High-gamma oscillations (HGOs) (60-99 Hz) have been suggested to correlate with seizure onset zones and seizure outcomes. We investigated the correlation between the extent of removal of ictal HGO generating areas and postoperative seizure outcome in neocortical epilepsy (NE). **Method:** 23 patients with medically intractable NE underwent chronic intracranial electroencephalography (iEEG) using subdural electrodes. Ictal HGOs and superimposed undersampled ripples within ± 3 s of video-iEEG ictal onset were extracted by wavelet clustering and thresholding. Cluster epileptogenicity indices (CEIs) were calculated. The temporal analysis window was locked to the timing of the maximum CEI wavecluster. Root mean square amplitudes, cross-correlation synchronies and the local focus indices within the temporal window were calculated.

**Results:** Percentages of resected maximum CEI waveclusters and HGO zones with high standardized amplitudes (> 3), high cross-correlation synchronies (> 0.9) and high local focus indices (> 2) were significantly higher in the seizure-free group compared to the not seizure-free group (p = 0.036, p = 0.018, p = 0.026 and p = 0.026, respectively).

**Conclusion:** The automatic quantitative ictal HGO analysis may be effective in delineating the epileptogenic zone. The automatic quantitative ictal HGO analysis may be effective in delineating the epileptogenic zone.

# p059

# Clinical characteristics and prognosis of patients showing periodic lateralized epileptiform discharges

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**Purpose:** To analyze the etiology, clinical characteristics and prognosis of patients showing periodic lateralized epileptiform discharges (PLEDs) and to compare these results with preexisting data.

**Method:** Twenty eight patients were selected by the results of scalp EEG that containing PLEDs. Etiology, occurrence of seizure or status epilepticus (SE), presence of focal neurological deficit and prognosis at discharge were studied in each patient through chart review. PLEDs were defined as focal or hemispheric, periodic epileptiform discharges (spikes, spike and waves, polyspikes, sharp waves) usually occurring every 0.5 to 2 seconds.

**Results:** The most common etiology was anoxic encephalopathy (21.4%), others were cerebral infarction (14.3%), infection of central nervous system (CNS) (10.7%), intracranial hemorrhage (10.7%), toxic-metabolic encephalopathy (7.1%) and postinfectious demyelinating disease (3.5%). The remaining nine patients (32.1%) had no clear etiology. Twenty seven of 28 subjects (96.4%) developed electro-clinical seizures, SE occurred in 14 of 28 patients (50%). The incidence of SE in aspect of each etiology was similar, but all patients with intracranial hemorrhage and toxic-metabolic encephalopathy developed SE. PLEDs were developed during acute stage of illness in 14 of 19 (74%) patients with known etiology and 8 of 28 patients died in the hospital.

**Conclusion:** Anoxic encephalopathy was the most common cause of PLEDs. Most patients developed electro-clinical seizures and SE occurred in half of these patients. The prognosis of patients with PLEDs was poor in almost all patients in this study. The occurrence of SE and prognosis at diacharge were significantly correlated with the cause of PLEDs, respectively.

# Six year old children of women with epilepsy have higher prevalence of epileptiform abnormalities in EEG

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**Purpose:** To study the seizure outcome and EEG patterns in 6 year old children of women with epilepsy (WWE).

**Methods:** All children born to women who were prospectively followed up at Kerala registry of epilepsy and pregnancy (KREP), aged 6-8 yrs were invited .Out of the 254 children who responded, evaluations were complete in 185(female-101,male-84).A 30 min EEG was recorded, including hyperventilation and photic stimulation on 18 channel digital machine.166 EEG'S recorded both sleep & awake state, while sleep could not be obtained in 19(10%).EEG was analyzed by neurophysiologists then attending to the EEG lab and blinded regarding the child's symptomatology.

**Results:** In 185 children aged 6-8yrs of WWE (who had focal epilepsy-81, generalized epilepsy-101), 7(7.3%) children had a history of minimum of one episode of seizure (4- neonatal, 1- febrile seizure, 2- childhood onset single unprovoked seizure) and none had epilepsy. Interictal epileptiform abnormalities (IED) were detected in 37 i.e.20% (19-males & 18-females), in which 30(81%) had focal and 7 (19%) had generalized IED. In 16(43%) out of 37 children, IED's were detected in sleep alone, while 6(16%) had in awake state.Only 2(5.4%) out of 37 children who had IED's had history of seizure. Our study shows a significantly higher prevalence rate than previous studies performed with non-digital EEG.

**Conclusion:** Increased proportion of children of WWE exhibit clinically silent IED's in EEG at 6 years, warranting further regular follow-up.

# p061

# Discrepancy of simultaneous ictal recordings with scalp and subdural electrode arrays in patients with intractable temporal lobe epilepsy

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**Purpose:** The subdural electrocorticography (ECoG) is superior to scalp electroencephalography (EEG) in localizing seizure onset zone because the shielding effect of skull in EEG makes original electrical potentials to ambiguous rhythms over a large area of the cortex. We aimed to demonstrate the time differences of ictal onset rhythms between ECoG and scalp EEG. **Method:** We retrospectively reviewed 25 patients with 115 seizures in intractable temporal lobe epilepsy. We investigated the different ictal recordings on scalp and subdural electrodes, which

were obtained simultaneously during the intracranial EEG monitoring. The ictal onset times and end times between scalp ictal EEG and ictal ECoG were analyzed and compared. The postoperative outcome was assessed by Engel's classification.

**Results:** Mean age was  $27.8 \pm 9.8$  years (range 8 to 45 years), and 56.0% of patients were male. Ictal onset of ECoG started earlier ( $\cdot11.2\pm6.3$  sec) than scalp EEG ( $00.0\pm0.0$  sec, p< 0.001), and duration of seizure recorded by ECoG ( $96.7\pm29.9$  sec) was longer than scalp EEG ( $84.0\pm26.3$  sec, p< 0.001). The time differences between ictal ECoG and scalp EEG was  $5.1\pm7.5$  sec. Of 115 seizures, 9.6% (n=11) showed subclinical seizures, which was demonstrated only by ECoG, not scalp EEG and ECoG. Eighteen patients (73%, 18/25) showed postoperative good outcome (Engel classes I and II).

**Conclusion:** We found that ictal discharges started earlier and lasted longer on ECoG than scalp EEG and only ECoG could detect subclinical seizures. Analysis of ECoG with scalp EEG might provide useful localizing information in the same patient, and ECoG based operation could expect good outcome in patients with intractable temporal lobe epilepsy.

## Mapping interictal high-frequency oscillations in neocortical epilepsy

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**Purpose:** High-frequency oscillations (HFO) are emerged as electrophysiological biomarkers of epileptic brain, frequently recorded in seizure onset zone (SOZ) of mesial temporal lobe (MTL) epilepsy patients. However similar oscillations in neocortical epilepsy are poorly investigated. **Method:** We enrolled 14 patients with ictal origin outside MTL, who underwent intracranial EEG monitoring . At least six hours of interictal data were obtained with sampling rate of 2 kHz. Three 10 minutes of artifact-free epochs were randomly selected during sleep. HFOs were detected using an semi-automated approach. HFO-generating regions were identified and compared with clinically determined SOZ.

**Results:** A total of 1100 bipolar channels were analyzed and 31,235 ripple (R, 60~200 Hz) and 5015 fast ripple (FR, 200~500 Hz) events were detected. FRs were mostly concentrated in SOZ contacts and very rare in non-SOZ. Rs were more broadly distributed, nevertheless their occurrence rate was significantly higher in SOZ for 10/14 patients. Large-scale, simultaneously occurring HFOs were found in 12 patients, suggesting these HFO-generating tissues are pathologically interconnected. Patients with remaining HFO-generating tissue after surgery or incongruent HFO tissue with SOZ tended to have poor surgical outcome.

**Conclusion:** This study shows interictal HFOs are well localized in epileptogenic region in neocortical epilepsy cases. If present, FRs are strong indicator of SOZ in most patients. Rs also discriminated epileptogenic region from control region, suggesting its clinical significance. Presurgical mapping of these fast oscillations and surgical removal of brain regions with high HFO occurrence rate may be important for successful epilepsy surgery.

### p063

#### Occipital paroxysms in dizygotic twins

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**Case report:** We reported 10 years old dizygotic twins with EEG showing occipital paroxysm. The younger one at the age of 4 years 6 months developed seizure 30 minutes after she was falling to sleep, characterized by pallor and confusion next 20 minutes, terminated by vomiting. Few months later 40 minutes after she sleept she woke up pale, complaing of nausea, and gradually became confused. Than she turned her eyes to the right side and became unresponsive next 15 minutes. EEG performed few days after second seizure shown left sided occipital paroxysm.

Valproic acid (VPA) was introduced in daily dose of 500 mg with no recurrence of seizure. Neurological finding and MRI were normal. Psychological testing revealed average IQ. After one year of VPA therapy EEG became normal, and after 2 years Valproate was tappered gradually. During 5  $\frac{1}{2}$  years follow up ( 3  $\frac{1}{2}$  years after discontinuation of VPA), neither occurence

of seizures nor epileptiform abnormality on EEG was noted. The older twin did not develop any seizure, but her EEG also has shown focal epileptifom discharges over left occipital region which were recorded next 2 years in follow up period of 5  $\frac{1}{2}$  years. Her neurological examination and school performance were normal.

**Conclusion:** Benign occipital epilepsy of childhood is very largely genetic in origin, but monozygotic twin pairs would have significantly higher concordance than dizygotic pairs. It seems that focal epileptiform activity which is presented in 1/3 healthy siblings in family with children suffering from benign focal idiopatic epilepsy can explain epileptiform abnormality in healthy twin.

### The role of sleep deprivation on EEG recordings in possible epilepsy patients

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**Purpose:** To determine the important effects of different EEG protocols on the yield of interictal epileptiform discharges in people with possible new epilepsy.

**Method:** A randomized controlled study was conducted. The population target was all possible new epilepsy patients in the outpatient clinic of Dr. Cipto Mangunkusumo Hospital in Jakarta. 44 patients underwent either a non- sleep deprived (NSD) EEG or a sleep deprived (SD) one. **Results:** Out of 44 possible new epilepsy patients who demonstrated interictal epileptiform discharges SD EEG provoked abnormalities in 77,3% while NSD EEG produced 50%. Within the two groups, the most clinical characteristics are with partial seizure type with onset of seizure < 25 years of age, and seizure frequency > once a month. There is a statistical significance between onset of seizure < 25 years of age with the occurrence of IEDs (p = 0.035, RR = 2.41) **Conclusion:** Sleep deprivation increases the yield of interictal epileptiform discharges. The age of onset < 25 years also increases the yield of IEDs with or without SD EEG.

# p065

### Temporal intermittent rhythmic delta activity: prevalence and significance

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**Purpose:** To examine the occurrence and significance of temporal intermittent rhythmic delta activity (TIRDA).

**Method:** Consecutive routine EEG's performed in an adult general hospital were studied and reported using consistent diagnostic criteria.

**Results:** Focal temporal epileptiform abnormalities were identified in 533 of 9426 consecutive routine EEGs (5.6%). Sporadic temporal spikes or sharp waves were present in 492 (5.2%), temporal seizure discharges in 68 (0.7%), temporal PLEDs in 65 (0.7%). TIRDA was the least common in 41 recordings (0. 4%). Nonspecific focal temporal slowing was present in 1002 recordings (10.6%).

TIRDA was usually seen in association with temporal spikes, and when unilateral they were always on the same side. Isolated TIRDA was present in 18 records (15 patients, 0.2%), less common than isolated seizure discharges seen in 24 records.

All patients with TIRDA had clinically definite epileptic seizures, with epilepsy usually being temporal but 4 patients had seizures of extratemporal origin.

**Conclusion:** TIRDA is an accurate marker for partial epilepsy and its lateralisation in general hospital patients undergoing routine EEG. However, in contrast to the commonly seen nonspecific temporal slowing, TIRDA as an isolated finding was present in only 1 per 500 EEGs. TIRDA is better missed than misdiagnosed.

# p066

EEG pattern in seizure free epilepsy patients at Cipto Mangunkusumo Hospital

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**Purpose:** To describe EEG pattern in seizure free epilepsy patients.

**Method:** It was a cross sectional study. Subjects were epilepsy patients that had been seizure free for at least one year with Anti Epileptic Drugs (AED). Subject were recruited from EEG outpatient clinic Cipto Mangunkusumo Hospital from November 2010 to October 2011. Factors such as age at onset, diagnose of epilepsy syndrome, seizure free duration, and the amount of AEDs were collected.

**Results:** There were 60 seizure free epilepsy patients met inclusion criteria, consists of 25 males (41.7%) and 35 females (58.3%) with median age of 23 (2·71) years, 17 patients (28%) with IGE (Idiopathic Generalized Epilepsy) and 43 patients (71.3%) with SGS (Secondary Generalized Seizure). Among the 43 patients with SGS, 15 (34.9%) were diagnosed with TLE (Temporal Lobe

Epilepsy) and the other 28 (65.1%) with ETLE (Extra Temporal Lobe Epilepsy).

The age at onset  $\leq$  18 years old and >18 years old were 66.7% and 33.3%, respectively. The median duration of seizure was 3.75 (0.40) years while the median duration of seizure free period was 2 (1.13) years. Patients who had been receive monotherapy and polytherapy were 83.3% and 15%, respectively.

EEG was normal in 35 subjects (58.3%) and abnormal in 25 subjects (41.7%). Among subjects that had abnormal EEG, 92% had epileptic discharges in EEG pattern. There was no significant difference in terms of epileptic syndrome (p=0.844), age at onset (p=0.713), seizure free duration (p=0.725) and amount of AED (p=0.844) towards the EEG results.

**Conclusion:** Epileptic discharges still persist in seizure free epileptic patients. There was no relationship between EEG pattern and seizure free.

### Epidemiology

# p067

Epidemiological study of epilepsy in Zabaykalyie

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**Purpose:** To study epilepsy prevalence Asian and European population of rural district of Eastern Siberia (Russia).

**Methods:** Research was conducted by the method of door-to-door survey in a rural district of Zabaykalyie (Eastern Siberia). Researchers screened 92% of the population aged 14 years and older (middle age - 34.23±3.36 ye.o.). Aginsky district is the rural district in which two populations (Asian and European) live in neigboorhood. The Asian population includes 15991 persons (62.6% of all population), 98% of them are Buryats. Research included questioning of respondents about presence of epileptic seizures and AEDs. Patients with active epilepsy underwent neurologic examination, EEG, MRI. Final diagnoses were based on the Classification of epileptic seizures and syndromes (ILAE, 1989).

**Results:** Among 22.545 residents surveyed, 136 persons with epilepsy were identified, 78 of them having active epilepsy. Focal epilepsies was 87%. Symptomatic epilepsy (68%) etiology: brain trauma (38%), neuroinfections (16%), perinatal brain damage (12%), stroke (2%). Idiopathic epilepsy was 11%, cryptogenic epilepsy - 18%. Epilepsy prevalence was 6.03/1000, active epilepsy - 3.46/1000. Standardized epilepsy prevalence among Asians was 3.5/1000 vs 2.0/1000 among Europeans (p < 0.05). Epilepsy prevalence was more in 20-29 years (5.20/1000) and 50-59 years (5.42/1000). The therapy: 65.8 % of patients received one AED, 31.6% - 2 AEDs, and 2.6% - 3 AEDs.

**Conclusion:** Epilepsy prevalence in the rural district of Zabaykalyie was comparable to epilepsy prevalence in the Western Europe. Causes of high level of epilepsy prevalence among the Asian population (Buryats) requires further researches.

# p068

Toxocara and neurocysticercosis - risk factors for epilepsy: a community based study

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**Purpose:** This study has been planned to determine the association between epilepsy and exposure to toxocara and T solium in North Indian population.

**Method:** Door-to-door screening survey was done by trained field workers who administered prevalidated questionnaire in an urban slum area to identify individuals who had an epileptic seizure. These identified patients were investigated (detailed history and examination, EEG, MRI)

and diagnosis confirmed by a neurologist. The blood samples were drawn for Anti-Toxocara specific IgG (ELISA); further confirmed by immunoblot and antibodies against T.solium (EITB assay)both in cases and age and gender matched controls.

**Results:** A population of 15,750 was screened and 114 (males 69; females 45) were detected to be having epilepsy. The mean age was  $26.85\pm16.36$  years with mean duration of epilepsy of  $111.35\pm107.54$  months. The Case and Control positive rate for Anti-Toxocara specific IgG were 6.60% and 7.89% respectively (p=0.71). Among seven ELISA positive cases five were confirmed positive by immunoblot (4.72%) and of nine ELISA positive controls seven were confirmed positive rates for antibodies by EITB assay for cases and controls were 25.47 % and 11.40 % respectively (p=0.01).

**Conclusion:** NCC (16.7%) and substance abuse (10.5%) were the most common etiologies found in this community. Both these causes can be are prevented by health education and improvement in sanitation and reduce the burden of epilepsy.

#### p069

#### Seizure free in temporal lobe epilepsy patient Cipto Mangunkusumo Hospital June 2010 - May 2011

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**Purpose:** To know the seizure free rate after 2 years of TLE therapy in Cipto Mangunkusumo Hospital. **Method:** A cross sectional descriptive at Neurology Department Cipto Mangunkusumo Hospital on June 2010 · May 2011. Subjects were people with TLE based on semiology and EEG recordings. Inclusion criteria was subject been through at least 2 year of epilepsy therapy. Seizure free was defined as no seizure at least more than 12 months by monotherapy or politherapy.

**Results:** Sixty three subjects are included in the study, 31.7% are seizure free more than 12 months, 68.3% non-seizure free or free seizure less than 12 months. Seventy nine point one percent subjects in non-seizure free group and eighty percent subjects in seizure free group use monotherapy. Twenty point nine percent subjects in non-seizure free group use polytherapy. Most monotherpy drugs used are carbamazepine (27%), phenitoin (22.2%), phenobarbital (12.7%) dan valproate (11.1%). Seventy point four percent subjects using valproate free from seizure more than 12 months. The most frequent combination used in polytherapy were carbamazepine.phenitoin (4.7%).

**Conclusion:** Seizure free frequency of TLE patient is 31.7% and 80% among them use monotherapy (mostly carbamazepine). Neither monotherapy or polytherapy were significant in seizure or seizure free groups.

# Genetics

### p070

### Genetic and epileptic features in Rett syndrome

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**Purpose:** Rett syndrome is a severe neurodevelopmental disorder in females. Most have mutations in the methyl-CpG-binding protein 2 (MECP2) gene (80·90%). Epilepsy is a significant feature in Rett syndrome and commonly accompanied. Our study is aimed at comprehensive analysis of genetic and clinical features in Rett syndrome patients especially in epileptic features. **Method:** We retrospectively reviewed 20 patients who were diagnosed with MECP2 mutations at Severance Children's Hospital between January 1995 and July 2010. All patients met clinical criteria for Rett syndrome. Evaluations included clinical features, epilepsy classification, EEG analysis, and treatment of seizures.

**Results:** Ages ranged from 3.6 to 14.3 years (7.7  $\pm$  2.6). Fourteen different types of MECP2

mutations were found, in which a novel in-frame mutation (1153-1188 del36) was included. Fourteen (70.0%) had epilepsy, and average age of seizure onset was  $3.0 \pm 1.8$  years. Epilepsy was diverse, including partial seizure in four patients (28.5%), secondarily generalized seizure in six (42.8%), generalized tonic seizure in two (14.3%), Lennox-Gastaut syndrome in one (7.1%), and myoclonic status in non-progressive encephalopathy in one (7.1%). Motor functions were delayed so that only ten patients (50.0%) could walk independently: five (35.8%) in the epilepsy group and five (83.3%) in the non-epilepsy group. Average developmental scale was  $33.5 \pm 32.8$  in epilepsy group and  $44.4 \pm 21.2$  in non-epilepsy group. A clear genotype-phenotype correlation was not found.

**Conclusion:** There is a tendency that motor impairment and cognitive deterioration are more serious in epilepsy group.

