INTERNATIONAL LEAGUE AGAINST EPILEPSY

IBE

ILAE

Final Programme and Abstract Book

8th Asian & Oceanian Epilepsy Congress

MELBOURNE, AUSTRALIA 21st - 24th October 2010

www.epilepsymelbourne2010.org

LONDON 2012

September 30th - October 4th

10th European Congress on Epileptology

10th European Congress on Epileptology

LONDON ILAE-CEA

The year of the Olympics and the European Epileptology Congress

CONTENTS

WELCOME MESSAGE FROM THE CHAIRS OF THE SCIENTIFIC ORGANISING COMMITTEE AND THE SCIENTIFIC CONSULTATIVE COMMITTEE

Dear Friends and Colleagues,

On behalf of the Scientific Organising and Consultative Committees, it gives us great pleasure to welcome you to the 8th Asian & Oceanian Epilepsy Congress (AOEC) in Melbourne, Australia. This Congress has been organised by the regional organisations of the International League Against Epilepsy (ILAE) and the International Bureau for Epilepsy (IBE).

More than 90 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 will be: "Epilepsy Surgery – Who and When?", "Outcome in Newly-diagnosed Epilepsy", "Prevention of Symptomatic Epilepsy" and "Psychological Wellbeing in Epilepsy". In addition to the main and post main session topics are parallel sessions which focus on 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 (ASEPA) has organised a series of didactic lectures by world-renowned experts in their respective fields, pre-congress workshops on epilepsy surgery and basic science, and a workshop on epilepsy research in the region. Furthermore, an exciting programme on Epilepsy and Society that will be of great interest to both individuals living with epilepsy and to staff from community organisations supporting people living with epilepsy is taking place on Thursday.

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 morning. During the Welcome Ceremony, Hasan AZIZ *(Pakistan)*, P SATISH CHANDRA *(India)*, Tatsuya TANAKA *(Japan)*, Xun WU *(China)* and Kazuichi YAGI *(Japan)* will receive the inaugural Asian and Oceanian Outstanding Achievement Epilepsy Award.

Melbourne is recognised as Australia's sporting and cultural capital and is notable for its architecture, restaurants, tram network and beautiful parks and gardens. Melbourne has a diverse, multicultural society and is consistently ranked as one of the most livable cities in the world. Melbourne also has several academic precincts in which internationally-recognised neuroscience research is conducted, including epilepsy. There is no doubt that you will feel at home in Melbourne. We hope the Congress meets your highest expectations and that you enjoy your stay here in Melbourne.

Welcome!

With warm regards,

Simon HARVEY *(Australia)*
Co-chair
Scientific Organising Committee

Shih Hui LIM *(Singapore)*
Co-chair
Scientific Organising Committee

Shunglon LAI *(Taiwan)*
Co-chair
Scientific Organising Committee

Terry O'BRIEN *(Australia)*
Chair
Scientific Consultative 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 of Epilepsy (IBE), we take great pleasure to welcome you all to Melbourne for the 8th Asian & Oceanian Epilepsy Congress.

The Congress offers a unique panorama of trends and innovation in the field of epilepsy research and an unmatched opportunity to meet and network with colleagues from various cultures in the region. In recent years, the Asian and Oceanian Epilepsy Congress has grown in stature and this 8th congress presents us with an opportunity to further this positive development and also to share and communicate different experiences of epilepsy from around the world.

The backdrop to this year's congress is the dynamic city of Melbourne. Often referred to as the cultural capital of Australia due to its diverse mélange of history, arts and entertainment, Melbourne represents an exciting venue with a lively atmosphere. The city's contemporary style can be witnessed through the modern architecture in the bustling Federation Square, while its artistic side can be explored through the range of cutting-edge museums such as the National Gallery of Victoria. The multicultural make-up of Melbourne's society provides a fitting environment for the variety of cultures represented at this congress. We hope that the wonderful cultural diversity of Melbourne will keep you entertained, stimulated and enthused.

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

With best wishes,

Solomon L. MOSHÉ *(USA)*
President ILAE

Mike GLYNN *(Ireland)*
President IBE

WELCOME MESSAGE FROM THE MINISTER FOR HEALTH GOVERNMENT OF VICTORIA

Welcome to Victoria and the 8th Asian & Oceanian Epilepsy Congress.