#### p071

# Pharmacogenetic testing of antiepileptic therapy: a pilot study

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**Purpose:** Assessment of role of CYP2C9 gene polymorphisms as unmodified risk factor of ADRs development in case of intake of therapeutic doses of VPA drugs at patients with epilepsy. **Method:** Sampling included 124 patients with epilepsy (age - from 1 till 59 years, median - 21 years). We used analysis of doses of VPA preparations, therapeutic drug monitoring of VPA level in serum, biochemical blood analyses (level of aspartate aminotransferase, alanine aminotransferase, bilirubin, amylase, complete albumen), illustrating functional activity of liver; video-EEG-monitoring; pharmacogenetic testing of SNPs of gene CYP2C9 (chromosome10q24.1-24.3): wild-type allele variant CYP2C9\*1/\*1 without mutation, mutant-type allele variants (CYP2C9\*2, 430 C>T; CYP2C9\*3,1075 A>C).

**Results:** The frequency of polymorphous allelic variants gene CYP2C9: CYP2C9\*1/\*1 was 64.52% (80/124), mutant polymorphous allelic variants was 35.48% (44/124). The carrier of allele variant CYP2C9\*1/\*1 had 47.50% (38/80) occurrence of ADRs in case of standard usage of VPA drug dosage (kg/ body weight per daily). The carriers of polymorphous allelic variants CYP2C9\*3, both homozygous and heterozygous carriers and also in case of their combination had 72.72% (32/44) occurrence of ADRs in case of VPA drug dosage.

**Conclusion:** The polymorphous allelic variants gene CYP2C9 could be used as markers for optimization of the drug therapy of epilepsy. The personalized approach to VPA drugs dosing in treatment of people suffering from epilepsy is clinically grounded. This approach allows prevent overdose as a cause of ADRs development, and thus improves life quality of patients with epilepsy.

### p072

#### Advances in the molecular genetics of the benign epilepsies of infancy

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**Purpose:** The benign epilepsies of infancy are a group of three autosomal dominant disorders in which similar seizure types occur. The disorders differ in their average ages of onset and offset. Benign familial neonatal epilepsy is caused by mutations in the genes KCNQ2, KCNQ3 while benign familial neonatal-infantile epilepsy is caused by mutations in SCN2A. To determine the molecular basis of benign familial infantile epilepsy (BFIE), we sequenced genes in the chromosome 16p11.2-q12.1 region to which many families have been linked.

**Method:** Ten probands from BFIE families with confirmed or suspected linkage to chromosome 16 were sequenced for the coding regions of the candidate genes. An additional 13 probands were sequenced for the gene thus identified. Family members and controls were tested by sequencing or high resolution melting analysis.

**Results:** Mutations in the gene PRRT2 were identified in 19 out of 23 families with BFIE. All mutations segregated with the phenotype in families and were not observed in controls. Five different mutations were identified, including a recurrent insertion present in 15 families.

**Conclusion:** Mutations in PRRT2 are the major molecular cause of BFIE. The gene codes for a protein of unknown function and represents a new gene family involved in the pathogenesis of epilepsy. This finding resolves a long-standing question in epilepsy genetics, as the chromosome 16 BFIE and ICCA locus has been known for many years without the gene being identified. The molecular basis of a large proportion of cases of benign neonatal and infantile epilepsy is now known.

#### p073

### CAMSAP1L1 is a potential epilepsy gene in the Chinese population

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**Purpose:** In a genome-wide association study of Chinese epilepsy and control subjects, we found association of the CAMSAP1L1 gene with epilepsy. Because very little is known about the protein, we sought to explore its localization and function.

**Method:** We performed Western blotting of homogenates of human and mouse brain and human neuroblastoma SH-SY5Y cells, and immunohistochemistry of fixed brain sections and SH-SY5Y cells.

**Results:** Two antibodies (161A and 162A) map to different epitopes of human CAMSAP1L1 and stained a band at the expected molecular weight of approximately 170 kDa in humans. Only 162A also stained this band in mouse samples, as predicted by conservation of the epitope sequences in mice. CAMSAP1L1 immunoreactivity appeared in neurons in human hippocampal sections and in the cytosol and neurites of SH-SY5Y cells. Proteins of the CAMSAP family have a conserved C-terminal region called the CKK domain, which is predicted to bind to microtubules and may block microtubule function to inhibit neurite outgrowth. Double immunofluorescence staining in SH-SY5Y cells showed partial colocalization of CAMSAP1L1 with class III beta-tubulin.

**Conclusion:** A genetic variant of CAMSAP1L1 may be associated with epilepsy. CAMSAP1L1 is expressed in neurons and may bind microtubules, suggesting a potential role in epileptogenesis via an effect on the extension of neurites.

### Neurobiology

# p074

# System administration of neuregulin- $\beta 1$ inhibits epileptogenesis in pilocarpine induced epilepsy rat model

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**Purpose:** Neuregulin/erbB4 signal pathway enhances depolarization-induced GABA release, however, its role in epilepsy is unknown. Neuregulin 1, one member of epidermal growth factor family, can promote activity-induced GABA release through its cognate receptor erbB4 expressed in the interneuron. Here, we aim to explore the role of neuregulin/erbB4 signal pathway in epilepsy.

**Method:** Pilocarpine-induced epilepsy rat model was used. 1) The expression of neuregulin and the level of erbB4 phosphorylation in the hippocampus and cerebral cortex of epileptic rats were assayed by western-blot. 2) Neuregulin-B1, a 62 amines peptide, which could mimic the function of neuregulin protein, was system administrated intraperitoneally 30 minutes before intraperitoneal administration of pilocarpine. The behaviors of epileptic rats were observed according to the method of Racine RJ. The level of erbB4 phosphorylation in the hippocampus and cerebral cortex of epileptic rats was assayed by western-blot.

**Results:** 1) In pilocarpine-induced epilepsy rat model, neuregulin was up-regulated correlated well with erbB4 phosphorylation in the hippocampus and cerebral cortex. 2) System administration of neuregulin- $\beta$ 1 could inhibit epileptogensis and promote the phosphorylation of erbB4 in the hippocampus and cerebral cortex of epileptic rats in a dose-dependent manner.

**Conclusion:** The study indicates that neuregulin/erbB4 signal pathway negatively regulates epilepsy. The study identifies a novel signal pathway involved in epileptogensis, and may provide a new strategy for epilepsy.

## p075

# Attenuation of kainic acid-induced neurotoxicity by an antioxidant in organotypic hippocampal slice culture

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**Purpose:** Kainic acid (KA) has been used to study the mechanisms of status epilepticus-induced neuronal damage and epileptogenesis since KA treatment of organotypic hippocampal slice culture (OHSC) induces region-specific neuronal death and reorganization of hippocampal circuitry. The present study investigated the neuroprotective effects of lipoic acid, an antioxidant, against oxidative stress induced by KA in OHSC of rats.

Method: Cultured slices were injured by exposure to 5  $\mu\text{M}$  KA for 18 hr and then treated with different concentrations of lipoic acid.

**Results:** Neuronal cell death measured as propidium iodide uptake was reduced at 24 hr after lipoic acid treatment. We also observed an increased number of surviving CA3 neurons in the lipoic acid-treated groups using cresyl violet staining. Lipoic acid treatment significantly decreased the 2',7'-dichlorofluorescein fluorescence and the expression of NQO1 in the lipoic acid-treated groups was significantly lower than that in the KA only group.

**Conclusion:** These results suggest that lipoic acid may protect hippocampal neurons against oxidative stress.

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#### Neuroimaging

#### p076

A study on glucose hypometabolism sites and electrophysiological changes from 18F-FDG-PET in patients with temporal lobe epilepsy

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**Purpose:** Accurate identification of the epileptic focus on preoperative tests is essential for achieving a favorable seizure outcome following epilepsy surgery. 18F-fluorodeoxyglucose PET (FDG-PET) has been reported to be a useful preoperative test for localizing the epileptic focus in intractable epilepsy. However, the question of whether glucose hypometabolism sites in FDG-PET accurately reflect the distribution of interictal epileptic discharges (IEDs) or indicate histological changes remains a topic of debate even today.

We created a normal database and used a statistical analysis to study how glucose hypometabolism sites in pre-operative FDG-PET correlate with IEDs and pathological changes in temporal lobe epilepsy. **Method:** Subjects were 31 patients who underwent unilateral hippocampectomy after being

diagnosed with temporal lobe epilepsy, and were divided based on MRI results into a group in which hippocampal sclerosis was present (n=14; five men and nine women; mean age, 30 years) and a group in which hippocampal sclerosis was not identified (n=17; 13 men and four women; mean age, 42 years) for comparison.

**Results:** No statistical correlation was found between the number of ROIs of hypometabolism sites in diseases having an epileptic focus and the number of spikes in intraoperative EEG findings. However, we did find a positive correlation between the number of ROIs and the pathological findings by Watson class, and a negative correlation (correlation coefficient: -0.465; significance probability: 0.013) between the number of ROIs and an evaluation of the postoperative prognosis by Engel class. The group with Engel class I had a significantly higher number of hippocampal ROIs than the II to IV groups (P< 0.01).

**Conclusion:** It seems that glucose hypometabolism sites indicated by FDG-PET are a reflection of pathological change that enable confirmation of lesions on MRI, and that surgery has the potential to provide a favorable prognosis in patients with temporal lobe epilepsy having a clear confirmation of glucose hypometabolism at these sites by FDG-PET.

# p077

### Crossed cerebellar abnormality in three patients with refractory partial seizures

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**Purpose:** Crossed cerebellar diaschisis (CCD), defined as a reduction in metabolism and blood flow in the cerebellar hemisphere contralateral to a supratentorial cerebral lesion, which was mainly reported in patients with stroke and other destructive lesions. The current study was to assess the CCD and crossed cerebellar atrophy (CCA) in patients with epilepsy.

**Method:** Crossed cerebellar volume in patients with partial seizure was evaluated systemically in China-Japan friendship hospital during 2010-2011. CCD was assessed in patients underwent 18F-FDG PET. While, CCA was defined as prominent cortical sulci with parenchymal volume loss of the cerebellum, which is asymmetric when compared with the contralateral side in MRI axial, coronal sequences.

**Results:** Three patients were identified with CCD or CCA. Patient 1 was detected with diffused lesion in right temporal lobe. Furthermore, epileptogenicity was confirmed by neurophysiological investigation, and PET showed obvious CCD. This patient was operated for refractory seizure and focal cortical dysplasia (CD) was revealed histologically. While, patent 2 with the radiological feature of FCD on left occipital lesion demonstrated severe CCA. Similarly, patient 3 diagnosed as hemicovulsion-hemiplesia epilepsy, also showed significant CCA.

**Conclusion:** CCD was thought as the result of interruption of afferent corticopontocerebellar pathways which resulted in decreased blood flow and metabolism in the contralateral cerebellar hemisphere. However, the current observation revealed cerebrum involved in the epilepsy and the over excitatory also led to CCD and even CCA. Alternatively, it is suggested cerebellum could be as the potential target to modulate the excitatory of cortex.

### p078

# Hypertrophic amygdala in three patients with medial temporal lobe epilepsy

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**Purpose:** Medial temporal lobe epilepsy (MTLE) was of special interest because of its high prevalence. Although hippocampal sclerosis (HS) was the most frequently pathological findings in MTLE, the role of amygdala remained uncertain. Herein, we present three patients with hypertrophic amygdala (HA) to depict the clinico-radiological characteristics.

**Method:** We assessed the amygdalar volume and signal alteration in patients with TLE in China-Japan friendship hospital during Sep 2010 to Oct 2011. Enlargement of amygdalar volume with significant asymmetry in MRI coronal and axial sequences, together with increased signal on FLAIR sequences was considered as HA. Subsequently, clinical data, semiology, EEG, MRI were investigated comprehensively.

**Result:** Three patients including one male and two female were identified with distinct HA. The seizure onset ages were 13, 12 and 5 years old, respectively. Ictal fear was reported as the habitual aura in patient 1 and 3, followed by unresponsiveness, oroalimentary, and repetitive hands automatism. Patient 2 manifested as complex partial seizure without obvious aura. Interictal EEG showed epileptiform discharges, and ictal rhythmic discharges with 5-7Hz initially were detected on unilateral anterior temporal lobe in all three patients. Radiologically, ipsilateral HA were recognized, while hippocampus kept intact. Finally, patient 1 was operated because of refractory seizures, and patient experienced seizure free in follow up.

**Conclusion:** Concerning clinical features and radiological findings, patients with HA might represent a discrete subtype of MTLE.

#### p079

# Resting-state fMRI study of catamenial epilepsy patients with correlated changes of ${\sf E}/{\sf P}$ serum ratio concentration

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**Purpose:** To investigate the different characterize regional brain activation in catamenial epilepsy patients with C1 pattern, comparing them to themselives in different menstrual period phases and also to the random phase of healthy controls.

**Method:** Twelve catamenial epilepsy patients with C1 pattern and 6 non-epileptic controls were studied by using the a gradient-echo echo-planar imaging (EPI) sequence on a Trio Tim (3T) magnetic resonance (MR) imaging system which once in the early Menstrual phase(M phase) and the second in the ovulatory phase(O phase ) of the menstrual cycle. Simultaneous sexual hormones was taken for evaluating the E/P ratios and correlated to the brain function. Amplitude of low-frequency (0.01-0.8Hz) fluctuations (ALFF) of the blood oxygenation level-dependent (BOLD) signal, which is thought to reflect spontaneous neural activity, was used to characterize regional functional alteration. The data was processed and analyzed using REST and SPSS11.5 software.

**Results:** The patients with catamenial epilepsy showed significantly higher activity in the left occipital lobe, left temporal gyrus, parahippocampal gyrus, amygdala, as compared to the other groups (p < 0.005, corrected). The brain network connectivity of the catamenial epilepsy patients within the prefrontal- system also was decreased in patients with lower E/P ratios (p < 0.005, corrected).

**Conclusion:** The catamenial epilepsy patients with C1 Pattern have a characteristic alteration pattern and disruption of the brain network at the onset of seizure. The low E/P ratios correlated with regional brain activities. The present study offers further insight into the underlying neuropathophysiology of the catamenial epilepsy and the effect of the E/P ratio.

#### p080

# Longitudinal assessment of brain function on patients with temporal lobe epilepsy after anterior temporal lobectomy: a resting state fMRI study

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**Purpose:** The purpose of this study is to evaluate the course of brain funtional change during 3 months after left anterior temporal lobectomy on patients with temporal lobe epilepsy (TLE) by using resting state functional MRI (rfMRI).

**Method:** In 7 left TLE patients who underwent left anterior temporal lobectomy, resting state fMRI was performed before surgery, and 3 months after surgery using a gradient-echo echo-planar imaging (EPI) sequence on a 3T MR imaging system (EXCITE, General Electric, Milwaukee, USA). Amplitude of low-frequency (0.01-0.8 Hz) fluctuations (ALFF) of the blood oxygenation level-dependent (BOLD) signal, which is thought to reflect spontaneous neural activity, was used to characterize regional functional alteration. The amplitude of LFF (ALFF) was calculated using REST

software. Voxel based analysis of the ALFF maps between baseline and 3 months after surgery was performed with two sample t test by using the SPM8 (P < 0.05, FWE corrected).

**Results:** Repeated rfMRI demonstrated a significant increased ALFF value in the left parahippocampus and contralateral insula lobe 3 months after surgery. No reduced ALFF was observed. **Conclusion:** Our study provides the neuroimaging evidence of brain function alteration pattern in TLE patients after surgery. The increased brain function regions may played the role of compensatory of the resection, which prompted the compensatory mechanism of the damaged regions. Functional MRI could be a sensitive method in monitoring the clinical outcome of surgery and can help further investigation of the underlying mechanism.

## p081

# Inter-ictal FDG PET in temporal lobe epilepsy. Correlation with ictal EEG, MR imaging, neuropsychology and outcome

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**Purpose:** To identify inter-ictal FDGPET patterns and to correlate with ictal EEG, ictal SPECT, MRI, and surgical outcome in patients with refractory temporal lobe epilepsy (TLE).

**Method:** Retrospective analysis of inter-ictal FDGPET, Ictal EEG, Ictal SPECT and MRI data was performed in 88 patients with refractory TLE and one year follow up after surgery. Ictal SPECT was performed in 65.

**Results:** Mean follow up was 32 (12-62) months; 55% were males; Mean age at surgery was 26.5 (5-52) years. Pathologically verified hippocampal sclerosis (HS) was found in 68; Favorable outcome (Engel's Class I&II) was noted in 81.81%. PET showed unilateral hypometabolism in 82, bilateral in 4 and contralateral in 2, while extended hypometabolism was noted in 7 patients. PET correlated with MRI in 98.6%, ictal EEG in 86.8% and neuropsychology in 80.7%. The sensitivity of ictal SPECT & inter-ictal PET was 93% and 93% and specificity 97% and 88% respectively. Ictal EEG onset was discordant/uncertain in 10 (11.3%) & surgery was done after concordant ictal SPECT and interictal PET. MRI was normal in 16 and surgery was done after concordant ictal EEG, ictal SPECT & interictal PET; 9 (56%) had favorable outcome. After multiple regression analysis using the McHenry's algorithm, abnormal imaging, unilateral interictal spikes, ipsilateral PET Hypometabolosm and typical ictal SPECT and Inter-ictal FDGPET is specific and sensitive non-invasive modality during pre-surgical evaluation of patients with refractory TLE. Ictal SPECT and Inter-ictal FDGPET are complimentary, together obviate the need for invasive EEG in resource poor countries.

### p082

# Characteristics of altered brain function in epilepsy patients with different seizure type: evidence from resting-state fMRI

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**Purpose:** To investigate the different brain function pattern in epilepsy patients with different seizure type.

**Method:** We recruited 18 epilepsy patients with complex partial seizure (CPS), 21 patients with complex partial seizure and secondary generalized tonic clonic seizure(CPS·SGTCS), and 22 patients with idiopathic generalized tonic clonic seizure(GTCS). All subjects were scanned using a gradient-echo echo-planar imaging (EPI) sequence on a 3T MR imaging system (EXCITE, General Electric, Milwaukee, USA). Amplitude of low-frequency (0.01-0.8 Hz) fluctuations (ALFF) of the blood oxygenation level-dependent (BOLD) signal, which is thought to reflect spontaneous neural activity, was used to characterize regional functional alteration. The amplitude of LFF (ALFF) was calculated using REST software. Voxel based analysis of the ALFF maps between each patient group were performed with ANOVA using the SPM8 (P < 0.05, FWE corrected).

Results: ALFF values in bilateral superior longitudinal fasciculus (SLF), predominantly in the left

SLF area, were increased sequentially from CPS, CPS-SGTCS to GTCS group. Meanwhile, compared with GTCS group, decreased ALFF value in cingulated cortex was observed in CPS-SGCTS group. **Conclusion:** Our result demonstrated the different brain function pattern in epilepsy patients with variant seizure type, characterized by hyperactivity in specific brain network. This alteration brain function may be related with brain preparation for the seizure propagation.

# p083

# Directed transfer function analysis on generalized spike-and-wave discharge of juvenile myoclonic epilepsy

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**Purpose:** Conventionally, generalized idiopathic epilepsy (IGE) was explained using corticothalamic network. However, some researches show that intracortical relation takes an important role of IGE. Hence, in this paper, we studied causal relations among selected cortical regions by analyzing generalized spike and wave discharge (GSWD) which is a characteristic of IGE.

**Method:** GSWDs were collected from 3 patients with juvenile myoclonic epilepsy (JME) using 58 or 62 electrodes. By visual inspection, epochs including GSWD are extracted. Then, using boundary element method for forward solver and minimum norm estimation for inverse problem, current source distribution and its propagation on cortical surface are acquired. Based on these results, 5 regions including temporal, dorsolateral prefrontal, frontal, central, and occipital areas were selected for directed transfer function (DTF). To analyze time-varying causal relationship, moving window DTF was adopted.

**Results:** The result of inverse analysis shows some different pattern of propagation such as temporal-dorsolateral prefrontal-frontal-central, frontal-central, and frontal-frontotemporal-inferior temporal-inferior temporal-temporoccipital (or occipital) patterns. Although these topographic results show intrapatient and interpatient differences, wave forms of selected regions show common characteristics such that phase difference between occipital and other selected region is about 90 degrees. We divided a GSWD into small windows to detect causal relation using moving window DTF. Even though amplitude of occipital activation is small, moving window DTF shows that outflow from occipital lobe was dominant in the window which occipital activation decreased and other activation increased.

**Conclusion:** Interregional relationship of cortex was studied using inverse analysis and DTF. Characteristic of IGE can be defined using wave form of cortical sources and DTF results imply that occipital region takes an important role of GSWD (or IGE).

# p084

# Is SISCOM (Subtraction Ictal SPECT Co-registered to MRI) helpful to the successful surgery in nonlesional extratemporal epilepsy?

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**Purpose:** Nonlesional extratemporal seizures are challenging to plan respective epilepsy surgery. Surgical outcomes have been reported to be worse than those for temporal lobe epilepsy. The aim of this study was to evaluate the overall surgical outcomes of nonlesional extratemporal epilepsy and to determine the usefulness of SISCOM (Subtraction Ictal SPECT Co-registered to MRI) in the prediction of successful postsurgical outcome.

**Methods:** From January 2007 to April 2010 at Samsung Epilepsy Clinic in Samsung Medical Hospital, 25 patients with nonlesional extratemporal lobe seizures (17 female) were enrolled who had undergone comprehensive presurgical evaluation and subsequent intracranial EEG monitoring for epilepsy surgery (a postoperative period of ≥ 1 year). Their clinical data including SISCOM analysis were all reviewed and postsurgical outcome was determined using Engel classification. **Results:** Mean age at surgery was 26.8 years, mean disease duration before surgery was 13.7 years, and mean follow up was 35.9 months. There were 12 Frontal lobe epilepsy, 6 Temporo-parietal lobe epilepsy, 4 Parietal lobe epilepsy, and 3 Occipital lobe epilepsy. In 12 cases, hyperperfused areas in SISCOM were congruent to surgical resection margin, and were noncongruent in 13 cases. Hypometabolic areas in FDG-PET were congruent in 11, and were non congruent in 14 cases.

Post surgically 10 (40%) were seizure free (Engel class 1A), and 14 (60%) were not seizure free. Pathology was all focal cortical dysplasia.

Seizure free outcome was predicted 6 of 11 (54.5%) in congruent SISCOM, but only 4 out of 11 (36.4%) in noncongruent SISCOM.

**Conclusion:** Our results suggest that seizure free surgical outcome were seen in 40% of cases in nonlesional extratemporal epilepsy.

It seems that SISCOM analysis is helpful to replace limited role of MRI as image study for nonlesional extratemporal epilepsy.

#### p085

# Cerebral activations during Chinese verbal memory processing in temporal lobe epilepsy: a functional magnetic resonance imaging study

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**Purpose:** We used functional magnetic resonance imaging (fMRI) recordings to clarify the cerebral correlates of Chinese verbal memory processing in temporal lobe epilepsy (TLE) patients and agematched healthy volunteers.

**Method:** Our memory task contained 120 concrete words in memory encoding phase, and then these 120 old words were intermixed with 60 new words for subsequent memory recognition. We evaluated the effect of epilepsy on verbal memory and determined the BOLD change in the mesial temporal lobe with respect to the side of main epileptic focus.

**Results:** A frontal-temporal network was clearly involved in verbal memory-related processing in patients and controls, but the activation in the superior temporal and inferior parietal regions was smaller in patients. Moreover, the bilateral hippocampal activation was decreased in patients. Compared with right TLE patients, left TLE patients exhibited a decreased activation in the inferior parietal area, medial frontal regions, and left hippocampus.

**Conclusion:** The present fMRI study suggests that the cerebral activation during verbal memory processing is relatively impaired in left TLE than in right TLE patients.

# p086

#### Verbal memory function in temporal lobe epilepsy: a MEG study

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**Purpose:** Currently, the standard tool used to determine hemispheric language and memory dominance is the Wada test, and it has been considered the gold standard for pre-surgical planning. However, Wada test is an invasive procedure and has potential risk. In this study, we want to develop an MEG task to evaluate functional lateralization of verbal memory and language in Chinese to replace Wada test.

**Method:** Eight healthy right-handed subjects (4 males and 4 females, mean age 25.7) and four patients(3 males and 1 females, mean age 25.5) were recruited in our study. We developed Chinese word recognition task to identify memory-related neural circuitry with MEG. The task contained 120 concrete words in memory encoding phase, and these 120 old words were then intermixed with 60 new words for subsequent memory recognition. Minimum current estimate and sequential dipole modeling are used to analyze MEG response in both phases. Averaged MEG data was displayed on a

magnetic contour map from 150 to 1000 ms for finding dipole field by visual inspection. **Results:** Our task elicited neural response in mesial temporal lobe in both healthy subject and patient groups. Our data also showed that verbal memory processing is left hemisphere dominant in healthy subjects, and seems contra-lateral hemisphere dominant to lesions in patients. **Conclusion:** Our task elicited neural response in mesial temporal lobe in both healthy subject and patient groups. Our data also showed that verbal memory processing is left hemisphere dominant in healthy subjects, and seems contra-lateral hemisphere dominant to lesions in patients.

### Neuropsychology

#### p087

#### The impact of epilepsy on the subjective well-being in men following neurosurgery RAFFAELE MP

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**Purpose:** This study provides a framework for understanding the subjective well-being in men with adult onset epileptic seizures post elective neurosurgery. How the participants may be perceived in all social environments and participant's self-regulation will be discussed.

**Method:** As each participant is different and each case will not represent the same finding, a multiple case study research structure was chosen. The qualitative Interpretive Phenomenological Approach was chosen to explore in detail how each participant made sense of their personal and social environments. In-depth interviews, focus groups and document analysis will be applied to gain all the information of their Subjective Well-Being along with the relationship between neurosurgery and the psychological effects that this might create when these men are re-establishing themselves.

**Results:** The results from previous studies and review literature have said that a lack of psychoeducation is a factor that negatively influences participants when re-establishing themselves in all relevant social environments. However, this study focuses on their Subjective Well-being in men with Epilepsy after neurosurgery, which might have similar or different factors from those previous ones. **Conclusion:** It can be assumed that rehabilitation is most important for attaining positive Subjective Well-Being post-surgery. Psycho-education influences higher Subjective Well-Being, as confidence arises through knowledge. All relevant social environments must work together to ensure that psychological stability is attained and maintained for patients with epilepsy post-surgery. Without psycho-education, life's cycle of adaptive change runs a far more restricted path.

# p088

# Comparison between prospective and retrospective memory failures in epilepsy patients: preliminary report

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**Purpose:** Many epilepsy patients suffer from cognitive impairments, including memory disturbance. Memory disturbance is one of the most critical cognitive impairments that epilepsy patients experience. Memory can be categorized into prospective memory (PM) and retrospective memory (RM), and PM failures disrupt everyday function and independent life. Most previous studies on memory, nonetheless, have focused on RM, and only few examined PM. This preliminary report of the present study compared epilepsy patients' subjective PM and RM failures.

**Method:** The present study compared epilepsy patients' subjective PM and RM failures using a questionnaire. Patients who visited or admitted to a neurology department of a university affiliated hospital in Korea were asked to answer a prospective retrospective memory questionnaire (PRMQ) developed by Smith et al. (2000). There were 7 male (18 female) patients, and their average age was 51.88. In order to see if memory type (PM vs. RM) and memory cue (self-initiated vs. environmental) affect epilepsy patients' subjective memory failures, a 2-way within-subject ANOVA was performed.

**Results:** The analysis revealed that the interaction between memory type and memory cue was significant. This pattern of results suggests differential effects of memory cue in the PM and RM

conditions. In other words, subjective memory failures were not affected by memory cue in the PM condition, whereas they were more impaired with environmental cues than with self-initiated cues in the RM condition.

**Conclusion:** The present study examined memory of epilepsy patients in terms of relative impairment of subjective PM and RM. Epilepsy patients were not affected by memory cue in the PM condition, but were affected in the RM condition.

## p089

### Parenting stress, attitude and quality of life in children with epilepsy

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**Purpose:** This study examined the characteristics and relationship between parental stress, attitude and quality of life in children with epilepsy.

**Method:** 43 epilepsy children (boys=21, girls=22), aged 5 to 13 years, were administered Intelligence test, depression, anxiety, and quality of life. Parental attitude and stress were assessed by means of questionnaires.

**Results:** About one thirds of Children with epilepsy within normal intelligence showed high depression and anxiety. Their parents had also generally high parenting stress, particularly on parenting competency, attachment, reinforcement, isolation, health, and children's acceptability. On parenting attitude, parents of epilepsy children showed low supportive expression and rational explanation, meanwhile high level of involvement, punishment, superintendence, expectation, and inconsequence. As a result, more depressive mothers were significantly related with higher involvement parenting attitude and lower satisfaction level on general behavior and memory in cognitive function of quality of life in epilepsy children. In the same context, higher parenting stress on general characteristics of epilepsy children showed significant relationship with higher level of parents' superintendence and also lower satisfaction level of health and memory in epilepsy children's quality of life. On the other hand, supportive expression and rational explanation of parenting attitude had positively significant relationship with self-esteem and cognitive function of epilepsy children.

**Conclusion:** Parenting stress and attitude seemed to be conjunctly connected with quality of life in children with epilepsy in addition to seizure-related variables, including on cognition, behavior, mood and health. Comprehensive and well-guided parent education programs for parents with epilepsy children were suggested to increase their quality of life along with managing epilepsy.

# p090

#### Korean version of the neurological disorders depression inventory for epilepsy (NDDI-E) KO PW<sup>1</sup>, LIM HW<sup>1</sup>, KIM SH<sup>2</sup>, PARK SP<sup>1</sup>

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**Purpose:** Depression is the major comorbid disorder in people with epilepsy(PWE). However, it is relatively underdiagnosed and undertreated comorbid by clinicians due to a busy clinical setting. The purpose of this research was to evaluate the Korean version of the Neurological Disorders Depression Inventory for epilepsy (NDDI-E) which is developed for the rapid and objective assessment of depression.

**Method:** This study involved 121 outpatients who underwent neuropsychologic evaluation and clinical interview. All subjects completed reliable and validated self-reported heath questionnaires, including Beck Depression Inventory (BDI) and Mini International Neuropsychiatric Interview-Plus Version(MINI-Plus). They also performed a Korean version of the NDDI-E.

**Results:** The NDDI-E were confirmed as being easy to understand and without any other problems. At a cutoff score  $\geq$  11, NDDI-E had a sensitivity of 92.3%, a specificity of 80.0%. Cronbach's  $\alpha$  for the Korean version NDDI-E was 0.906 that indicates reasonable internal consistency reliability, and
there was also a positive correlation between the NDDI-E and the BDI (p< 0.001). **Conclusion:** The use of the NDDI-E allowed us to enhance our outpatient evaluation by improving the identification of depression more rapidly and accurately. Our results of the study were similar to previous studies. The Korean version of NDDI-E can be used as a practical screening tool to improve recognition of depression in Korean PWE.

### p091

## Postoperative changes in psychological features in patients with mesial temporal lobe epilepsy according to seizure outcome

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**Purpose:** Frequent seizures negatively affect on psychiatric features of epilepsy patients and successful epilepsy surgery improves quality of life and reduces neuropsychological burdens of patients. However, there were few studies which compared the postoperative changes of psychiatric features according to seizure outcome.

**Method:** We enrolled 138 MTLE patients who performed the Multiphasic Minnesota Personality Inventory (MMPI) 1 month before and 1 year after anterior temporal lobectomy and amygdalohippocampectomy (ATL +AH). They were divided into seizure free (Engle class I) and seizure not free (Class II-IV) groups according to postoperative seizure outcome (<sup>3</sup> 2 years after surgery). Resection margins were determined by comprehensive presurgical evaluation. **Results:** In seizure free group, 106 patients (76.8%; M:F=54:52, mean age 28.5 ± 8.8 yrs) were allocated and 32 (23.2%; M:F=14:18, mean age 29.5 ± 10.4 yrs) were in seizure not free group. Preoperative seizure frequency were not differed statistically between groups (3.5/m in seizure free vs. 5.4/m in seizure not free). There were no significant differences in MMPI profiles preoperatively between seizure free and seizure not free patients. When comparing the changes of the MMPI scores before and after surgery, there were significant decreases in 3 scales (hypochondriasis, depression, and hysteria), which are the index of neuroticism (p< 0.001) and the scores in scale 7 (psychathenia) and 8 (schizophrenia) were significantly reduced (p < 0.001) in seizure free group. Whereas, in seizure not free group, there was a tendency of decrease in most of scales and of increase in the scale 6 (paranoid), but they did not reach statistical significance. **Conclusing:** Our results found that MTLE positions the score of scales and of increase in the scale 6 (paranoid), but they did not reach statistical significance.

**Conclusion:** Our results found that MTLE patients showed a significant improvement in both neuroticism and contents of thoughts after successful epilepsy surgery.

### p092

## Analyses of personality profiles in patients with psychogenic non epileptic seizures, epileptic seizures, or combined type

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**Purpose:** Although psychogenic non-epileptic seizures(PNES) may coexist with epileptic seizure(ES), it is now widely recognized that PNES is caused by psychological factors. Patients with PNES have a higher prevalence of past traumatic experiences, anxiety, depression, fibromyalgia, and chronic pain. The aim of this study is to investigate the differences of psychiatric features among patients with psychogenic non epileptic seizures(PNES) with or without epileptic seizures(ES) and cryptogenic(no MRI lesions) ES using Multiphasic Personality Inventory (MMPI-2).

**Method:** We enrolled consecutively 65 patients (37male, 28female, mean age 32.2yrs) who were diagnosed with PNES, cryptogenic ES or combined(PNES+ES) by means of suggestion techniques or video-EEG recordings. Clinical demographics such as seizure onset age, duration, frequency, semiology, EEG and brain MRI findings, a history of antiepileptic drugs were reviewed in all patients. The MMPI-2 results were compared among the three groups.

**Results:** Twenty patients(22.2%) were included in PNES, 15(16.7%) were in PNES+ES, and 30(33.3%) were in cryptogenic ES group. There were no significant differences in clinical demographics among groups. Among three groups, there were significant differences of MMPI-2 profiles in Pt(Psychasthenia) and Sc(Schizophrenia) in major clinical features and some

subclinical features such as Hy4(appeal to physical symptoms), Sc3(lack of self- integration for cognition), Sc5(lack of self-integration for inhibitory dysfunction), Sc6(bizarre sensory experience), Ma2(hyperactivity mental exercise), Si3(internal and external isolation) (non-parametric, p< 0.05). PNES patients had the highest scores and cryptogenic ES patients had the lowest scores in those features. In the comparison of MMPI profiles between two groups, PNES patients showed definitely higher scores in Pt, Sc, Hy4, Sc3, Sc5, Sc6, Ma2, Si3 than ES and higher scores in Sc5, Sc6 than PNES+ES. There were no differences in MMPI profiles between PNES + ES and ES patients **Conclusion**: Our findings showed that PNES, PNES + ES and cryptogenic ES group had different psychiatric characteristics, especially in Pt, Sc and some subclinical scales. It seems that PNES patients had more frequent and complex psychiatric problems than PNES +ES or ES.

### Others

p093

# Complementary clinical use of video-EEG and MEG in frontal lobe epilepsy: correlation to outcome

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**Purpose:** This study is aiming to find out how Magnetoencephalography (MEG) and Video-Electroencephalography (VEEG) could be complementary to each other in frontal lobe epilepsy (FLE).

**Method:** Thirty patients with surgery of FLE were enrolled. VEEG recordings were obtained with an IT-med system using 10/20 system. Regional localization of spikes was defined as spikes discharged from adjacent electrodes and no further propagation to a large and/or contralateral area. MEG was continuously recorded for the purpose of focus assessment. MEG spikes were detected for dipole localization of the epileptogenic area. In MEG, regional localization was defined equally to mono-focal epileptogenic area localized by MEG; on the contrary, non-regional equaled to multi-focal epileptogenic area.

**Results:** In interictal VEEG, 20 patients (20/30) had regional spike discharges, including 11 (11/20) with Engel 1 outcome. In ictal VEEG, regional spikes discharges were found in 17 patients (17/30), including 9 (9/17) with Engel 1 outcome. Twenty patients (20/30) had mono-focal localization in MEG, including 14 (14/20) with Engel 1 outcome. Thirteen patients had regional spikes in both interictal and ictal VEEG, among which 7 patients (7/13) achieved Engel 1 outcome, including 5 patients with multi-focal localization in MEG. Conversely, 6 patients located only by MEG (without regional spike discharged in neither interictal nor ictal VEEG), among which 3 (3/6) had Engel 1 outcome postoperatively.

**Conclusion:** In clinical practice, VEEG is undoubtedly the routine procedure in the presurgical evaluation; moreover, MEG also has the advantages of locating the epileptogenic area in FLE with short-term investigation. Combination of VEEG and MEG can optimize the non-invasive presurgical evaluation and contribute to a favorable postoperative outcome in FLE.

## p094

# Verbal memory disorder and auditory cognitive event related potentials in epilepsy patients with secondary generalized seizure

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**Purpose:** To know the prevalence of verbal memory disorder and features of Auditory Cognitive Event Related Potentials in epilepsy patients with secondary generalized seizure **Method:** It was a cross sectional study. Memory disorders was assessed by Ray Auditory Verbal Learning Test. All patients were undergone binaural auditory cognitive event related potentials to compare the P300 latency between group with and without verbal memory disorder. **Results:** Verbal memory impairment was found in 22.6% patients from 93 patient with secondary generalized seizure. Factors that significantly related with verbal memory impairment is seizure frequency more than 4 times/month (p = 0.009). Mean latency of auditory P300 in all patients is 340 + 32.84 ms, patients with memory impairment has significantly longer latency (385.1+12.81 ms) than group without memory impairment (327.89+24.53 ms).

**Conclusion:** Verbal memory impairment in epilepsy patients with secondary generalized seizure is related with frequent seizure. Memory impairment is related with prolongation of P300 latency in Cognitive Event Related Potentials.

## p095

#### Therapeutic drug monitoring, Epilepsy Clinic, Srinagarind Hospital, Thailand

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**Purpose:** To study the correlations between the reason behind TDM order and the prescriptive level, interpretation of concentration and pharmacists' advice, and physicians' compliance to pharmacists' interpretation of drug levels.

Method: This is a prospective descriptive study in which data collection was done from January 1 to December 31, 2010, the period when pharmacists took part in assessing the appropriateness in measurement and interpretation of TDM in order to provide suggestions for physicians. **Results:** The 112 patients, 254 samples were collected for therapeutic drug monitoring. Phenytoin was submitted mostly for drug monitoring at 46.46%. 44.49% of submissions for drug level monitoring were made owing to a suspected sub-therapeutic level. Correlations were found between reasons behind sending samples for drug level monitoring, i.e., 66.67% of drug levels were found so low that they were undetectable in sample for patients' compliance investigation, 40% of the cases were found to be in toxicity range in the cases with suspected over-therapeutic levels and monitoring levels. Pharmacists used the interpreted results in patients' care by recommending physicians to monitor therapeutic drug closely, to adjust the dosage regimen, and to recommend checking patients' compliance at 56.5, 38.9, and 4.3%, respectively. Physicians' responses were found to be absolutely, partly, or not follow in 77.95, 11.03, and 7.48%, respectively. **Conclusion:** It was proved that therapeutic drug monitoring services at the Epilepsy Clinic was useful in supporting clinical information queries. It should be noted that physicians accepted pharmacists' suggestions, denoting multi-professional patient treatment that would lead to greater efficiency.

### p096

#### A case of sleep-induced cortical myoclonus

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**Background:** Sleep-induced cortical myoclonus is very rare. We describe a patient presenting with trunk and bilateral leg jerking movement during sleep-inducing period.