The Victorian Government is committed to boosting Victoria's medical research capabilities and has provided significant investment to enhance the capability of researchers, institutions and organisations in attracting international partnerships and investment in this sector. We continue to support clinical research, as advances in medical science directly benefit patients around the world.

The world-renowned Florey Neuroscience Institutes (FNI) in Victoria is one of the top 10 neuroscience research facilities, and incorporates the Brain Research Institute and the National Stroke Research Institute. In 2011, the FNI will also include two new purpose-built, state-of-the-art facilities - one at Parkville and the other at Austin Health in Heidelberg, which have been supported by the Victorian Government and other financial partners. The Mental Health Research Institute and University of Melbourne neuroscientists will co-locate with the FNI at these new facilities.

Victoria is also home to six biotechnology precincts including Parkville, the Alfred Medical Research & Education, Monash Health Research, Werribee, Bundoora and Austin Biomedical Alliance. In addition, the Bio21 Cluster links 21 research organisations to support shared infrastructure and expertise in biotechnology. The Victorian Government also supports the development and commercialisation of biotechnology research, and has made significant investments in this sector since 2001.

Whilst in Melbourne for the 8th Asian & Oceanian Epilepsy Congress, I encourage you to explore the many attractions of this city, including diverse arts and cultural opportunities, exciting entertainment options and a vibrant food and wine culture. If you have time before or after the Congress, I strongly encourage you to explore the variety and stimulation offered across regional Victoria, including breathtaking ocean views and tranquil river scapes, glorious mountain scenery, family-owned wineries and restaurants, regional art galleries and other cultural opportunities.

I congratulate the Congress Scientific Organising Committee and conference organisers on a comprehensive and contemporary programme, including many eminent presenters and facilitators. I extend my best wishes to all participants for a stimulating and productive Congress, which will facilitate the exchange of ideas and provide opportunities to forge new partnerships and friendships.

Daniel Andrews.

Daniel ANDREWS

GENERAL CONGRESS INFORMATION

Scientific Organising Committee

Simon HARVEY *(Australia)* Co-chair
Shih Hui LIM *(Singapore)* Co-chair
Shunglon LAI *(Taiwan)* Co-chair

Byung-In LEE *(Korea)*
Vinod SAXENA *(India)*
Graeme SHEARS *(Australia)*
Chong-Tin TAN *(Malaysia)*
Grace TAN *(Singapore)*
Tatsuya TANAKA *(Japan)*

Scientific Consultative Committee

Terence O'BRIEN *(Australia)* Chair

Hasan AZIZ *(Pakistan)*
Sam BERKOVIC *(Australia)*
Jill BICKNELL-ROYLE *(Australia)*
Andrew BLEASEL *(Australia)*
Leonor CABRAL LIM *(Philippines)*
Denise CHAPMAN *(Australia)*
Robert COLE *(Australia)*
Wendyl D'SOUZA *(Australia)*
John DUNNE *(Australia)*
Christopher FRENCH *(Australia)*
M. GOURIE-DEVI *(India)*
Yushi INOUE *(Japan)*
Satish JAIN *(India)*
Sunao KANEKO *(Japan)*
Patrick KWAN *(Hong Kong)*
Shichou LI *(China)*
Weiping LIAO *(China)*
Michael SAILING *(Australia)*
Ingrid SCHEFFER *(Australia)*
Ernest SOMERVILLE *(Australia)*
Jing-Jane TSAI *(Taiwan)*
Anannit VISUDTIBHAN *(Thailand)*

GENERAL CONGRESS INFORMATION

FACILITIES TIMETABLE

	Wednesday	Thursday	Friday	Saturday	Sunday
Registration	11:00-14:00	07:30-18:30	07:00-18:00	07:00-18:00	07:00-13:00
Speakers Ready Room	11:00-14:00	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

Business Centre
The Business Centre is situated at the front of the MCEC Exhibition Centre in which photocopying, printing, faxing and stationery purchases are all available. The Business Centre is open daily from 08:00-16:00.

Certificate of Attendance
A Certificate of Attendance will be available for all delegates for collection from the registration area on Sunday.