**Case:** A 39-year-old man visited our clinic because of abnormal rhythmic jerking movement of trunk and both legs during asleep, producing sleep disruption with a complaint of insomnia. The abnormal movement was never seen during awake. It developed just before sleep, make sleep disturbance. There were no consciousness alternation. He said the jerks were started when he feel sleepy. He was healthy until two years prior to visiting our clinic. The onset mode of jerky movements was gradual, it was not have progressive course. For 2 year, he felt fatigues because of sleep disturbance. We performed video-EEG monitoring and polysomnography. The video-EEG monitoring showed sleep-induced myoclonus of trunk and both legs associated with cortical activities. Cortical myoclonus were developed just before asleep and continued during sleep N1, and than disappeared during stage N2 and REM sleep. Myoclonus was dramatically disappeared after use of levetiracetam.

## Zonizamide as monotherapy to genralized seizures in Indian /Asian: an observational study $\underline{\sf NAGARAJAN \ V}$

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**Purpose:** To observe the right dose of Zonizamide among the Asian , Indian population , as Monotherapy compared to Carbamezapine in age matched control group of patients suffering from Generalized Seizure disorder. Also to establish that the Western patient's dosage are not necessarily to be adopted in Asian patients.

**Method:** Total of 360 patients has been selected with Zonizamide grup (Gr A) of 200 and Carbamezapine group of 160.(Gr B) The average age group A is 30, with standard deviation of 13.23 and in Gr B 28.13 with the standard deviation of 12.10 The dosage of Zonizamide (Zonegren) given to these patients, 100 to 300 mg and the dosage of Carbamezapine group was 200 to 400 mg per day. The parameters on which the study was based on reduction of clinical and seizure discharges in EEG records. These observations in the study group were observed over a period of 12 weeks. The gradual clinical seizure episode sreduction and EEG seizure discharges were observed , and results were interpreted. The side effects of both the drugs as monotherapy were evaluated .

**Results:** With Zonizamide , the post study control of EEG seizure discharges reduction with the Student "t" distribution and the probability scoring is 0.0387 which P < \_ 0.05 , the Clinical Seizure reduction with the probability of 0.2019 which is >\_0.05 which is significant. Hence in Indian, Asian patients Zonizamide appears to be best drug with monotherapy with very minimal side effects.

**Conclusion:** With Zonizamide , the post study control of EEG seizure discharges reduction with the Student "t" distribution and the probability scoring is 0.0387 which P < \_ 0.05 , the Clinical Seizure reduction with the probability of 0.2019 which is >\_0.05 which is significant . Hence in Indian, Asian patients Zonizamide appears to be best drug with monotherapy with very minimal side effects.

#### p098

### Antiepileptic drug initiation in relation with seizure free period in partial epilepsy

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**Purpose:** The purpose of this study was to seek possible relations between antiepileptic drug initiation, duration of drug consumption, epiletic focus, mono/polytherapy and seizure free period. **Method:** It was a cross-sectional study. Subjects were patients with partial epilepsy admitted to the Neurology Department, Cipto Mangunkusumo Hospital, from January to August 201. Data collected including age, sex, epileptic focus, duration of drug consumption, the initiation of antiepileptic drug since the first seizure, the use of mono and polytherapy, and seizure free period. Seizure free period was divided into 3 groups, less than 12 months, between 12-24 months, and more than 24 months. **Results:** One hundred and forty nine partial epilepsy patients were analysed. The majority of patients were women (61.7%), at the age of  $34\pm14$  years old, began their first seizure at less than 19 years old (49%), started anti epileptic drug mostly after 24 months (62.4%) since the first seizure, and epileptic focus were predominantly temporal (67.8%). Monotherapy was used in 78.5% of the case. Patients who started treatment less than 12 months after the first seizure, had significantly longer seizure free period (df=3, p=0.01) compared with later initiation. The duration of drug consumption also affect seizure free period (df=3, p=0.038).

**Conclusion:** Seizure free period was related to antiepileptic drug initiation since the first seizure, hence emphasizing the importance of early treatment in epilepsy. The duration of drug consumption was equally related to seizure free.

#### Paediatric epileptology

## p099

### Siblings of the suspicious Lafora disease in Japan

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Purpose: We present a Japanese siblings of the suspicious Lafora disease.

Method: Patient 1 is the middle daughter born to unrelated parents. She has two sisters: one elder the other younger. Patient 1's growth and development was normal until she was 13.0 yo. She first experienced bright flashes in her visual field when she was 13.5 yo, thereafter experiencing the same several more times. After 3 months from for the first feeling, she caused the initial generalized tonic-clonic convulsion. After approximately 6 months, she began to display developmental regression. She gradually experienced deteriorated in her calculation skills, speech fluency, and her capacity to walk without assistance. She was admitted to our hospital at the age of 13 years 7 months, by which point she could do almost nothing by herself for her daily life. She was hypotonic, and had tremors and dysmetria in her upper arms. We could not induce her deep tendon reflexes. Myoclonic movement has been sometimes observed in her hands. Routine laboratory blood and urine examinations returned normal. Her skin and rectal biopsies showed that there is no presence of Lafora bodies. Electroencephalography showed background slow activity, generalized spike and wave complex, and continuous delta slow waves we are detected throughout, but especially in the occipital area accompanied by polyspikes. We found Giant SEP potential from her median nurve stimulation. Patient 2 is the younger sister of Patient 1. Like Patient 1, she experienced bright flashes in her visual field just after she played a computer game when she was 13.0 yo. Soon afterwards she suffered initial generalized tonic-clonic convulsion. Her electroencephalography showed the same pattern to Patient 1.

**Results and conclusion:** We have already recerch for all of the known point mutation for the EPM2A and NHLRC1 gene with GENIDA (Genetic Diagnostic Network in Belgium), that resulted in no point mutation in those genes. Clinical feature, however, is exactly the same as that of the Lafora disease.

## p100

Alice in Wonderland syndrome associated with Panayiotopoulos syndrome

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**Purpose:** Alice in wonderland syndrome (AIWS) is a rare condition involving visual perceptions and may associated with viral infections, migraine and epilepsy. Panayiotopoulos syndrome (PS) is an epilepsy with autonomic symptoms and multifocal spikes on EEG. We report a case of AIWS with PS. **Case presentation:** A 7 years-old girl experienced suddenly and prolonged visual distortions after head banging. She falled down and banged midline of forehead to the steel bumping post when she was walking to home. She complained visual abnormality at night. Her mother's face were stretched widely, like a view on concave mirror or third mother's eye positioned between eyes or under the left eye. She experienced macropsia and micropsia when she was 6 years old. Her hand expanded threefold. Her mother, 160 cm tall, got smaller to 6 cm length. Her father suffered migraine and had same visual conditions until 12 years old. She vomited at night seven times from 4 years old. Clinical examination, Brain MRI were normal. EEG showed high amplitude spike and waves over bilateral occipital ~left posterior temporal areas. Medication of valproate improved both visual abnormality and womiting seizures.

**Conclusion:** AlWS associated with epilepsy, especially PS, is a rare condition. Both conditions evolves usually self-limited. We treated as epilepsy because stereotyped vomiting during sleep continued. Anti-convulsants may be useful for epilepsy and migraine.

#### Early onset rolandic epilepsy is intractable?

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**Purpose:** The aim of this report is to discuss the relation between the age at seizer onset and the response to antiepileptic drug (AED) in rolandic epilepsy (RE). While RE is the most common epilepsy in children with favorable prognosis, there have been reported some atypical cases complicated by frequent seizure recurrence or higher brain dysfunction.

**Methods:** We classified thirty-four children into three groups: those who were taking more than one AED were assigned to Group A; taking one AED to Group B; and observed without medication to Group C. Regarding Group A, the meaning of 'more than one' is not co-administration but alteration of the remedy.

**Results:** Characteristics of the patients (Number of cases/ Mean age at seizer-onset/ Mean frequency of seizer) were as follows. Group A:(10 /4.2 $\pm$ 1.5 /16.4), Group B:(14 /7.2 $\pm$ 1.4/4.6), Group C:(10 / 6.0 $\pm$ 1.6 / 2.2). Eighty percent of patients in Group A had once been prescribed carbamazepin.

**Discussion:** [1] The age at seizer onset was remarkably lower in Group A. [2] The seizure frequency was higher in Group A. [3] The early onset cases are evolved into intractableness to medication. [4] Carbamazepin is likely to be ineffective for the patients in group A.

**Conclusion:** The early-onset RE tends to show poor response to initial treatment and thus need to use multiple sort of AED. In those cases, relatively resistance to carbamazepin is suggested, so we need to consider the strategy for the management.

#### p102

#### Experience of epilepsy treatment in Angelman syndrome in Korea

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**Purpose:** Angelman syndrome (AS) is a neurogenetic disorder with epilepsy present in more than 80% of affected individuals. The aim of this study is to investigate the natural history of epilepsy and response to antiepileptic drug treatment in patients with AS in Korea.

**Method:** We retrospectively reviewed the clinical records of 14 patients, all with the genetic diagnosis of AS. These patients were seen at the Paediatric neurology in Severance Children's Hospital between March 2005 and March 2011.

**Results:** Fourteen subjects (9 boys and 5 girls), with a median follow-up of  $6.7 \pm 5.6$  years (range, 2.1 ~ 23 years) were identified. Three patients (21.4%) were determined by maternal deletion of chromosome 15q11-13 and 11 patients (78.6%) were determined by abnormal methylation of maternal allele. Age at seizure onset was a median of  $1.9 \pm 0.8$  years (range, 7 months ~ 4.0 years). The most common seizure types were complex partial seizure (n=9), myoclonic (n=7), generalized tonic-clonic (n=7), atonic (n=6), atypical absence (n=2). Multiple seizure types were observed in 8 patients (57%). At the time of the study, patients had an average of 2.6 current antiepileptic drugs (AED) medications, with 14% currently on monotherapy and 86% having tried multiple medications. The most commonly prescribed AED were valporic acid (n=9, 64%), lamotrigine (n=7, 50%), levetiracetam (n=5, 36%). Complete control of seizures was achieved in 9 patients. Partial control was achieved in 4 patients, while one patient was not controlled. Control of seizures was achieved at an average age of 7.5 ± 4.1 years (range, 3.6 ~ 14.1 years).

**Conclusion:** Epilepsy is observed in the great majority of AS patients, may have early onset and is often refractory to treatment. There are few reports about epilepsy in AS in Korea. This study will be helpful in understanding epilepsy in AS in Korea. There is also need for further clinical research for improving management of problem such as behavior, communication, learning, motor impairment, and sleep disturbances in AS.

**Remote effect of spike discharges in patients with atypical benign partial epilepsy: a SPECT study** <u>HAGINOYA K<sup>1,2</sup>, UEMATSU M<sup>2</sup>, FUKUYO N<sup>2</sup>, MATSUMOTO Y<sup>2</sup>, NAKAYAMA T<sup>2</sup>, KOBAYASHI T<sup>2</sup>, KAKISAKA Y<sup>2</sup>, NAKASATO N<sup>3</sup></u>

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**Purpose:** Most of patients with atypical benign partial epilepsy (ABPE) are suffering from poor school performance including writing, calculation, reading and phonation problem. The correct diagnosis was sometimes difficult to make in the early stage since seizure semiology and EEG findings are silimar to those of benign rolandic epilepsy. To characterize the remote effect of spike discharges frequently appearing in the rolandic areas in patients with ABPE, we performed SPECT study and compared to the normal control.

**Methods:** Two right-handed patients with ABPE (7 years old) had frequent rolandic seizures, atypical absence, head drop seizures since 3 and 4 years old, respectively. EEG showed bilateral spikes and spike-waves in both rolandic region. CSWS was sometimes observed in both patients. MEG also showed dipole localization in the bilateral rolandic areas in one and rolandic area and sylvian fissure in another. At the peak of frequent seizures, both had disturbed oral function, decrease in speech, dysarthria, dysgraphia, and dyscalculia. ECD-SPECT study was preformed at this period, when both patients admitted to our hospital for seizure control and preoperative evaluation of seizure type. IQ was 75 and 53, respectively. After diagnosing ABPE, we used ethosuximide in one and ethosuximide and acetazolamide in another, which resulted in complete disappearance of seizures and dramatic improvement of school performance as well as remarkable improvement of EEG.

**Results:** ECD-SPECT analyzed with e-ZIS clearly showed significant hypoperfusion in the left inferior frontal cortex in both patients. However, the second SPECT in one patient, which was studied after the gain of seizure control, showed normal perfusion in the same region.

**Conclusions:** This study first showed remote effect of rolandic discharges to the speech processing region in ABPE. A long lasting frequent seizure period might bring profound effects on this area. A precise mechanism of this remote effect remains to be elucidated.

#### p104

## Analysis of interictal epileptiform discharges in benign Rolandic epilepsy: predict to prognosis of seizures

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**Purpose:** Benign rolandic epilepsy (BRE), most commonly suffered by children between the ages of 3 to 13, is focal epilepsy with centrotemporal spikes in electroencephalography (EEG). Our aim was to measure the influence of such factors as onset age, duration, frequency of seizures, and use of anticonvulsants on prognosis, through analyzing BRE patients' interictal EEG clues.

**Method:** Between January 2005 and December 2010, seventy four patients, who had had follow-up for over a year after being diagnosed with BRE, were enrolled at Department of Paediatrics, Korea University Guro Hospital. The seizure onset age, frequency, use of antiepileptic drugs (AED), period of AED use, seizures with medication, age of EEG normalization, last seizure age, frequency of interictal discharges over time and average voltage in each arousal and sleep phase were analyzed. **Results:** The patients' average age was  $11.9 \pm 3.1$  years old. Thirty-eight girls (51.4%) and thirty-six boys (48.6%) were enrolled. Higher number of febrile seizure histories was associated with the younger age of seizure onset (P=0.005). And the younger age of seizure onset was correlated with the younger age of last seizure (P=0.001) and EEG normalization (P=0.004). The patients who had taken anticonvulsants showed significantly lower intelligence (P=0.010). Frequent episodes of interictal epileptiform discharges during sleep was correlated with the younger age of seizure onset of AED therapy (P=0.022) and the younger seizure cessation age (P=0.027). It also had more interictal epileptiform discharges during wakefulness (P=0.006), higher average voltage (P=0.043) period.

**Conclusion:** In BRE patients, the higher number of interictal epileptiform discharges during sleep phase in EEG, the more seizure events were observed even after anticonvulsant therapy. These will be helpful for us to predict patient's prognosis in the future.

#### p105

## Retrospective review of the etiologies of acute symptomatic seizures in children with acute lymphoblastic leukemia (ALL)

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**Purpose:** Chemotherapy for acute lymphoblastic leukemia (ALL) has been described to be associated with intractable epilepsy. Recently, chemotherapy related seizures have been observed less frequently because of less neurotoxic chemotherapy protocols. However, data on seizures in children receiving treatment for haematological malignancies remain sparse. We aimed to identify patients with ALL who developed seizures and to identify the etiologies and risk factors associated with seizure occurrence.

**Method:** This is a retrospective review of clinical charts of children with ALL who had seizures from July 2002-September 2011. Seizure semiology, electroencephalography (EEG), neuroimaging, and patients' clinical profile were reviewed.

**Results:** Thirteen out of 120 patients (10.8%) with ALL had seizures. 7 were ongoing induction chemotherapy using MASPORE protocol, 2 were on reinduction chemotherapy for relapse, 3 were post-stem cell transplantation, and 1 was in remission. 62% of EEGs done were consistent with seizure semiology. Neuroimaging showed various etiologies, including cerebral sinovenous thrombosis (CSVT), generalized cerebral swelling, left parietal fungus ball, and posterior reversible leukoencephalopathy. 5 patients were still on anticonvulsants.

Five patients had L-asparaginase-related seizures during induction chemotherapy. Patients received L-asparaginase between 3 to 7 days prior to seizure events. Two had headache before having seizures. Fibrinogen levels were 30% to 50% below normal.

**Conclusion:** While children with ALL are at risk of seizures during induction chemotherapy and post-stem cell transplantation, the overall incidence of seizures remained low at 10.8%. However, CSVT appeared to be more frequent in individuals receiving L-asparaginase and the neurotoxicity of L-asparaginase should be considered in future protocols.

## p106

#### Outcome of antiepileptic drugs withdrawal in childhood onset nonidiopathic focal epilepsies <u>PAVLOVIC M1</u>, JOVIC N2, PEKMEZOVIC T3

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**Purpose:** To assess the risk of seizure recurrence and factors influencing antiepileptic drugs (AED) withdrawal outcome in patients with nonidiopathic (cryptogenic and symptomatic) focal epilepsies (NIFE) commencing at the preschool age.

**Methods:** Medical records of consecutive patients with NIFE in the setting of the tertiary level hospital were monitored and evaluated during the period between 2001.and 2009. Inclusion critera were: diagnosis of cryptogenic (CFE) and symptomatic (SFE) focal epilepsies according to the ILAE criteria, (2) age at onset of epilepsy of < 7 years, (3) established clinical remission of at least two years before AED withdrawal, (4) no generalized EEG abnormalities before AED withdrawal and (5) follow up period of at least two years after withdrawal or until seizure relapse in patients who relapsed. Time to seizure relapse and variables considered as possible predictive factors were analyzed by survival methods. Seizure relapse was considered as the main outcome endpoint. **Results:** The cohort consisted of 44 patients (15 females, 29 males), aged 6-28 years (median 16) at the end of the study. Cryptogenic etiology was assigned to 23 and symptomatic to 21 patients. Patients were followed up 1-13 years (median 4) since the beginning of AED withdrawal. Relapse occurred in 18 (40.9%) patients (in 7 with CFE and in 11 with SFE). Most of relapses (76.5%) occurred during the first 12 month since withdrawal. Univariate analyses indicated following factors

being significantly (p< 0.05) correlated with seizure relapse: (1) age at onset of epilepsy < 4.5 years; (2) symptomatic etiology; (3) EEG focal abnormalities before withdrawal and (4) mental retardation (IQ < 70). Multivariate Cox regression analyses revealed that age at onset of epilepsy < 4.5 years and mental retardation remained as independent predictive factors for seizure recurrence upon AED withdrawal.

**Conclusion:** Recurrence risk after AED withdrawal in patients with NIFE was higher than usually reported. The first year since withdrawal was the period of highest risk for seizure relapses. Younger age at onset of epilepsy < 4.5 years and mental retardation were distingished as independent predictive factors related to seizure recurrence upon withdrawal.

#### p107

## Cortical dysplasia in the genesis of refractory epilepsy in children

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**Purpose:** To study the medical history, neurological, electrophysiological and neuroradiological features of epilepsy in children with cortical dysplasia.

**Method:** 7 children aged from 1 to 6 months (boys - 4, girls - 3) with diagnosis of "resistant form of epilepsy."

**Results:** The analysis revealed the presence of prenatal chronic fetal hypoxia in 100% of cases, grade 2-3 anemia (100%), primary CMV infection in the mother (3 cases), exacerbation of chronic herpes infection (4 cases). 4 children were born prematurely. Condition at birth was assessed as severe in all children. Early neonatal seizures were observed in 5 children. In the future all the children in this group developed postneonatal epilepsy: syndrome Ohtahara (1), West syndrome (2), symptomatic partial epilepsy (2). 2 children had debut of epilepsy on the type of generalized tonic clonic seizures at the age of 3 months. Electroencephalographic data: typical hypsarrhythmia (2), atypical hypsarrhythmia (2), high-voltage fast activity in the beta range (1), burst-suppression (2). In the neurological status were observed severe delay in psychomotor development (100%), spastic paresis (4 cases), severe muscle hypotonia (3 cases), optic atrophy (5 cases). Neuroradiological examination revealed the presence of cortical dysplasia: lissencephaly (1), heterotopia (1), pachygyria (2), schizencephaly (3) in combination with other anomalies: hypogenesis of the corpus callosum, hypogenesis of the cerebellum, hydrocephalus.

**Conclusion:** In the etiology of epilepsy in children of this group we have traced the role of viral infections and chronic fetal hypoxia. Epileptic syndrome in all cases had a severe course, a high frequency of seizures and was resistant to medication. Therapeutic strategy included the appointment of anticonvulsants in polytherapy in high doses. All the children had a severe neurological deficit.

## p108

## Hypsarrhythmia severity; correlation with infants age, duration from onset of spasms and spasms load

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**Purpose:** Hypsarrhythmia is the electroencephalographic hallmark of epileptic spasms. A 16-point score using 5 key features has been described to assess its severity previously (Kramer et al 1996). The aim of this study was to examine the association of hypsarrhythmia severity score with the age of the infant, duration since onset of spasms and spasm load.

**Method:** Thirty previously untreated patients with infantile spasms were subjected to a 30 minute digital EEG within 30 minutes of falling asleep. Background disorganisation, diffuse delta activity, voltage of epileptic discharges, presence of spike and sharp waves were each marked on a scale of 0-3 and presence of electrodecremental pattern, burst suppression in sleep, absence of normal sleep patterns and relative normalisation in wakefulness were marked as one point each. Scoring was performed by a paediatric neurologist blinded to the clinical or treatment details. Fisher's Exact and Spearman Coefficient Score were used to describe a possible association.

Results: The median age at presentation was seven months (range=2.5 · 50), mean time interval

between spasm onset and treatment was 4.7 weeks (SD±9.99), mean number of clusters a day was 5.0 and mean number of spasms/day was 29.13. The mean hypsarrhythmia severity score was 10.8 (SD + 2.53); it was considered "low score" (< 10) in 12 and "high score" (> 10) in 18. When applied Fisher's exact probability test, a high hypsarrhythmia score was not associated with age at presentation (p= 0.99), down time for treatment (p=0.57) or spasm load (p=0.29) described using total number of clusters a day as well as the total number of spasms on day one of therapy. When taken as continuous variables, Spearman correlation coefficients were < 0.2 in all three indicating that there is no strong association.

**Conclusion:** The results show that there is no association between a high hypsarrhythmia score and age of presentation with spasms, duration without treatment or spasms load.

#### p109

#### Efficacy of levetiracetam in refractory childhood epilepsy

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**Purpose:** To evaluate the efficacy and safety of levetiracetam adjunctive therapy for reducing the rate of seizure frequency in children with intractable Paediatric epilepsy.

**Method:** We reviewed the medical records of 86 patients with intractable Paediatric epilepsy who visited our hospital between March 2005 and February 2010. Levetiracetam was included in the previous anticonvulsant regimen for at least 6 months and the reduction in the rate of seizure frequency was determined in follow-up examinations. We analyzed demographic data, seizure types, antiepileptic drug history, levetiracetam dose, adverse effects of levetiracetam therapy, treatment outcome, electroencephalogram findings, etc.

**Results:** More than 50% reduction in the seizure frequency was observed in 62 of the 86 (72.1%) patients; 44 patients (51.1%) became seizure free, while the seizure frequency increased in 5.8% patients. The associations between seizure reduction rate and age, associated diseases, seizure types, and seizure frequency before treatment were not significant. However, the duration of disease, dose of levetiracetam, duration and frequency of anticonvulsant administration before levetiracetam therapy were significantly correlated. Electroencephalogram findings and the cause of epilepsy showed partial correlation. 40 (46%) patients showed adverse symptoms; the symptoms in the order of their frequency were somolence, hyperactivity, irritability, aggressiveness, tiredness, etc. **Conclusion**: The findings of our study provide the evidence that levetiracetam adjunctive therapy is efficacious and well tolerated in various refractory childhood epilepsy.

#### p110

## A clinical study of febrile seizures in children over 5 years old without organic problem $\underline{\sf PARK}\,HJ^1,\,{\sf LEE}\,\,{\sf KS}^2$

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**Purpose:** Febrile seizure occurring after 5 years of age might be estimated as epilepsy. Most data for these patients are from mainly epilepsy populations and might not be relevant to these children, and the requirement of any specific treatment or workup is not yet established either. Thus we tried to obtain a unique perspective.

**Method:** We retrospectively reviewed and analyzed data of 259 patients over age 5 who were admittend to the Eulji University Hospital because of seizure with fever.

**Results:** Among 259 patients, 121(46.7%) were epilepsy, 13(5.0%) were caused by infection, metabolic disorder, leukemia and so on, and 23(8.9%) had previous neurological defect. Lastly 102(39.4%) were included to this study. In 28(27.5%) patients, the onset of seizure was after 5 years of age, while 74(72.5%) were before 5 years of age. The sex ratio was 2:1. Generalized seizures occurred predominantly within 15mins in both of them. Most seizures occurred less than 24hrs after fiver. Five(17.9%) of group A and 16(21.6%) of group B showed abnormal electroencephalogram , but there were no abnormal findings in lumbar puncture and brain MRI. **Conclusion:** Febrile seizure in over 5 years old without organic problems has similar characteristics to febrile convulsion in under 5 years old. And lumbar puncture and brain MRI do

not require a routine checkup for the evaluation of febrile seizure in over 5 year olds. However, electroencephalography needs to be examined.

#### p111

#### Gastroesphageal reflux disease in infants with epilepsy

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**Purpose:** Gastroesophageal reflux disease (GERD) during infancy can have variable manifestations such as vomiting, irritability, arching, choking and apnea. Although GERD can be frequently misdiagnosed as seizures, it is possible that infants with epilepsy may have both conditions. We identified GERD in infants with epilepsy, and studied their characteristics and outcome of seizures. Method: We retrospectively reviewed the records of 230 infants (< 6 months) diagnosed with epilepsy at Asan Medical Center between January 2007 and July 2011. GERD was diagnosed through esophagography or 24-hour pH monitoring in infants with typical symptoms of GERD (vomiting, choking), symptoms that may mimic seizures (arching, apnea) or seizures associated with feeding or position (seizures during or after feeding, seizures during supine position). Results: The incidence of GERD was 18.7% (43/230) among infants with epilepsy. These infants showed typical symptoms of GERD (20/43, 46.5%), symptoms that may mimic seizures (15/43, 34.9%) or seizures associated with feeding or position (8/43, 18.6%). The mean age of seizure onset was 1.7 months (0-5 months). Developmental delay was present in 26 (60.5%) infants. Intractable seizures were present in 11 (25.6%) infants. The risk of intractable seizures was higher in infants with GERD and epilepsy, compared to infants only with epilepsy, regardless of developmental delay (OR=6.09, 2.38-15.5, p< 0.005).

**Conclusion:** Although GERD can be frequently mistaken for seizures, a high proportion of infants had both conditions. Presence of GERD was related to higher risk of intractable seizures in infants with epilepsy, regardless of their neurodevelopmental status. Therefore, identification of GERD may be helpful in the management of infants with epilepsy.

## p112

#### Seizures in children with demyelinating changes of the brain

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**Purpose:** To study the etiological factors, the characterization of the debut seizures, clinical and neuroradiological manifestations in children with demyelinating changes of the brain. **Method:** 9 children under the age of 3 years (F-3, M-6). Applied clinical, neurological, neuroradiological and laboratory examination.

**Results:** We studied children with demyelinating changes the brain (n = 9). Anamnesis analysis showed that CMV infection was the main etiological factor in the form of CMV encephalitis  $\cdot$  n = 1 (11%), generalized form of infection with CNS, liver, lung disorders  $\cdot$  n = 3 (33%), and flowed in a latent form  $\cdot$  n = 5 (56%). Convulsions debut in children with demyelinating changes in the form: symptomatic epilepsy  $\cdot$  tonic infantile spasms in the neonatal period (1), 3 children have  $\cdot$  manifested in the adversive seizure (1), partial seizures with secondary generalization (1), myoclonus epilepsy (1), at the rest (5)  $\cdot$  epileptic syndrome manifested at the second year of life in the form of generalized tonic-clonic seizures with infrequent episodes, abnormal vision, delayed psycho-motor development. Neuroradiological investigation (T2MRI) in 4 patients  $\cdot$  large 2-3 focuses of demyelination in the white matter of the cerebral hemispheres and cerebellum, and 5 small disseminated areas of demyelination (subacute leukoencephalitis) in the periventricular region.

**Conclusion:** a convulsive syndrome in children with demyelinating changes of the brain occurs polymorphically. Viral infection plays a leading role in the etiology. Clinical manifestations are compared with neuroradiological changes.

## Congenital vascular malformation in the genesis of focal epilepsy in children. Clinical case <u>NUKEBAYEVA Z</u>, LEPESSOVA M, MENDIGALIYEVA N

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**Purpose:** to study an epilepsy in children with congenital venous angioma as an example of a clinical case.

Method: A girl of 6 years.

**Results:** The complaints of convulsions with loss of consciousness. The debut of seizures at 1.5 years against the background of increasing body temperature. The second episode of convulsions - at 6 years against the background of the transferred viral infection. Attack was preceded by an aura in the form of severe headaches. In postconvulsion period - condition was severe , consciousness was inhibited. Craniocerebral innervation - Install horizontal nystagmus, deviation of the tongue. Transient right-gemisyndrom, positive Babinski sign. Mental development of appropriate age. There was constant subfebrilitet.

**Surveys:** CSF analysis · within limits. EEG · cortical rhythm is disorganized. Specific epileptiform activity there, residual character changes;

MRI - venous angioma in the basal medial temporal lobe of the left hemisphere, the signs of sclerosis of hippocampus.

While in hospital the child was observed a short episode of partial seizures with subsequent psychomotor stimulation.

Was appointed to the drug valproic acid at a dose of 37.5 mg / kg, dexamethazone 0.3 mg / kg. The child's condition has stabilized over time, seizures were not repeated.

**Conclusion:** This case report describes symptomatic focal epilepsy, which lies in the pathogenesis of cerebral blood flow of ischemic type in the background of venous angioma. Seizures triggered by hyperthermia and proceed against the background of a long subfebrile. The presence of the temperature, symptoms of intoxication, hypoxic-ischemic brain tissue with gliosis in the absence of changes in cerebrospinal fluid exclude neuroinfection. Necessary to differentiate this case from the syndrome FIRES.

### p114

### Risk factors of recurrence febrile seizure

GUNAWAN PI

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**Purpose:** The aim of this study is to determine the risk factors of RFS in children age 6 months to 5 years old at Dr Soetomo Hospital Surabaya.

**Method:** This is a cohort study. Data was taken from all children with FS who were admitted at Paediatric ward Dr Soetomo Hospital from August 2009 to November 2010. They were observed for 1 year to ascertain wether FS recurred. The relationship between variables like sex, age, temperature during the episode of fever, family history of seizures, diagnose at onset, maternal illness, abnormal delivery and recurrence FS were analyzed using logistic regression.

**Results:** Recurrence FS occured in 65 % of the 100 children at one year, and 26% of them had further attack. Male to female ratio was 2:1.Thirty-one (77.5 %) were < 12 months-old. Logistic regression analysis revealed temperature  $\leq$ 38.5°C (p=0.019, OR 5.496 (CI95% 0.082;0.800)) and complex FS (p=0.025,OR 5.032(CI95%1.143;7.256)) were most related to increased risk of reccurence.

**Conclusion:** The risk of recurrence febrile seizure increases with temperature  $\leq$ 38.5C and diagnosed as complex febrile seizure at first febrile seizure.

### Prognosis

#### p115

#### Clinical features of epilepsy developed after head trauma

<u>NAKANO H</u>, KINOSHITA M, SAWADA H National Hospital Of Utano, Kyoto, Japan **Purpose:** To investigate clinical features of the patients with partial epilepsy caused by head trauma.

**Method:** We retrospectively reviewed medical records of 577 patients who visited our epilepsy clinic between January and December, 2010. Patients who fulfilled the following criteria were included; (1) with exact epileptic seizures, (2) no history of seizures before the head trauma, (3) history of the trauma severe enough to impair brain, (4) records of EEG and either of CT or MRI, (5) over 20 years old at the time of clinic visit, (6) partial seizures, and (7) only one traumatic episode before the onset of seizures. Characteristics of the trauma (age at injury, cause, skull fracture, intracranial hemorrhage, brain contusion), the seizures (onset after injury, type, frequency, intractability, EEG findings), and the other risk factors for epilepsy were investigated.

**Results:** 32 patients were enrolled. 8 patients (25%) developed their seizures more than 10 years after the trauma. 2 patients had seizure semiology compatible with mesial temporal origin as well as hippocampal atrophy, without traumatic brain lesions detectable on imaging studies.

**Conclusion:** Epileptogenesis following a head trauma may take more than 10 years. A trauma might promote an epileptogenic process of mesial temporal lobe epilepsy with hippocampal atrophy.

### p116

### Outcomes of idiopathic generalized epilepsy syndromes in a non-paediatric setting

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**Purpose:** To evaluate outcome of idiopathic generalized epilepsy syndromes (IGES). **Method:** We investigated 136 patients who were diagnosed as having IGES from the Yonsei Epilepsy Registry database, documented by a physician (K. Heo). The patients remaining seizure-free for at least 1 year at the last of follow-up were regarded as being in remission.

Results: Twelve patients were excluded due to loss of follow-up, poor compliance, or short followup period. Average age of onset in IGES was 10s (mean, 16.8 years; range, 3.51 years). Average age of onset in patients with epilepsy with generalized tonic-clonic seizure (GTCS) only was older compared with those with other IGESs ( $22.9\pm10.7$  vs.  $14.4\pm4.0$ , p< 0.001). Twenty of 124 (16.1%) patients had a history of febrile seizure, and generalized epileptiform discharges were found in 81 of 124 (65.3%) patients. Ninety-five (77.6%) patients had not taken antiepileptic drugs (AEDs) at their initial visit. Juvenile myoclonic epilepsy (JME) was the most common IGES and was present in 62 patients (50%), followed by epilepsy with GTCS only in 35 (28.2%) patients, invenile absence epilepsy (JAE) (n=15, 12.1%), and childhood absence epilepsy (CAE) (n =12, 9.7%). One hundred four (83.9%) patients achieved remission (JME, 91.9%; GTCS only, 88.6%; JAE, 66.7%; and CAE, 50.0%). Eighty-six (82.7%) of the patients achieving remission had been treated with AED monotherapy [valproate (n=41, 47.7%), lamotrigine (n=25, 34.7%), topiramate (n=8, 9.4%), etc.]. Seventeen (16.3%) patients had taken AED duotherapy (including VPA in 16). Three of 20 patients with uncontrolled seizures was initially controlled with valproate but could not achieve remission with other AEDs switched because of concern for pregnancy, and nine patients had not experienced valproate therapy.

**Conclusion:** Idiopathic generalized epilepsy syndromes had excellent prognosis, most of whom achieved remission usually with a single antiepileptic drug, particularly valproate.

#### Seizure semiology

#### p117

#### Epilepsia partialis continua with recurrent transient left arm monoparesis PARK HS<sup>1</sup>, YOON YH<sup>2</sup>

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**Background & Significance:** Arm monoparesis is characterized by decreased frequency and strength of upper extremity movements due to weakness. Although brain, cervical spine, peripheral nerve below the brachial plexus, and etc. can cause arm monoparesis, we usually think of as peripheral or spinal cord lesion.

Case: A 40-year-old man presented with weakness of the left arm, especially below wrist. After he

admitted, his arm weakness lasted for a few hours and was fully recovered. After a while, a clonic movement appeared around wrist during about 1 minute and he had a weakness in the left arm, especially below wrist. Since that event, he had a few more symptoms for three days. Brain imaging showed acute infarction in right occipital lobe(non motor cortex). Brain PET showed metabolic defect in right occipital cortex and ictal brain SPECT showed focal increased perfusion on right frontotemporal cortex considered hand motor area. EEG showed that ictal activity started on right occipital areas spread rapidly to involve all of ipsilateral frontotemporal lobe. His arm weakness was not recurred after the use of antiepileptic medications. Recurrent transient arm weakness was caused by Todd's paralysis.

**Conclusions:** We report a interesting case with arm monoparesis caused by non-motor area stroke. Seizure disorder, such as epilepsia partialis continua should included in the differential diagnosis of arm monoparesis.

## p118

# A case of hypoparathyroism presented with epileptic seizures and dystonia mimicking pseudo seizure

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**Purpose:** Parathyroid hormone (PTH) deficiency, a rare disorder that may cause hypocalcemia and hyperphosphatemia, is associated with a variety of neurological symptoms, from being asymptomatic to extrapyramidal manifestations, psychosis, dystonia and even intractable seizures. The diagnosis is often delayed or missed if physicians are not aware of this disorder. We report a case of a young girl with epileptic seizure and dystonia.

**Case:** A 19-year-old girl complained of recurrent transient motion arrest without altered consciousness was transferred to our hospital. Because she denied altered consciousness, first impression was psychogenic non-epileptic seizure. For differential diagnosis, video-EEG monitoring was done. Three times of ictal event were recorded. All attacks showed partial seizures with altered consciousness. She was treated with trileptal and seizure was well controlled for 1 year with anticonvulsant. After that time, dystonic movements of extremities and dysarthria occurred for several seconds to minutes without altered consciousness. The video-EEG monitoring was retried. and showed several simple dystonic movement of extremities without altered consciousness. Her vital signs were within normal range. Physical examination was unremarkable. Laboratory tests showed a serum ionized calcium level of 0.51 mmol/L (normal, 1.13-1.32 mg/dL), serum phosphate level of 8.0 mg/dL (normal, 2.50-4.50 mg/dL) and serum magnesium level of 0.9 mg/ dL(normal, 1.5~2.7 mg/dL). Renal function tests, serum sodium, potassium, plasma cortisol, and thyroid function tests were within normal range. Serum PTH level was below 0.1 pg/mL (normal, 12.72 pg/mL). The MRI of brain revealed normal findings. The patient was treated with calcium gluconate intravenously and oral vitamin D. After calcium supply, she maintained no more dystonic movement and seizure.

#### p119

Clinical significance of epileptic aura in patients with epileptic surgery

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**Purpose:** Epileptic aura in isolation corresponds to a simple partial sensory seizure and reflects activation of functional cortex by a circumscribed seizure discharge. We evaluate the localization and prognostic value of epileptic aura in patient with epileptic surgery. We tried to determine whether stimulation-induced aura (SIA) during brain mapping could help us define the location of the epileptogenic zone which has to be resected to favorable surgical outcome.

**Method:** All patients who had epileptic surgery with preoperative invasive study at Seoul National University Hospital from 1995 to 2008 were recruited. Patients with severe mental retardation or too young patients to explain their subjective symptoms are excluded. Electronic medical records

were retrospectively reviewed.

**Results:** Total 292 patients (300 cases) were recruited. Mean age of patients is 28.8 years old and men are 62.7 %. Temporal lobe epilepsy was the most common type of epilepsy syndrome (48.6 %; medial TLE, 17.3 %; lateral TLE including dual pathology, 31.3 %) and frontal lobe epilepsy was next (26.3 %). Among these patients, about 75 % of patients had epileptic auras. 72.9 % had just one kind of habitual aura, 27.1 % had auras with two or more symptoms presenting simultaneously or independently. Psychic, emotional and epigastric aura was the most common type of aura and only one patient had sexual aura. About 63.3 % of patients had SIA. The most common SIA was visual type (22.2 %) and second common type was somatosensory and psychic aura. Detailed clinical parameters and correlation studies are presented in the following conference.

**Conclusion:** Epileptic aura is common in focal epilepsy. In spite there are various new diagnostic tools to help us to localize an epileptogenic zone, thorough history taking about aura is still important and evaluation of SIA has clinical significance.

## p120

#### Seizure semiology in temporal lobe epilepsy

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Purpose: To describe seizure pattern in people with TLE.