Coffee Breaks
Coffee, tea and snacks will be served in the exhibition area on the ground floor level of the MCEC Convention Centre from 10:30-11:00 on Friday, Saturday and Sunday and also from 16:00-16:30 on Friday and Saturday.

Cloakroom
The cloakroom is located on the ground floor of the MCEC Convention Centre beyond the concierge desk.

Exhibition
A trade exhibition will be held in conjunction with the 8th 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 on ground floor of the MCEC Convention Centre.

Internet Area
Internet stations are located within the exhibition area on the ground floor level of the MCEC Convention Centre; please note that these internet stations are open during exhibition hours only. There is payable wireless internet available throughout the MCEC Convention Centre.

GENERAL CONGRESS INFORMATION

Language
English is the official language of the 8th Asian & Oceanian Epilepsy Congress.

Liability and Insurance
The Congress Organiser will not accept 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 Melbourne. It is therefore recommended that participants arrange their own personal health, accident and travel insurance.

Lunch
Lunch will be served in the exhibition area on the ground floor level of the MCEC Convention Centre on Friday and Saturday from 12:30-13:30. Friday's lunch is kindly sponsored by Eisai Co. Ltd. and Dainippon Sumitomo Pharma Co. Ltd. Saturday's lunch is kindly sponsored by UCB.

Melbourne Convention and Visitors Bureau Survey
During the 8th Asian & Oceanian Epilepsy Congress, Melbourne Convention + Visitors Bureau (MCVB) will be conducting a survey to find out more about your experience in Melbourne during your congress stay. The study seeks to understand delegate behaviour and travel patterns when visiting Melbourne. We encourage you to complete this survey, which should only require five minutes of your time, after which you will receive a small Australian souvenir. Look for MCVB staff in blue t-shirts in the exhibition area to obtain your survey and souvenir.

Message Board
A message board can be found near to the registration area on the ground floor level of the MCEC Convention Centre. All delegates are invited to check it regularly.

Posters
Posters are exhibited on level 1 of the MCEC Convention Centre outside meeting rooms 101-112. 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. Presenting authors must be in attendance at their poster on Friday and Saturday from 12:30-13:30.

Prayer Room
A Prayer Room is located on the ground floor of the MCEC Convention Centre beyond the concierge desk.

Recording of Sessions
The congress sessions will be available for purchase on CD. Please visit the Medical Update stand at booth 29 in the exhibition area for further information. Please note that it is strictly forbidden to record any of the sessions using personal recording devises such as camcorders, cameras or mobile phones. Anyone found recording any part of a session will be removed from the session and may have their recording equipment withdrawn for the duration of the congress.

Registration
The registration area is located in the foyer of the ground floor level of the MCEC Convention Centre; 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$25 will be charged for a replacement badge.

GENERAL CONGRESS INFORMATION

Secretariat Office

Members of the Congress Secretariat can be contacted at the registration area on the ground floor level of the MCEC Convention Centre. For queries arising after the congress, please contact:

8th Asian & Oceanian Epilepsy Congress,
ILAE/IBE Congress Secretariat,
7 Priory Hall,
Stillorgan,
Dublin 18,
Ireland.
Tel : +353 1 2056 720
Fax : +353 1 2056 156
Email : **melbourne@epilepsycongress.org**
Website : **www.epilepsymelbourne2010.org**

Smoking Policy

The MCEC Convention Centre is a non-smoking area.

Speakers Ready Room

The Speakers Ready Room is located in Speakers Room 101 on level 1 of the MCEC Convention Centre. 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 at early morning sessions are required to submit their material before 17:00 on the day prior to their scheduled session.

Sponsors

The International League Against Epilepsy (ILAE), International Bureau for Epilepsy (IBE) and the 8th Asian & Oceanian Epilepsy Congress Scientific Organising Committee (AOEC SOC) would like to thank the following government and industry partners who contributed to this year's 8th AOEC:

Government Sponsors

Victoria
The Place To Be

MELBOURNE
AUSTRALIA

GENERAL CONGRESS INFORMATION

Platinum Sponsor

Gold Sponsor

Venue Information
The 8th Asian & Oceanian Epilepsy Congress will be held at the MCEC Convention Centre.
Address:
1 Convention Centre Place
South Wharf, Melbourne, Victoria 3006

Wheelchair Access
All conference rooms in the MCEC Convention Centre are wheelchair accessible.