**Method:** A descriptive cross sectional study in people with TLE attending the EEG Clinic at Cipto Mangunkusumo Hospital, Jakarta in the period of August 2010 - January 2011. Epileptic syndrome was determined from the seizure semiology and epileptiform focus on EEG recordings. Seizure semiology were obtained from anamnesis. Seizure types were based on 1981 ILAE classification. MTLE were defined by epileptic focus in anterior temporal whereas lateral TLE (LTLE) by a focus in medial and posterior. All datas were collected from medical records

**Results:** From 48 people with TLE, 36 (75%) are MTLE and 12 (25%) are LTLE. History of febrile seizure were found in 14 (38,9%) with MTLE and only 1 (3,8%) with LTLE. Twenty nine (80,6%) of people with MTLE experienced aura, which evolve into secondary generalized seizure in 21 (78,4%). Only a third (33,3%) of those 21 exhibit head turning. Seven (24,1%) MTLE with aura evolve to partial complex seizure with more than half (57,1%) experienced aura, and all of them evolve to secondary generalized seizure. Post ictal state were experienced in 31 (86,6%) MTLE and 8 (66,7%) LTLE. The most common auras reported in both MTLE and LTLE are headache, epigastric discomfort and subjective fear

**Conclusion:** Most people with TLE reported auras which evolve into secondary generalized seizure and almost all experienced post-ictal state. The most common auras are headache, epigastric discomfort and subjective fear. More than half of the MTLE which evolve into complex partial seizure exhibit oro-alimentary automatism. Typical TLE ictal manifestation such as head turning happened only in MTLE.

### Social issues

## p121

## Relationship between stigma about epilepsy and the attitude towards people with epilepsy in university nursing students

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**Purpose:** The attitudes towards people with epilepsy (PWE) have a wide-ranging influence. The aim of this study was to examine the relationship between the perception of stigma attached to epilepsy and the attitudes towards PWE in university nursing students.

**Method:** A 48-item questionnaire on knowledge, attitudes regarding epilepsy and PWE and the stigma scale of epilepsy (SSE) (Fernandes PT et al. 2007) was distributed to nursing students in Tottori University in 2010, and a total of 293 copies were collected. The relationship between

knowledge of epilepsy, attitudes towards PWE and SSE score was statistically evaluated. **Results:** Of the respondents, 79.9% had heard epilepsy, 51.9% had read/seen something in the epilepsy, 12.3% knew someone with epilepsy, and 14.3% had witnessed a seizure. About 37% knew the treatment for seizure attack, and 62.4% correctly identified epilepsy as a nervous system disorder, while 55.0% attended the lecture about epilepsy. Of the respondents, 42.9% would not allow their children to marry someone with epilepsy, 35.5% objected to having their children play with PWE. While 26.6% would offer PWE equal employment, only 1.4% was against it. With respect to the epilepsy-related knowledge, only personal acquaintance with PWE reduced SSE score. The attitudes towards PWE were significantly related SSE scores; students with favorable attitudes towards PWE. **Conclusion:** The findings suggest that epilepsy stigma perception might influence the attitudes towards PWE. An educational program is necessary to improve the understanding of epilepsy and attitudes towards PWE.

## p122

## Referral patterns of epileptic patients to the epilepsy center in Korea urban area LEE JH1, HWANG SH2, PARK JH3

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**Purpose:** We investigated the referral patterns of epileptic patients in National Health Insurance Corporation IIsan Hospital Clinic and their social variables, in order to formulate the optimal role of tertiary epilepsy program.

**Method:** From July 2007 to July 2010, 271 patients were serially registered to National Health Insurance Corporation IIsan Hospital Epilepsy Clinic for the first time in their lives. The contents of epilepsy registry were reviewed and analyzed.

**Results:** 1) Referral Route: self referral through mass media was 39.2% and physician referral was 60.8% (primary physician 32.7%, psychiatrist 13.4%, neurologist 10.6%, rose club 4.1%) 2) Majority of the patients (88.2%) was seeking for the better management of longstanding epilepsy, whereas 11.8% was for initial diagnostic issue. 3) Duration of illness before the referral was less than 1 year 8.2%, 1 to 5 years 28.7%, 5 to 10 years 20.4%, 10 to 20 years 30.2%, over 20 years 12.5% 4) Age at the registration was below 10 7.7%, 10 to 20 26.6%, 20 to 30 38%, 30 to 40 19.7% over 40 8.2% 5) Tentative variables such as seizure type, frequency, education, rural vs urban living and job occupancy were not correlated with referral patterns.

**Conclusion:** At the present time, a tertiary epilepsy center confronts a variety of heterogenous patient population, with wide clinical spectrums, which renders the formulation of specific task very difficult.

#### p123

### How to improve quality of life people with epilepsy in I-San of Thailand

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**Purpose:** I-San or Northeastern part of Thailand has a population of 22 million people. Most have a low level of education and poverty. There are approximately 147,400 people with epilepsy (PWE) in I-San. Most patients do not have access to appropriate treatment due to a lack of any expert in epilepsy or medical facility for epilepsy.

**Method:** We did epilepsy research regarding patients' and public knowledge on epilepsy, medical facility for epilepsy service in I-San. Then, we developed local clinical practice guideline (CPG), which based on results of research.

**Results:** 1. Patients' and public knowledge on epilepsy. We found both PWE and the public have a poor level of knowledge, attitude and practice in epilepsy. Some incorrect concepts include : putting materials into patient's mouth to prevent tongue biting (75% of surveyed subjects), chest compression (50%), or stretching patient's legs and arms (50%) during an epileptic attack, taking antiepileptic medication only during an epileptic attack (10%), and the belief that eating pork causes epilepsy (12%).2. Medical facility for epilepsy service. Most PWE are treated at community

hospitals with general practice physicians who lack of experience, knowledge, and confidence. This phenomenon leads to the inappropriate referral to provincial or university hospitals. As a result, patients need to pay for transportation or leave their work and their relatives need to take time of work. To improve knowledge on epilepsy, several strategies should be employed including a continuous exhibition or mass media to educate students, teachers, parents, and public. Books(picture), brochures, poster, or calendar can enhance knowledge. When people look at the calendar, it could be a reminder about epilepsy particularly if the calendar has a picture of the beloved King of Thailand. An epilepsy network should be developed for improving equally quality of service.

**Conclusion:** We hope that the problems of epilepsy in I-San will be solved in a systematic manner, as outlined above, to achieve the goal of a good quality of life of PWE.

#### p124

## Characteristics of patients with epilepsy who use a web-based health providing program of epilepsy, 'Epilia'

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**Purpose:** This study was performed to describe characteristics of online and offline epilepsy patients and to find out how 'Epilia', an epilepsy web site since 2003, (http://www.epilia.net), can help epilepsy patients.

**Method:** We recruited 367 patients from epilepsy clinics and 153 epilepsy patients from 'Epilia'. The patients were asked of their social, epilepsy, and psychological characteristics as well as attitude alteration after beginning to use 'Epilia'. Among online group patients, we tried to identify factors which affected attitude alteration. Student's t-test, Mann-Whitney test, and chi-square test were used, where appropriate.

**Results:** Age and sex did not differ statistically between two groups. Online and offline groups showed statistically significant differences in the following scales: Liverpool Seizure Severity Scale  $(34.8\pm21.2 \text{ vs. } 45.3\pm20.9, \text{ p} < 0.001)$ , Liverpool Adverse Events Profile  $(39.6\pm11.0 \text{ vs. } 44.3\pm9.8, \text{ p} = 0.017)$ , 36-Item Short Form (146.3 vs. 111.0, p < 0.001), Quality of Life in Epilepsy Inventory-10 (1.8 vs. 2.7, p < 0.001), and Hospital Anxiety and Depression Scale (13.0 vs. 18.0, p < 0.001). Attitude alteration was influenced by frequency of visit to 'Epilia', average amount of time spent on 'Epilia' per visit, and the presence of provocation factors such as alcohol, sleep deprivation, and drug discontinuance.

**Conclusion:** Online epilepsy patients had more severe epilepsy, more adverse effects of antiepileptic drugs, and worse general health, hence more severe depression and worse quality of life, compared to the offline epilepsy patients. 'Epilia' may contribute to self-management by providing information and education for epilepsy patients with these characteristics.

#### p125

#### Epilepsy Action clinics within the acute hospital setting YOUNG M

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**Purpose:** To describe the patient benefits of being able to access Epilepsy Action Australia community based epilepsy nurses in the hospital clinic setting.

**Method:** Formal collaborative partnership between Epilepsy Action Australia and hospital based epilepsy clinics, providing the services of community based epilepsy nurses within the acute care hospital setting. The initial patient/epilepsy nurse contact within clinic setting includes an informed consent, provision of an information pack and needs assessment. A comprehensive needs assessment is completed in the community setting with further tailored services offered to meet the individual needs identified. Post service evaluations are completed and independently evaluated and reported.

**Results:** 100 number of patients have accessed 4 clinics based in Sydney over the last 6 months. The age ranges from 3 months old to 82 years old.

The types of epilepsy seen range from focal epilepsy to generalised and complex epilepsy syndromes. The types of Contact seen range from one service contact, two service contacts and three or more services. The breakdown of services provided include but are not exclusive to: information, seizure diary, Education, home visit, peer support referral, lifestyle workshop referral. Post service Survey results indicate support in the community they were not aware of, increased knowledge, ease of accessing service, reduced anxiety stress to name just a few.

**Conclusion:** The inclusion of community based epilepsy nurses in Hospital based clinics working collaboratively with neurologists has proven to be beneficial to people with epilepsy in bridging the service gap between the acute care and community setting.

#### Status epilepticus

### p126

Treatment of status epilepticus following Glufosinate Ammonium intoxication: a case report  $\underline{CHANG}$  H, CHEONG J, LEE H

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**Background:** Glufosinate ammonium(GLA, BASTA), a non-selective herbicide, is widely used in many countries including Korea. The neurological complications of GLA intoxication are manifested as a loss of consciousness, convulsion, or memory impairment.

**Case:** We encountered a 59-year-old male, who was presented to the emergency department 1 hour after orally ingesting GLA. The patient developed mental disturbances, impaired respiration and generalized tonic-chronic seizures. Although the patient was initially treated with lorazepam and phenytoin, the seizures continued to occur. Through continuous infusion of midazolam along with respiratory support, the occurrence of the seizures was controlled. The patient was discharged with no medical and neurological abnormalities except for a short-term memory loss.

**Conclusion:** We report a case, status epilpeticus following Glufosinate Ammonium intoxication.

### p127

#### Resective surgery in status epilepticus: report of three cases

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**Purpose:** In selected cases with a clearly definable radiological lesion and/or electrophysiological evidence of a focal onset, emergency surgical resection is a treatment option for refractory status epilepticus (RSE).

Materials and methods: Retrospective review of case records.

**Results: Case 1:** An 18-year-old girl a known case of drug resistant right focal epilepsy (15-20 per day) with occasional secondary generalization (SG) since the age of 12 years was admitted in CSE, failed to multiple AEDs and midazolam infusion for 5 days. Investigation revealed left frontal focal cortical dysplasia (FCD) and ictal video-EEG showed left anterior frontal epileptogenic zone. RCSE remitted with resective surgery of the lesion, FCD Type IIb.

**Case 2:** A 6-year-old boy a known case of drug resistant epilepsy, multiple attacks of asymmentrical spasm in a day since the age of 3 years presented with altered mental status with subtle myoclonic jerks with in between periods of relatively preserved mental state but irritable of 3 weeks duration. Video-EEG showed disorganized background rhythm with frequent generalized spike- and sharpwave activity, suggestive of NCSE. MRI showed left fronto-temporal FCD. NCSE remitted with resective surgery of the lesion, FCD Type IIa.

**Case 3:** A 37-year-old female, a known case of drug resistance left focal motor seizures with SG with five episodes of CSE, was admitted in CSE which failed midazolam infusion for 6 days. Ictal SPECT showed right frontal hyperperfusion and ictal video-EEG showed right anterior frontal epileptogenic zone. RCSE remitted with right anterior frontal lobe resection, FCD IIa. **Conclusion:** In patients with a definable epileptogenic zone, resective surgery remits RSE.

## Short-term oral or rectal diazepam at bedtime for clinical non-convulsive status epilepticus in remote areas

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Purpose: Non-convulsive status epilepticus (NCSE) is confirmed by EEG and treated by intravenous antiepileptic drugs (AEDs) or long-term oral AEDs, and its termination is confirmed by EEG. The patients in remote areas, however, cannot reach either EEG or such treatment promptly. We developed a new treatment for clinical NCSE (C-NCSE) for the patients in remote areas. Method: C-NCSE was defined as the condition that the patients had unusual daily activities or seizures, including decreased response or activity and frequent minor seizures, lasting for several days. When C-NCSE was reported by telephone call, DZP, 0.3-0.5mg/kg, prescribed beforehand, was ordered to administer the patient orally or rectally at bedtime for 4 or 5 days, and to repeat it after one week interval when the previous condition was not restored by the first treatment. A total of 36 episodes were treated with DZP in 17 patients with 1 and 6 episodes of C-NCSE, consisted of 6 patients with Lennox-Gastaut syndrome, 4 with symptomatic frontal lobe epilepsy, 4 with symptomatic generalized epilepsy, and 3 with other epileptic syndromes, aged 5 and 33 years at C-NCSE. They had been followed up for 3 and 25 years and their usual seizure status, daily life and activities were well known by the authors. NCSE was confirmed by EEG before DZP therapy in 13 episodes in 9 cases. When the second treatment failed, the patients visited our outpatient clinic and were treated with intravenous midazolam or other medicines.

**Results:** C·NCSE was terminated by the first treatment in 19 episodes and by the second trial in 11 episodes, and 6 episodes did not respond to this therapy. Somnolence and ataxia occurred in 3 episodes at the second 5-day treatment in non-responders, but other side effects were not seen. Termination of NCSE was confirmed by EEG in 4 episodes.

**Conclusion:** Short-term oral or rectal diazepam at bedtime is easy, effective and safe for C-NCSE, particularly for patients in remote areas.

#### p129

## Electroclinical and neuroimaging features in patients with aphasic status epilepticus

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**Purpose:** Aphasia is very uncommon as sole manifestation of status epilepticus (SE). Early identification of aphasic SE is clinically important because it commonly misleads false diagnosis. We report clinical, electroencephalography (EEG) features, and neuroimaging findings of aphasic SE. **Method:** Fourteen patients who presented aphasia as sole or most predominant symptom of SE in a single epilepsy center between Feb. 2001 and May 2011 were included. Ictal EEG and MRI were obtained in all and ictal single photon emission computed tomographys (SPECT) were done in selective patients.

**Results:** Six patients presented global aphasia, one patient showed motor type, and the others showed sensory type. Diffusion-weighted imaging failed to reveal any significant changes in all except two patients showed fluid restriction in the left hemisphere. Fluid-attenuated inversion recovery MRI showed newly developed, left hemispheric cortical hyperintense lesions in five patients. Ictal EEG showed continuous widely-spread left hemispheric slowing in five out of six patients with global aphasia. Periodic lateralizing epileptiform discharges (PLEDs) of the left hemisphere were found predominantly in patients with non-global type aphasia. There was no definite classic evolving feature of ictal EEG in all. Ictal SPECT disclosed hyperperfusion in the left hemisphere in all seven patients. The aphasic symptom was gradually improved over several days (mean  $\pm$  SD = 6.83  $\pm$  6.16) after antiepileptic drug treatment in all except two patients who had improved over several months.

**Conclusion:** Atypical ictal EEG pattern commonly occurs, and MRI is negative in majority of the patients with aphasic status. Diffuse hemispheric slowing is more common in global aphasia, but the presence of PLEDs suggests more restrictive pattern of aphasia. Hyperperfusion on ictal SPECT and the gradual clinical improvement are outstanding features of aphasic status epilepticus.

## EEG

#### p130 The EEG findings in sleeping sickness <u>CHAN J</u>, TUCH P, LAWN N, DUNNE J Royal Perth Hospital, Neurology, Perth, Australia

**Purpose:** To describe the EEG findings in a patient with Human African Trypanosomiasis (HAT) during the phase of cerebral involvement.

**Result**: A 19-year-old Sudanese-born Ugandan refugee presented with an 8-month history of progressive hypersomnolence, weight loss, myoclonus, pruritic rash, cervical lymphadenopathy and extrapyramidal features. EEG showed generalised semi-periodic complexes during apparent wakefulness and without clinical accompaniment. These complexes comprised an admixture of high amplitude anteriorly dominant delta waves with a periodicity of 3-8 seconds. The EEG transition from wakefulness to sleep was ill defined and normal sleep architecture was lost. Cranial CT was normal, and MRI scan showed bilateral symmetrical diffuse white matter hyperintensity. CSF examination showed raised protein and mild mononuclear pleocytosis. The diagnosis of Human African Trypanosomiasis Gambiense was confirmed by strongly positive serology in both serum and CSF. The patient received a 14-day course treatment with Effornithine and made an uneventful recovery. Follow up EEG showed resolution of the periodic complexes but moderate generalised nonspecific slowing.

**Conclusion:** HAT, like SSPE, can produce the EEG pattern of generalised semi-periodic complexes with long inter-complex intervals. It should be considered in patients who come from endemic African regions.

### Surgery

### p131

## Wada test verbal memory results of patients with temporal lobe epilepsy (TLE) and their 3-month postoperative verbal memory indices

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**Purpose:** We investigated whether Wada test findings of verbal memory in patients with TLE are related to their 3-month postoperative memory indices.

**Method:** We included 12 postoperatively TLE patients (7 patients, right; 5, left) who were preoperatively determined to be left language dominancy by Wada test. Pre-testing verbal index (CV = number of correct answers/number of verbal questions) was calculated as controls. After 10mg of propofol injection into each carotid artery, the right- and left-side verbal memory indices (RV and LV) were determined. These indices were divided by those of the control, and the RV/CV and LV/ CV were used as representative values of the Wada test for memory. WMSR tests were performed before and 3 months after the operation. The Verbal Memory index (WMSRv) that is one of the WMSR indices was obtained by dividing postoperative verbal value with preoperative one. **Results:** The Wada test verbal memory indices of the left TLE group were RV/CV =  $1.14 \pm$ 0.21 and LV/CV =  $0.66 \pm 0.44$  while those of the left TLE were RV/CV =  $0.90 \pm 0.62$  and LV/CV =  $0.33 \pm 0.12$ . WMSRv of the right TLE group, verbal verbal memory indices tended to increase compared to that of the controls, while after left injection in left TLE group, these indices tended to decrease. Since verbal memory tends to decrease postoperatively in patients with left TLE, it may be reflected in these results.

## Vagus Nerve Stimulation (VNS) implant surgery: operative technique assessment in a single surgeon implant series

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**Purpose:** Since its introduction VNS has established itself as a valid therapeutic option for seizure (SZ) management. Successful VNS therapy depends not only upon correct epileptic diagnosis but also upon surgical expertise because implant technique is often not straightforward. We endeavored to review the procedural details of the VNS implant cases performed at a single institution by the presenting author (NR), focusing on technically difficult situations.

**Methods:** Data were collected on all VNS patients in a concurrent and prospective fashion from 2002 through 2011. This was reviewed for relevant information from a clinical and imaging standpoint and their relation to intra-operative findings as well as reasons for revision surgery. **Results:** A primary VNS implant for SZ's was performed in 41 patients (17F/24M) of which 28 were Paediatric age (10F/18M), whereas depression was the diagnosis in one adult woman. In addition we revised a VNS in 3 adult epileptic men previously implanted at another institution. Paediatric revisions were done for infection (N=2) and for dislodged electrode wires and/or pulse generators

(N=3). One adult patient posed particularly challenging surgical revision conditions because of a cervicothoracic hyperkyphosis-related fixed chin-on-chest deformity. From a SZ management standpoint SZ frequency was markedly reduced in almost all patients, except one. Despite this encouraging result, only one patient achieved actual SZ freedom without anti-epileptic drug (AED) use. Several of our patients were tiny and very young (14 months for the youngest), requiring technical adjustments to the actual surgical procedure to adequately expose the vagus nerve and fashion the stimulator pocket. Anatomical features will be demonstrated to clarify these procedural considerations.

**Conclusions:** Our surgical implant series shows that even difficult situations such as can be encountered in very small children and in the presence of cervicothoracic spinal deformity can be handled well by appropriate adaptation of surgical technique and careful attention to the individual challenges of each case. This results in good surgical outcomes with correspondingly improved SZ management.

#### p133

# Surgical treatment for epilepsy induced by angiocentric glioma and literature review (analysis of 2 cases)

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**Purpose:** Study clinical, electrophysiological, imaging and pathological features of angiocentric glioma (AG), discuss the diagnosis, treatment and prognosis.

**Method:** Reviewed retrospectively 2 cases AG of associated with epilepsy through surgical treatment. Two cases of AG patients were male, aged 14 to 22 years, an average of 18 years of age. Early age of onset of epilepsy in 9-13 years, mean 11 years old. Initial symptoms are seizures. According to the different parts of the lesion, we took different surgical approaches. A routine take left frontotemporal craniotomy and medial temporal lobe structures plus the former lesion resection. Another case took the right temporal-parietal-occipital craniotomy, temporal lobe lesions Canada epileptogenic resection in the navigation beause MRI shows abnormal signal naked eye can not distinguish. Patients in both the downlink in the light anesthesia state cortical EEG monitoring. **Results:** Pathological diagnosis was AG. Postoperative there was no any complications. After 6 to 12 months of follow-up, two cases of seizures in patients with AG were completely disappear (Enge I class).

**Conclusion:** Diagnosis of AG depends on pathological examination which tumor cells form rosettes around the blood vessel-like structures and EMA-positive spots are its unique histological features. Electron microscopy of tumor tissue to determine the source and the differential diagnosis is great significance. Complete surgical resection without chemotherapy, and can effectively control the seizures.

# Definitive surgical treatment of hypothalamic hamartoma based on 23 consecutive surgical experiences of hamartomas

<u>HÔRI T</u>

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**Purpose:** Hypothalamic hamartoma (HH) with gelastic seizure is medically intractable, and is surgically also very difficult to cure. There are at least three options to manage HH surgically, that is, thermocoagulation (4), Gamma-Knife treatment (10), and surgical excision (9). Concerning thermocoagulation, very good cure rate has been reported recently, but in reality, among other institutions doing the same procedure, cure rate is not satisfactory. Gamma-Knife is also effective, but not perfect in its results. Open surgical excision is sometimes very effective, but is often complicated with hypothalamic dysfunction related to surgical manipulation. Based on the treatments of these three methods for 23 HH patients, the authors finally established a definitive surgical procedure for two HH patients with immediate postoperative cure of the intractable seizures. Here, we will report the surgical technique and operative results.

**Method:** One 10-y-old boy afflicted with gelastic seizure and precocious puberty and one girl of 14-y-old reoperated after 10 years since the first operation for HH with arachnoid cyst are patients managed with this definitive surgical treatment. Anterior interhemispheric trans lamina terminalis approach has been adopted. After opening the lamina terminalis HH is exposed. The interface of hamartoma with hypothalamic wall is identified, and the interface has been transected sharply resulting nearly total removal of the hamartoma. Anterior interface of the tumor hidden by optic chiasm has been left in place, but tumor tissue is enulcleated as much as possible.

**Results:** Immediately after the operation, intractable seizures has stopped without any complications in these two patients, and they have shown Improvement of their cognitive function postoperatively.

**Conclusion:** Nearly total removal of HH and transection of the tumor from hypothalamic wall is reasonable surgical treatment without surgical complications.

#### p135

## Long-term seizure and cognitive outcomes after surgical treatment of therapy-resistant temporal lobe epilepsy

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**Purpose:** The aim of the present study was to analyze a long term seizure and cognitive outcomes following epilepsy surgery for therapy-resistant temporal lobe epilepsy (TLE) and to identify predictors of success or failure after resection.

**Methods:** We retrospectively reviewed the medical records of 43 patients who underwent temporal lobe surgery for therapy-resistant epilepsy after comprehensive presurgical evaluation at the Severance Children's Hospital between 2003 and 2011. Histopathologies, postoperative seizure outcome, changes in seizure outcome over time, complications and cognitive outcomes were studied. We also analyzed the clinical characteristics of patients who failed epilepsy surgery. Postoperative seizures outcomes were evaluated by Engel's classification.

**Results:** Twenty-one patients underwent left temporal resection and twenty-two patients underwent right temporal resection. Anterior temporal lobectomy was performed in 88.4% of patients, whereas in 7% underwent lesionectomy and 4.6%. cortisectomy. The duration of follow-up ( $\pm$ SD), was 6 months to 8 years (mean 3.4 years;  $\pm$ 2.1 years). Number of Engel class I patients was 37 (88.1%) after 6 months; 34 (83.2%) after 1 year; 14 (74.1%) after 5 years; and 5 (61.8%) more than 6 years of follow-up. Twenty (90.9%) of 22 patients with low-grade brain tumors, cavernous angioma or mesial temporal sclerosis achieved Engel class I outcomes in contrast to 12 (60%) of 20 patients with other pathologies at one year follow-up (> 0.05). Patients with seizure-free rate have showed positive changes in verbal and full scale intelligence quotients.

**Conclusion:** Epilepsy surgery is safe and effective approach for the treatment of therapy resistant TLE with positive effects on cognitive function.

#### Insular epilepsy surgery under neuronavigation guidance using depth electrodes

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**Purpose:** The insular cortex originated seizures can be confused with frontal, temporal or frontotemporal lobe seizures because of its hidden location. Subdural electrodes or depth electrodes inserted stereotactically, perpendicular to the midsagittal plane by transopercular route can be used but only for limited monitoring of the wide insular cortex. Therefore, we have tried various insular monitoring devices including open direct subpial depth electodes under navigation guidance to increase accuracy of epileptogenic zone and surgical outcome as a result.

**Method:** From March 2005 to December 2010, we operated on 6 consecutive patients with possible epileptogenic zones within the insula. The series contain equal number of male and female, with mean age of 50 months. All patients had medically intractable seizures and evaluated with video scalp EEG. Thorough preoperative work up was done. Postoperative MRI was done within 48 hours to confirm the location of grid and the extent of resection.

**Results:** Navigation guidance subpial depth insertion was done on three cases and subpial strip insertion was done on one case. Stereotactic depth insertion was done on two cases. Total or partial insulectomy were done in all cases and subpial depth or strip insertion resulted in Engel Class I outcome. Stereotactic depth insertion resulted 50% Engel Class I result

**Conclusion:** Based on our short series, navigation guided subpial depth or strip insertion provides precise monitoring of seizure location in insular and serves as good surgical landmark to result in better surgical outcome.

#### p137

## Minimum requirement and how to improve results of epilepsy surgery in limited resources $\underline{\text{MUTTAQIN } Z}$

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**Purpose:** Epilepsy Surgery (ES) is still under utilized in many parts of Asia, including Indonesia. With 0.5% prevalence, there are about 2 million epileptic in Indonesia and about 300.000 are potential candidates for ES. Number of ES increases every year reaching 40-50 cases per year in 2007-20010. The minimum requirement for patient's selection and ways to improve results of ES in limited resources are discussed

**Method:** Until Dec. 2010, there were 272 ES cases, including 244 temporal lobectomies. To evaluate patient's selection, we divide the ES cases into the first 5 years (56 cases) and the recent 5 years (216 cases). But for evaluating surgical results, only those with at least 48 months follow-up were included (106 cases) and grouped according to age and length of epilepsy at surgery **Results:** Besides semiology, MRI and routine EEG play decisive role for ES in 54 out of 56 (96.5%) TLE cases during the first 5 years, but this role decreased to 50% during the last 5 years. It means that other difficult presurgical evaluations were needed, such as long-term ictal EEG in 56, subdural grid EEG in 11, PET study in 12, and EcoG in 2 patients. The overall seizure free (SF) rate were 70.75%, but if grouped according to patient's age at surgery (less than or over 25 Y-old), the SF rates were 75.4% vs 66.04% respectively. So did if grouped according to length of disease (less than or more than 10 years), the SF rates were 78.72% vs 64.40% respectively

**Conclusion:** Besides semiology, Decreasing role of MRI during the last 5 years means that other presurgical work up is needed to improve ES results, especially for cases with doubtful laterality. SF rate was significantly higher for those who was operated at younger age and those with shorter duration of epilepsy. For a better result, ES should be offered earlier for those intractable TLE patients with obvious focus on MRI.

#### p138

## Analysis of management of neurosurgical patients with recurrent epileptic seizures YUAN G<sup>1</sup>. GAO D<sup>2</sup>. LIN J<sup>2</sup>

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**Purpose:** To investigate the clinical features and management principle and methods of neurosurgical patients with recurrent epileptic seizures .

**Method:** Clinical data of 9 cases with recurrent epileptic seizures were analysed retrospectively who were treated in our department. The analyses include the clinical data, the characteristics and the reasons for the aggravated seizures and the treatment methods and results.

**Results:** Of all the patients, glioma in 3 cases, arachnoid cyst in 1 case, cavernous hemangioma in 1 case, encephalomalacia in 1 case. The reasons for seizure aggravation include reducing drug (3 cases), brain tumor with recent diagnosis (3 cases), surgical operation for intracranial electrode implantation (1 case) and unknown reasons (3 cases). Epileptic seizure types include partial seizure and secondary generalized seizure. The frequency of seizure range from 3 minutes interval to several hours interval. Patients with epilepsy on antiepileptic drugs have a poorer treatment response, show refractory, who were gave multiple antiepileptic drugs--combination therapy, including oral and parenteral administration, the seizures got controlled. Levetiracetam was shown to good curative effect in the treatment.

**Conclusion:** The epilepsy seizure of neurosurgical patients ofen show drug refractory, hard to controlled. The treatment methods used for partial seizure has better curative effect of AEDs with combined therapy which dose should be higher than that of conventional initial dose including intravenous and intramuscular administration so as to control the seizures. Because of Levetiracetam for oral absorption fast, rapid onset of action and better antiepileptic effect, its adding treatment for recurrent seizures has better curative effect.

### p139

### Functional posterior quadrantectomy in paediatric intractable epilepsy

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**Purpose:** Subhemispheric epilepsy involving the posterior quadrant (temporal, parietal and occipital lobes) has been treated with anatomical posterior quadrantectomy and more recently functional posterior quadrantectomy. Periinsular posterior quadrantectomy developed by Daniel et al give solution other than wide respective surgery. We are here to report the result of 4 consecutive quadrantectomy cases.

**Method:** From January 2007 to December 2011, there were four cases of functional hemispherotomy. 3 were male and the mean age was 7.3 years, ranging from 4 years old to 10 years old. All patients suffered from intractable epilepsy and thorough presurgical workup was done. In three cases, functional hemispherotomy was added after frontal lobectomy according to intraoperative monitoring. Postoperative magnetic resonance imaging was done to assess the complete disconnection of posterior quadrant.

**Results:** All patients reached Engel Class I after surgery during at least one year follow-up period. All suffered mild hemiparesis and hemianopsia postoperatively. Three cases with frontal lobectomy done had found to be focal cortical dysplasia on pathology. There were no other surgical complications.

**Conclusion:** Functional posterior quadrantectomy is a safe and effective method to treat wellselected patients with posterior quadrant epilepsy. In addition, it is a useful method to complete disconnection after frontal lobectomy in certain cases.

## p140

Selective callosotomy in paediatric epilepsy under DTI navigation guidance

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**Purpose:** Corpus callosotomy is a palliative surgical procedure to control intractable seizure. However, disconnection syndromes, or split-brain syndrome are most problematic complications after corpus callosotomy, especially children over 10 years old. Therefore, we report two cases of selective callosotomy under intraoperative interhemispheric subdural grid monitoring using diffusion tense imaging magnetic resonance navigation.

Method: Two cases of 11 and 9 year old female patients underwent for selective callosotomy under

navigation guidance with diffusion tensor imaging techniques with motor fiber tracking. Both had intractable seizures involving both hemispheres in preoperative workups. Interhemispheric subdural grid was used and selective callosotomy was carried out under navigation guidance to prevent disconnection syndrome.

**Results:** For 11 year old female patients, after callosotomy from the genu to posteriorly 3cm, bilateral generalized synchrony was disappeared and she is free of generalized type seizure with medications. For 9 year old female, selective callosotomy upto the motor fibers was performed and her traumatic falling disappeared without any disconnection syndrome.

**Conclusion:** Selective corpus callosotomy is effective for seizure control of bilateral generalized synchrony without disconnection syndrome. Selective callosotomy can be considered especially for patients over 10 years old with thorough preoperative monitoring.

#### p141

## Dynamics of epileptic syndrome in patients with cerebral arteriovenous malformation after endovascular intervention

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**Purpose:** to assess the dynamics of epileptic syndrome (ES) after active endovascular embolization of cerebral arteriovenous malformation (AVM) using Hepasphere®Microshperes (BioSphere Medical, USA) loaded with anticonvulsant in combination with cyanoacrylate (Histoacryl®) **Material and methods:** 32 patients with AVM underwent endovascular intervention. The ES manifested with frequent simple partial (n=4) and/or complex partial seizures followed by secondary generalization (n=28). Anticonvulsants were administered perorally over all period of observation and treatment. AVMs were classified by Spetzler-Martin scale: I (n=3), II (n=6), III (n=18) and IV (n=5) grade. The embolization of AVM was carried out by HepaSphere® Microshperes (50-100 µm) with adsorbed anticonvulsants (Diazepam) which were delivered to the nidus of AVM through the feeding artery. After that for the same session embolization of AVM with Hystoacryl® was performed through the same feeding artery. Electroencephalogram (EEG) monitoring was carried out perioperatively.

**Results:** There were 46 embolizations in 32 patients. The operation was carried out for three (n=4), two (n=9) and one (n=16) sessions. We observed positive dynamics of EEG and considerable decrease of seizures' frequency over 2.5 years of postoperative observation. According to Engel's classification of outcomes 22 patients corresponded to class I, 8 · to class II and 2 · to class III. Total exclusion of AVM from circulation was achieved in 11 cases, 50·75% reduction of its size · in 8 cases, less than 50% · in 13 cases. There were no postoperative neurological complications. **Conclusion:** Proposed method owning to the addressed delivery of anticonvulsant directly into the brain and creation of its prolonged depot in the nidus of AVM allows to improve the effectiveness of endovascular treatment of patients with AVM and ES. Using this method of embolization permitted to control the frequency of seizures in more than half of the operated patients.

#### Translational research

#### p142

**Mechanisms of absence epilepsy in a P-type voltage-sensitive Ca2+ channel deficient mice** <u>YAMAMURA S</u><sup>1</sup>, TANAHASHI S<sup>1</sup>, FUKUYAMA K<sup>1</sup>, SAITO H<sup>2</sup>, SUZUKI N<sup>2</sup>, OKADA M<sup>1</sup> <sup>1</sup>Mie University, Division of Neuroscience, Graduate School of Medicine, Tsu, Japan, <sup>2</sup>Mie University Life Science Reserch Center, Department of Animal Genomics, Functional Genomics Institute, Tsu, Japan

**Purpose:** To study the pathogenesis and pathophysiology of absence epilepsy, we determined the transmission abnormalities in the frontal regions of P-type voltage-sensitive Ca2+ channel deficient mice, an established absence epilepsy model, using microdialysis with extreme liquid chromatography(xLC).

**Method:** Male Cacna1a deficient mice were developed and generated as described previously. A dialysis probe was implanted in the mice frontal-cortex. Effects of following agents on frontal extracellular levels of L-glutamate, D-serine and GABA were determined by xLC. The agents used in this study were carbamazepine(CBZ), fluorocitrate(FLC), w-conotoxin-GVIA(GVIA) and w-agatoxin-IVA(IVA).

**Results:** There were no differences of basal extracellular levels of GABA, L-glutamate or D-serine between Cacnala+/+ and Cacnala+/-; however, K+-evoked releases those of Cacnala+/- were larger than those of Cacnala+/+. CBZ produced seizure in Cacnala+/- without affecting behavioural abnormality in Cacnala+/+. Perfusion with FLC, decreased basal extracellular levels of glutamate and D-serine without affecting GABA level. The FLC-induced reduction of D-serine in Cacnala+/- was larger than that of Cacnala+/+. Perfusion with CBZ increased basal extracellular GABA level of Cacnala+/+ without affecting that of Cacnala+/- but decreased basal extracellular D-serine of Cacnala+/- without affecting that of Cacnala+/-. CBZ induced reduction of K+evoked releases of glutamate and D-serine of Cacnala+/+ were larger than those of Cacnala+/-, but that of GABA of Cacnala+/-. was larger in than those of Cacnala+/+.

**Conclusion:** These results suggest that relatively enhanced glial-transmission and inhibitory effects of CBZ on glial-transmission play important roles in respective mechanisms of absence epilepsy and CBZ-induced paradoxical intoxication.

## p143

**Targeting hyperphosphorylated tau with sodium selenate suppresses seizures in rodent models** JONES NC<sup>1</sup>, THANH NGUYENB T<sup>1</sup>, CORCORAN N<sup>1</sup>, VELAKOULIS D<sup>1</sup>, HOVENS C<sup>1</sup>, <u>O'BRIEN TJ</u><sup>2</sup> <sup>1</sup>The University of Melbourne, Parkville, Australia, <sup>2</sup>The University of Melbourne, The Department of Medicine, The Royal Melbourne Hospital, Parkville, Australia

**Purpose:** Tau hyperphosphorylation has been implicated in the pathogenesis of a variety of forms of human epilepsy. Here we investigated whether treatment with sodium selenate, a drug which reduces pathological hyperphosphorylated tau by enhancement of PP2A activity, would inhibit seizures in rodent models.

**Method:** Sodium selenate treatment was then tested against three different rodent seizure models. Firstly the propensity of 6-Hz electrical corneal stimulation to induce seizures in adult mice was assessed following acute treatment with different doses of sodium selenate. Secondly, effect of chronic treatment with sodium selenate in drinking water to rats on the number of seizures induced by pentylenetetrazole (PTZ) i.p. was quantified. Finally, fully amygdala kindled rats were chronically treated with sodium selenate in drinking water and the length and the severity of the seizures evoked by stimulation of the amygdala recorded.

**Results:** A dose-dependent protection of sodium selenate against 6-Hz stimulation induced seizures, and significant reduction in the total number of seizures following PTZ injection, was seen. Amygdala kindled rats chronically treated with sodium selenate had significantly shorter seizure durations compared controls, with more pronounced effects observed as the duration of treatment increased.

**Conclusion:** Targeting hyperphosphorylated tau by treatment with sodium selenate has anti-seizure effects in a broad range of rodent models, and may represent a novel approach to treatment of patients with epilepsy.

#### Women and pregnancy

#### p144

Women with epilepsy and infertility have abnormal reproductive hormonal profile

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**Purpose:** We aimed to compare the hormonal profile of women with epilepsy (WWE) and infertility with that of WWE but no infertility.

**Method:** In the Kerala Registry of Epilepsy and Pregnancy we identified 42 WWE and infertility (failure to conceive even after one year of unprotected intercourse) and compared their hormonal profile with 16 age-matched WWE who had pregnancy. Statistical significance of differences was tested with Unpaired t test (for continuous variables) and chi square test (for proportions).

**Results:** Their epilepsy classification was Generalized epilepsy-22, Localisation related-27. The AEDs used by infertility and fertility groups (in bracket) were carbamazepine 17 (10), valproate 12 (5), clobazam 10 (4) levetiracetam 6 (0) others 12 (5). The mean + SD of various hormone levels for the infertility groups: (values for the fertility group are given in brackets) were : Androstenedione 1.48  $\pm$ 1.26 (1.3  $\pm$ 1.18), Dihydroepiandrosterone (DHEA) 2.15  $\pm$ 1.83(0.98  $\pm$ 0.59), Follicle Stimulating Hormone (FSH) 13.09  $\pm$  26.27 (5.78  $\pm$  3.60), Luteinising Hormone (LH) 26.24  $\pm$ 33.20(5.43  $\pm$ 4.54), Progesterone 4.27  $\pm$ 7.44(9.77  $\pm$ 13.27), Prolactin 23.32  $\pm$  39.84(12.99  $\pm$ 7.11), Testosterone 0.58  $\pm$  0.49(1.17  $\pm$  2.78), LH/FSH Ratio 2.54  $\pm$  2.44(0.96  $\pm$  0.78). The infertility group had significantly higher levels of DHEA (p = 0.016), LH (p = .016) and LH/FSH ratio >2.