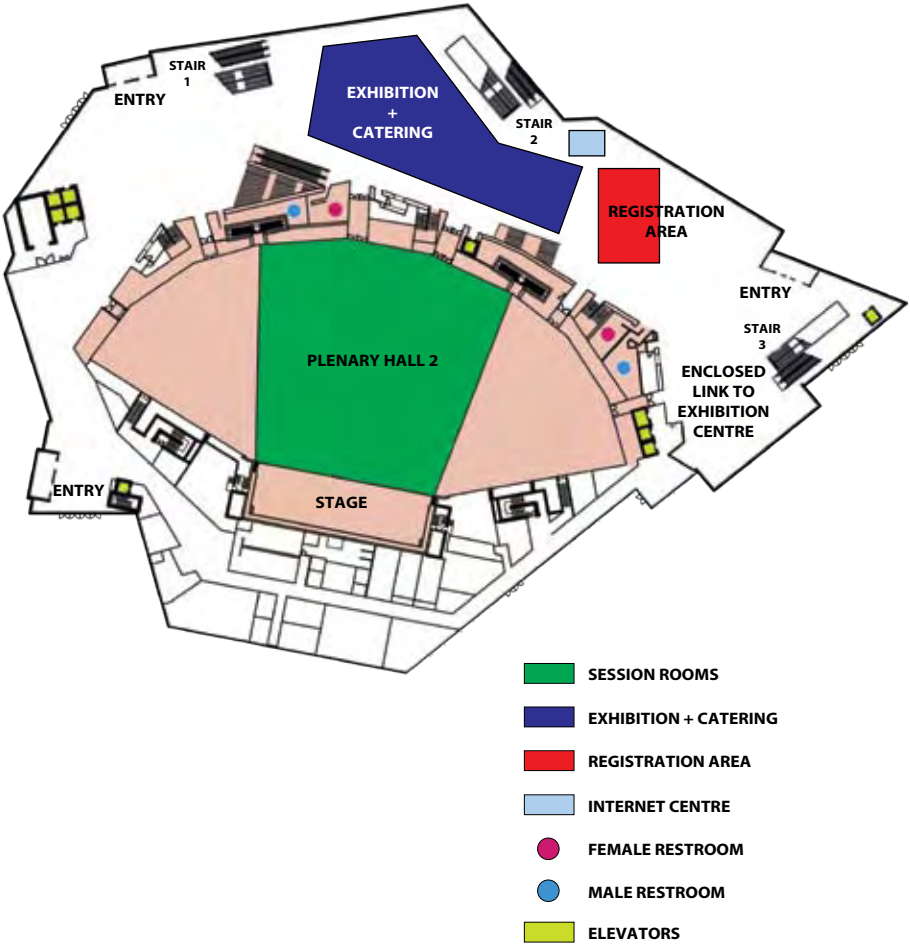
Social Events

Welcome Ceremony
The Welcome Ceremony of the 8th Asian & Oceanian Epilepsy Congress will take place in Plenary Hall 2 on the ground floor level of the MCEC Convention Centre on Thursday at 18:30. All delegates are invited to join the Welcome Reception which will be held following the Welcome Ceremony to the left of the exhibition area on the ground floor level of the MCEC Convention Centre.

Tours
There is an abundance of things to do in Melbourne and in the surrounding State of Victoria, not to mention further afield in Australia. Take advantage of being in this wonderful country and if you can, consider taking a few extra days after the congress to do an awe-inspiring post-congress tour. Alternatively, if time is short, there is a vast selection of half and full day tours to give you a taste for Australian culture and the city of Melbourne. Please visit the registration area for details on tours.

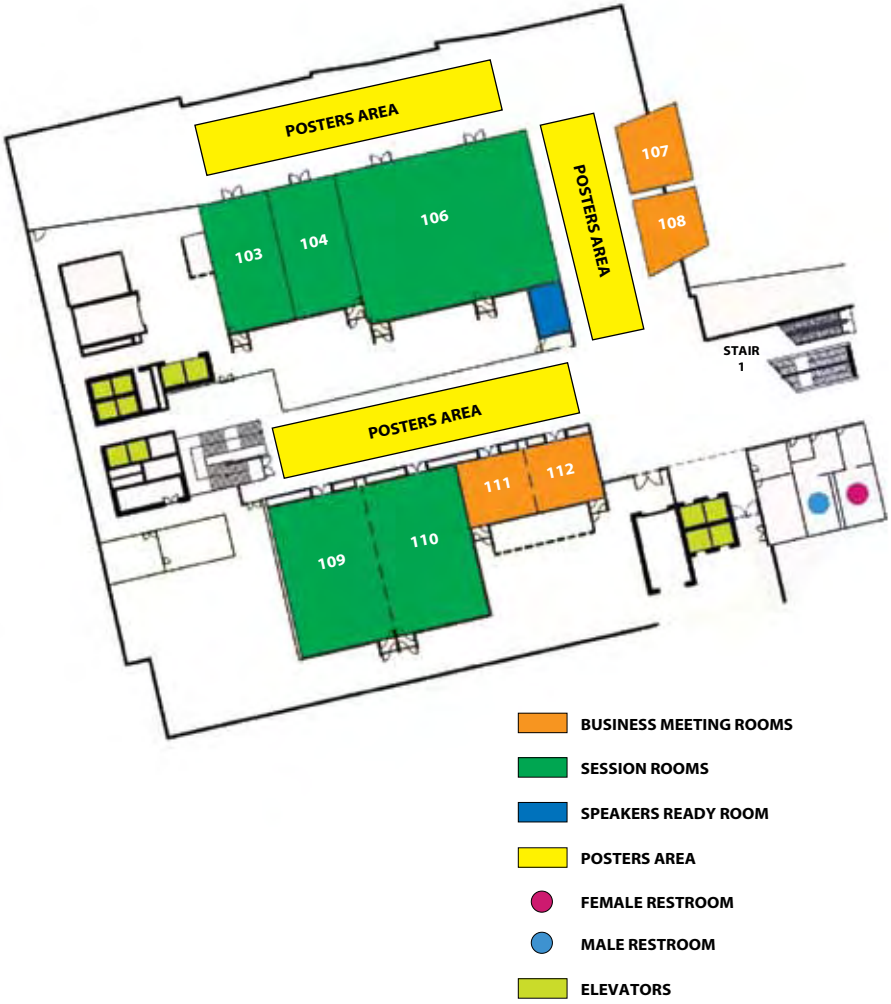
CONGRESS FLOOR PLANS

Ground Floor Level



CONGRESS FLOOR PLANS

Level 1



PRACTICAL INFORMATION ON MELBOURNE

Airport Departure Tax
Departure Tax should already be included in the price of the air fare.

Business Hours
Banks generally open from 09:30-16:00 on Monday to Friday. General office hours are 09:00-17:00 on Monday to Friday. Post Offices operate these hours too; however stamps are often available from hotels.

Climate
Melbourne is the southern most capital city on the mainland of Australia and offers a temperate climate; it has one of the country's lowest rainfalls too. However Melbourne does have a reputation for variable weather; it is not uncommon to have four seasons in one day! Unfortunately October can be one of the wettest months of the year; temperatures may vary between 12-22°C daily.

Communications
The international dialing code for Australia is 61 and the area code for Melbourne is 3. International Roaming facilities can connect mobile phone users. Phone cards are widely available throughout Melbourne.

Credit Cards and ATM's
Most hotels and large retail outlets accept major credit cards, the most widely used being Mastercard, Visa and AMEX. ATMs are widely available.

Currency
The Australian Dollar is Australia's unit of currency. Coins have values of 5, 10, 20 and 50 cents, and $1 AUD and $2 AUD; notes have values of $5 AUD, $10 AUD, $20 AUD, $50 AUD and $100 AUD.

Eating Out
Bistros, restaurants, cafes and coffee shops offer varied menus, prices and decor. Local specialties such as seafood and Australian wines are worth trying. The following website **www.eatability.com.au/au/melbourne/** provides helpful information on eating out.

Electicity
The electrical current is 240V 50HzHz. A two pin flat plug is used in Australia. Adaptors should be available at hotels.

Tax and Service Charges
Australia has a Goods and Services Tax (GST) of 10% on most goods and services such as accommodation, day tours, guides, translators, food, transport (including coach, rail and cruise) and other tourism services within Australia.

Time Zone
Melbourne is +10 hours ahead of Greenwich Mean Time/UTC in October.

Tipping
Tipping is at your discretion.

PRACTICAL INFORMATION ON MELBOURNE

Transport

An excellent system of trams, trains and buses serves Melbourne. Fares, timetables and network maps are available on the Metlink's website (**www.metlinkmelbourne.com.au**). For a list of trams and trains convenient for the Melbourne Convention & Exhibition Centre (MCEC) please view **www.mcec.com.au/Attend/Visitor-Info/Directions-and-Transport.html**. All Melbourne taxis are yellow. Taxi ranks are located outside the MCEC Convention Centre at the entrance beside the Hilton Hotel, as well as all major hotels and Southern Cross and Flinders Street Stations. Taxi meters are usually clearly visible, so passengers can keep check of their fares. Late night taxi trips between 22:00-05:00 often have to be paid for in advance.

What to Wear

October tends to be one of the wettest months and temperatures may vary between 12-22°C daily. The best advice is to consider 'layering'; choose clothing to suit the range of average temperatures stated, but allow for the odd day beyond these averages. Congress attire is smart casual.

GENERAL SCIENTIFIC INFORMATION

AOOAEA - The Asian and Oceanian Outstanding Achievement Epilepsy Award

The Asian and Oceanian Outstanding Achievement Epilepsy Award (AOOAEA) was introduced this year in order to recognize and pay tribute to medical or non-medical professionals for their extraordinary contributions to epilepsy care in this region. The award is bestowed on Hasan AZIZ *(Pakistan)*, P SATISH CHANDRA *(India)*, Tatsuya TANAKA *(Japan)*, Xun WU *(China)* and Kazuichi YAGI *(Japan)* and will be given out during the Welcome Ceremony on Thursday.

ASEPA EEG Certification Examination Part 1

The ASEPA EEG Certification Examination Part 1 will take place on Friday from 08:30-11:30.

ASEPA Workshops

Clinical Epilepsy Workshops: There are two clinical epilepsy workshops as pre-congress activities. 'Epilepsy surgery in neocortical epilepsy' takes place on Wednesday afternoon from 13:30-18:00 and then 'Epilepsy, memory and epilepsy surgery' takes place on Thursday morning from 08:30-12:00.
Basic Science Workshop: There is one basic science workshop as a pre-congress activity; 'The use of animal models for epilepsy research' takes place on Wednesday afternoon from 13:00-18:00.
Registration: To attend the ASEPA pre-congress workshops, you must register for them at the onsite registration desk. The registration fee for one half day workshop is US$30 and the registration fee for two half day workshops is US$50.
Full details on all three workshops may be found on pages 19, 20 and 24.

Epilepsy and Society Programme (IBE)

An exciting programme that will be of great interest to both individuals living with epilepsy and for staff from community organisations supporting people living with epilepsy will take place on Thursday from 08:45-18:15 in Room 106 on level 1 of the MCEC Convention Centre. This programme has been developed by local and regional committees of the International Bureau for Epilepsy (IBE). A well regarded group of presenters includes widely published sociologist Graham Scambler from University of Central London and the popular Australian television sports presenter and author Wally Lewis. The main topics focus on "Epilepsy and Stigma", "Employment and Advocacy" and "SUDEP". Full details may be found on pages 25-27.
Please note that separate registration is required for this programme, please enquire at the onsite registration desk.

ILAE / CAOA Chapter Convention

The ILAE / CAOA Chapter Convention will take place on Thursday from 11:15-13:45 in Room 109 on level 1 of the MCEC Convention Centre; lunch will be served during the meeting. All those attending the Chapter Convention are asked to kindly pick up their registration badges for the 8th Asian & Oceanian Epilepsy Congress in advance of the meeting.

GENERAL SCIENTIFIC INFORMATION

Tadokoro Award

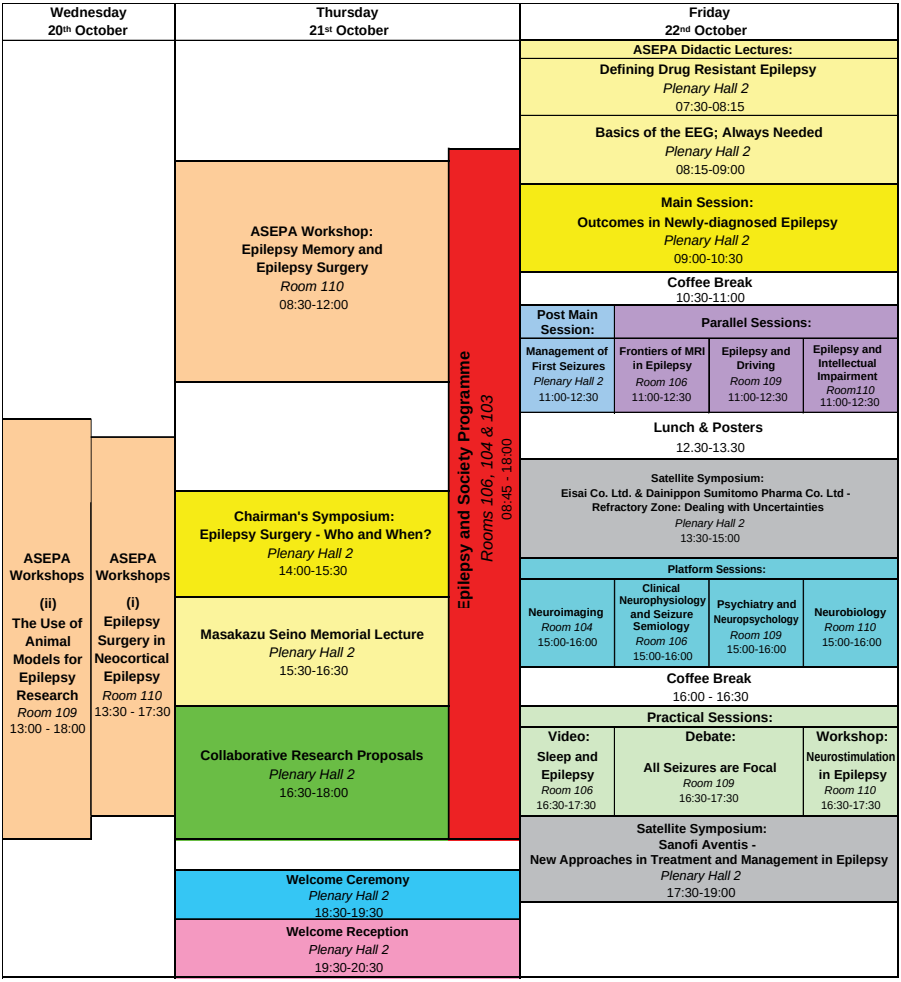
In order to encourage research in epileptology in the region, there will be best presentation prizes for both platform and poster presentations. Dr. Tadokoro is a retired epileptologist from Japan who contributed generously to the activities of the ILAE Commission on Asian and Oceanian Affairs (CAOA).
The first and second prizes for both the Platform and Poster session are US$300 and US$200 respectively and the recipients will be announced on Sunday before the main session.

Travel Bursary Awards

The Travel Bursary Award scheme was established to assist delegates in attending the 8th Asian & Oceanian Epilepsy Congress. A particular emphasis was given to those coming from developing regions, who are locally active in the field of epilepsy. A total of 32 Travel Bursary Awards were provided for the 8th Asian & Oceanian Epilepsy Congress; funding for these awards was provided by the International League Against Epilepsy (ILAE), the International Bureau for Epilepsy (IBE) and the ILAE Commission on Asian and Oceanian Affairs (CAOA).

SCIENTIFIC PROGRAMME

Full Programme Timetable



SCIENTIFIC PROGRAMME

Full Programme Timetable

Saturday 23rd October

ASEPA Didactic Lectures:

Genetics
Plenary Hall 2
07:30-08:15

Psychiatric Issues
Plenary Hall 2
08:15-09:00

Main Session:
Prevention of Symptomatic Epilepsy
Plenary Hall 2
09:00-10:30

Coffee Break
10.30-11.00

Post Main Session:	Parallel