**Conclusion:** The results indicate that WWE and infertility have significantly higher risk of abnormal reproductive hormone profile compared to WWE and fertility.

#### Late submissions

## p145

## Brain and blood miRNA expression profile of refractory temporal lobe epilepsy

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**Purpose:** MicroRNAs (miRNAs) are a class of small cytoplasmic RNAs that regulate the expression of other genes. Although miRNAs was found expressed extensively in the central nervous system and blood in physiological and pathological conditions, few studies have yet specifically examined miRNAs in hippocampal and blood of epilepsy patient. To find the role of miRNA in the molecular mechanisms of refractory temporal lobe epilepsy (TLE), we investigated the changes of its expression in brain tissue and blood of TLE patients.

**Method:** 10 TLE patients who got brain surgery treatment and 6 normal brain tissues from human brain storeroom with their blood (20~40 years) were brought into this study. The removed hippocampus of them were stored into liquid nitrogen until use. Total RNA of brain tissue and blood were isolated, and Affymetrix miRNAs array chip was used to detect TLE and control samples. The ratio of miRNAs expression of TLE group to normal group >2 was considered as exhibiting upregulated expression, and ratio < 0.5 was identified as downregulated expression. To verify the accuracy of microarray results, the difference miRNAs expression in both hippocampus and blood were selected for further confirmation using qRT-PCR.

**Results:** MiRNAs array chip analysis results showed that near to 50 miRNAs were expressed differentially in the hippocampus and blood of refractory epilepsy patients compared to normal human including downregulated and upregulated miRNAs. Furthermore, miR·455 was detected significantly upregulated simultaneously in both hippocampus and blood of TLE group compared to the control group, and further confirmed by qRT-PCR.

**Conclusion:** Our research found miR-455 to be differentially expressed in the hippocampus and blood of TLE, which demonstrates that TLE may alters the expression levels of a subset of miRNAs in the hippocampus and blood. Exploring the target genes and pathways of miR-455 and other differentially expressed miRNAs will be the next step which may contribute to understanding the role ofmiRNAs in the epileptogenesis.

## p146

Changes in structural and functional connectivity of the hippocampus-extratemporal networks in unilateral temporal lobe epilepsy with hippocampal sclerosis: a combined structural and functional MRI study

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**Purpose:** We studied changes in structural and functional connectivity between the epileptic hippocampus and extratemporal networks in mesial temporal lobe epilepsy associated with

hippocampal sclerosis (HS·MTLE), by using structural MRI, diffusion tensor MRI (DTI), and seed-based resting-state functional MRI (Rs-fMRI).

**Method:** 22 patients with unilateral HS-MTLE (12 left, 10 right HS) and 26 controls were examined on a 3T MR scanner by using the following sequences: (1) 3D volumetric T1-weighted images, (2) DTI (30 non-collinear directions), and (3) Rs-fMRI (245 EPI). Regionally specific differences in grey matter volume between the groups were assessed by VBM. Differences in white matter integrity between the groups were examined by tract-based spatial statistics (TBSS) analysis of fractional anisotropy (FA) and mean diffusivity (MD) images. Changes in functional connectivity of the pathologic hippocampus were evaluated by correlations between the seed (ipsilateral hippocampus) and other regions using Rs-fMRI analysis.

**Results:** VBM showed that both HS-MTLE patients had significant grey matter volume reductions in ipsilateral hippocampus, adjacent temporal lobe, insular cortex, and bilateral thalamus and caudate nucleus (corrected P< 0.05). TBSS analysis showed that both HS-MTLE patients had significant FA decreases and MD increases in ipsilateral temporal white matter, internal and external capsules, corpus callosum, anterior cingulum, and bilateral anterior thalamus and frontal white matter (TFCE-corrected P< 0.05). Compared to controls, left HS-MTLE patients showed decreased functional connectivity between the ipsilateral hippocampus and anterior cingulate, posterior cingulate, and left middle temporal gyrus (corrected P< 0.01). Compared to controls, right HS-MTLE patients showed decreased functional connectivity between the ipsilateral hippocampus and anterior cingulate, posterior cingulate, and regulate, posterior cingulate, and right middle and superior temporal gyrus.

**Conclusion:** Our results showed that HS·MTLE is associated with widespread pathological changes in structural and functional connectivity between the epileptic hippocampus and extratemporal structures, supporting the hypothesis that regional brain abnormalities in HS·MTLE exist not only in the hippocampus and temporal lobe ipsilateral to the epileptic focus, but also in the extensive extratemporal structures.

#### p147

# Predictable factors and timing of seizure recurrence after tapering and withdrawal of antiepileptic drug in epileptic patients with monotherapy

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**Purpose:** To evaluate predictable factors and timing of seizure recurrence after withdrawal of antiepileptic drug (AED) in patients with monotherapy.

**Method:** We had tapered and then withdrew AED for 3~4 months in 90 epileptic patients with monotherapy, who had been seizure-free for more than 3 years. We evaluated the timing of seizure recurrence and predictable factors for seizure recurrence such as type of seizures, number of seizures before medication, initial EEG findings, initial MRI findings, and physical fatigue etc. during AED tapering and after AED withdrawal during the 3 years follow up,

**Results:** Seizure recurrence during AED tapering and after AED withdrawal period occurred in 37(41%) epileptic patients during the 3 years follow up. Seizure recurrence rate during tapering period was 16%. Seizure recurrence rate were occurred 16% within 1 month, 27% within 6 months, 14% within 6 months ~ 1 year, 11% within 1 year ~ 2 year, 16% within 2 year ~ 3 year after AED withdrawal. Among predictable factors for seizure recurrence, abnormal MRI findings was statistically significant. But number of seizure before medication and seizure free duration were not significant associated with relapse.

**Conclusion:** Timing of seizure recurrence is most common within 1 year, especially within 6 months. With relatively high recurrence rate within 2 year  $\sim$  3 year after AED withdrawal, recurrence rate after withdrawal of AED in patients with monotherapy might be at least 40-50%. Although abnormal brain MRI finding had statistical significance to seizure relapse, I cannot confirm whether all of the abnormal brain MRI findings can be epileptic foci or not, so further consideration will be needed.

### p148

#### Long-term outcome of multiple hippocampal transection for temporal lobe epilepsy

KAWALK, USAMI K, KUBOTA M, SAITO N The University of Tokyo, Neurosurgery, Tokyo, Japan **Purpose:** Multiple hippocampal transection (MHT) is a novel surgical treatment for medial temporal epileptic focus developed to preserve memory function as a hippocampal counterpart to multiple subpial transection for neocortical foci. We present its long-term seizure outcome and cognitive outcome.

Method: 24 patients followed longer than 4 years after the surgery for temporal lobe epilepsy were evaluated. MHT was indicated when there was no apparent hippocampal atrophy and preoperative memory was better than subnormal level, thus in case that the medial temporal region to be treated was assumed to have significant residual function. Another indication was when additional contralateral medial temporal focus was suspected. After opening the inferior horn via transsylvian or trans T1 approach, hippocampus was transected perpendicularly to the long axis, 4 mm interval, preserving fimbria/fornix and lateral connection until the epileptiform discharges were completely abolished, 11 patients had MRI lesions and 16 underwent surgery on the left side. WAIS R, WMS R, and Miyake Paired Word Recall were tested preoperatively, 1 month and 6.12 months postoperatively. MRI images including fast STIR for volumetry and diffusion tensor images. FDG-PET, ECD-SPECT and IMZ-SPECT images were acquired preoperatively and 6-12 months postoperatively. Seizure outcome was evaluated using Engel's classification at the latest follow-up. **Results:** The percentages of Engel I/II/III were 71/25/4 at 1 year postoperatively and 50/42/8 at 4 years postoperatively. Seizure outcome was better in lesional cases than non-lesional cases. Most of patients who had normal or near-normal preoperative memory scores and underwent dominantsided MHT experienced transient deterioration of the scores but recovery to the preoperative level at 6-12 months

**Conclusion:** Although a significant percentage of patients experienced seizure recurrence in the long-term follow-up, seizure outcome was acceptable considering the refractoriness of epilepsy for which MHT is indicated. While memory scores were well preserved, it was not accompanied by recovery of glucose uptake, volume reduction and white matter integrity in the treated medial temporal area.

## p149

### Eleven cases of syncope which had been diagnosed as epilepsy

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Epilepsy and syncope are two main causes of transient loss of consciousness.

It is important to distinguish epilepsy from syncope. However, we recognized that there were many cases of syncope that had been diagnosed as epilepsy. We need to evaluate these cases and discuss causes of misdiagnosis. This paper reports eleven syncope cases which had been misdiagnosed as epilepsy in other clinics and hospitals. Nine cases have triggers and eight cases have prodromal symptoms that are characteristic of syncope. Six patients had involuntary movements while they lost their consciousness. Two cases were diagnosed to have long QT syndrome and needed pacemaking. We suspect that convulsive syncope may not be recognized by some physicians. The EEGs did not have the epileptiform discharges in all cases. We must observe triggers, prodromal symptoms and concomitant symptoms carefully and identify the existence of convulsive syncope to prevent misdiagnosis.

### p150

## Genetic counseling in NICU babies with seizures and neurometabolic disorders $\underline{\text{USHA}}$

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**Purpose:** Seizures are the most common presenting signs in majority of neurometabolic disorders and often get confused with infectious encephalopathy. Acute onset of neonatal seizures, at times leading to progressive seizure disorder in neonates and infants are frequent manifestations. A precise and reliable diagnosis becomes important to prevent childhood morbidity and mortality. Hence, screening of NICU babies was undertaken for simultaneous diagnosis of common IEMs of amino acids, organic acids, carbohydrates and nucleic acids.

Method: The non invasive method of MILS, Japan to detect marker compounds of IEM by Gas

Chromatography / Mass Spectrometry (GC/MS) from the urine was used. It simultaneously screens 100+ metabolic abnormalities and further confirms the IEM diagnosis in one testing. **Results:** Total 135 NICU babes till 4 months age with neonatal convulsions were screened for IEM; of these 43 (32%) showed metabolic abnormalities and 23% cases had seizure onset within 5 days. Male to female ratio was 1.6:1. Other signs were lethargy (10 %), hypoglycemia (3%) metabolic acidosis (5%) and hyperammonemia (5%), with h/o neonatal death (12%) and consanguinity (6%). The common IEMs were MSUD (3), MMA (6), PPA (2), Isovaleric academia (1), Hyperglycinemia (2), Tyrosinemia (2), Glutaric acidemia-I (1), Urea cycle disorders (2), Galactosemia (3). Thirteen cases of Lactic acid metabolism indicated organic acids or mitochondrial involvement. One neurodegenerative disorder (Canavan) was diagnosed within a week, and one MMA case was managed with Vit. B12 therapy.

**Conclusion:** In the absence of newborn screening in India, the precise and early diagnosis of IEM in critical neonates with episodes of seizures was helpful in avoiding the delay in diagnosis and management. The severity of metabolic brain damage and nature of seizures could be explained during genetic counseling which was thus important in prevention and risk estimation in the affected family.

#### p151

#### **Clinical and EEG characteristics of Jeavons syndrome**

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**Objective:** To study the clinical and EEG characteristics of Jeavons syndrome.

**Methods:** Video-EEG monitoring was carried out in 9 patients with Jeavons syndrome. The clinical, EEG characteristics, treatment and prognoses were analyzed.

**Results:** Of the 9 patients, 8 were female, and only 1 was male. The age of eyelid myoclonia (EM) onset was from 3y to 9y. The onset age was obtained through the ways of complained, prosecution or VEEG monitoring. Three cases were misdiagnosed and 2 cases were overlooked initially. Seven out of 9 patients had generalized tonic clonic seizures (GTCS) during the course of disease, and 5 patients encountered only once. GTCS was the cause for the first visits to hospital in 5 patients. Because the clinical manifestations of EM with or without absence often showed slightly, VEEG monitoring with eye closure and intermittent photic stimulation tests helped to induce discharges and seizures. Eye closure was more potent than intermittent photic stimulation as a triggering factor. Ictal EEG showed generalized spike and waves and polyspikes burst. The main treatment option was valproate monotherapy (6 cases) or combined other antiepileptic drugs (1 case). Levetiracetam, lamotrigine and topiramate were also used in our patients and were effective in some degree. Two patients lost follow up. The age of 7 patients at follow up ranged from 9y to 15y. Seizures were controlled in 1 case, suspiciously controlled in 1 case, decreased in 4 cases and were still frequent in 1 case. During follow up, normal intelligence found in the former 2 cases, difficult learning in 2 cases, and slightly intellectual impair in 2 cases.

**Conclusion:** Jeavons syndrome is one of the idiopathic generalized epilepsies characterized by EM with or without absence. The time of seizure onset might be difficult to be exactly established, as EM was often misinterpreted and overlooked initially. Clinic history combined VEEG monitoring with eye closure and intermittent photic stimulation tests could diagnose this disease. Valproate and other new antiepileptic drugs were effective to this disease. Jeavons syndrome is a lifelong disorder. Seizures sometimes could be well controlled. When seizures were resistant to treatment, cognitive and intellectual impair might exist.

Key words: Eyelid myoclonia; Epilepsy; Jeavons syndrome; Eye closure.

#### p152

Interleukin-1 beta (il-1ß), interleukin-6 (il-6) and interleukin-1 receptor antagonist (il-1 ra) as risk factors of memory impairment in patients with complex partial epilepsy PURWA SAMATRA DPG

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Memory impairment is one of the complex partial seizures debilitating complications yet, many of them are found without the memory impairment. This fact bears a notion that there is a

certain factor involved in producing memory impairmanet in patiens with complex partial seizure. Hippocampus as part of temporal lobe where the memory is formed. The formation is facilitated by glutamate, well known as facilitator of epilepsy as well. The increase of glutamate release induces the release of pro-inflammatory cytokines such as IL-1 $\beta$ , IL-6 and expression of IL-1Ra receptor. Cytokines over-expression will interfere memory formation through Longterm Potentiation (LTP) mechanism. Should the abundant expression of cytokines take place the following memory impairment will happen afterward.

**Purpose:** The purpose of the study is to find out the difference as  $IL \cdot I\beta$ ,  $IL \cdot 6$  and  $IL \cdot IRa$  level in complex partial epilepsy with memory impairment and without memory impairment, and whether the increase of  $IL \cdot I\beta$ ,  $IL \cdot 6$  level and decrease of  $IL \cdot Ra$  in complex partial epilepsy are the risk factor of memory impairment.

**Method:** Case control study was the research method used. The research was conducted in RSUP. Sanglah Neurology Outpatient Clinic, private office and Prodia Clinical Laboratory in Denpasar and Jakarta. Samples of complex partial seizure were divided into twoo groups, of one memory impairment and another one without memory impairment. The numbers of samples were counted using matched case control study. There were 30 samples of complex partial seizure grouped as case and 30 samples as control.

**Result:** The result were analysed with K-S normalized study, t-test and bivariate analysis( Mc Nemar). The result was presented in table and narration.

Bivariate analysis (McNemar) of memory impairment risk factors in complex partial epilepsy are: IL-1 $\beta$  with cut off point 0.63 pg/ml OR = 70, statistically significant p = 0.001, IL-6 with cut off point 2.87 pg/ml OR = 4,57 is statistically insignificant p = 0.007, IL- 1Ra with cut off point 471 pg/ml OR = 0,727 is statistically not significant p = 0.573.

**Conclusion:** The increase of IL-1 $\beta$  and IL-6 level in complex partial epilepsy are the risk factors of memory impairment. However the decrease of LI-1Ra level in complex partial epilepsy is not proven as the risk factor of memory impairment

Keyword: Memory impairment, complex partial epilepsy, IL·1β, IL·6, IL·1Ra.

### p153

## Prevalence and clinical spectrum of epilepsy in children with cerebral palsy at Fatmawati General Hospital Jakarta

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**Purpose:** From the literature, the prevalence of epilepsy in children with cerebral palsy around 15-90% and it is not easy to treat. Low birth weight, neonatal fit, epilepsy history in the family, mental retarded and first seizure before 12 months of age could be presdisposition factors to developed epilepsy in cerebral palsy. The treatment is not easy, and some of them become intractable epilepsy. The purpose of this study is to find out the prevalence and the spectrum of epilepsy in children with cerebral palsy at Fatmawati General Hospital.

**Method:** One hundered ninety nine cerebral palsy patients ( $0 \cdot 60$  months) who come to Fatmawati general hospital between 2008 · 2010 were studied restrospectively. A detailed history and examination anad electroencephalography (EEG) were done in all cases.

**Results:** Of the one hundred ninety one children with spastic cerebral palsy included to the study, 91 were male, 100 of 105 children. The prevalence of epilepsy among them is 50.8%. Thirty six point one had a history of birth asphyxia. Prematurity (21,6%), low birth weight (22,7%) and central nervous system infection (75,6%) become the predisposising factors. We found 40,2% of them had seizure onset onset before 1 year of age. Generalized seizures were the most common, followed by partial seizures, infantile spasms, and other myoclonic seizures. Head circumference, EEG and CT abnormalities were seen in 85,6%, 88,4% and 88,9% of the them. Seizures were controlled in 50,1% children with 20,6% of them were free from seizure for more than 1 year. Polytherapy was required in 24 children cases.

**Conclusion:** Many factors include birth asphixia, prematurity, low birth weight and central nervous system infection are found in cerebral palsy children with epilepsy, and the treatment is not easy, longterm treatment and some of them need polytherapy and almost half of the cases still have not seizure free.

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# EURAP



## An International Antiepileptic Drugs and Pregnancy Registry

## **Mission Statement**

**EURAP** is an international collaboration with the vision to establish a basis for a safer treatment of epilepsy during pregnancy.

The primary objective is to compare the safety of different antiepileptic drugs during pregnancy with respect to the risk of birth defects.



- The well-being of women with epilepsy and their offspring is of great concern.
- Our aim is to contribute to a better understanding of the risks associated with antiepileptic drugs during pregnancy.
- What we need is the collaboration of physicians from all corners of the world to join forces.

## Join the Project !!!

All physicians who care for women taking antiepileptic drugs during pregnancy are invited to contribute, but you need to register before you can start enrolling pregnancies.

Contact : Leonor Cabral-Lim ,M.D.

National Coordinator, EURAP PHILIPPINES Mobile No. 09175589966

## PREPph

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# Do you know a famous athlete?



# Stand Up For Epilepsy

The ILAE, through the Taskforce on Sports and Epilepsy, is launching an exciting and innovative new project.

Stand Up For Epilepsy aims to create a collection of photographs of famous sportspersons meeting people with epilepsy. The photographs will convey the message that people with epilepsy, like athletes themselves, can be inspired to achieve their goals and lead full and active lives.

International sports stars such as Usain Bolt and Stephanie Rice are already lending their support. We need *your help* to bring together more athletes with people with epilepsy, and to create more inspiring photographs.

The collection will be exhibited at the London 2012 European Congress on Epileptology, which will be held shortly after the London Olympics.

> If you're in contact with a well-known sportsperson, we'd like to hear from you.

# Email: laura@epilepsycongress.org

Find us on Facebook

Tel: +353 1 2056 720 follow us on

#### NOTES

#### NOTES


#### NOTES





# 1<sup>st</sup> African Epilepsy Congress

The International League Against Epilepsy and the International Bureau for Epilepsy are honoured to bring the 1st ILAE-IBE African Epilepsy Congress to Nairobi, Kenya, 21st-23rd June 2012

- · Over forty local, regional and international speakers
- · Congress bursaries open for applications
- Up to sixty scientific posters can be accepted
- Early registration rates available until 30th March 2012



For further information visit: www.epilepsycongress.org • www.epilepsynairobi2012.org





# **30<sup>th</sup> INTERNATIONAL EPILEPSY CONGRESS** MONTREAL 2013



# 23rd - 27th June 2013



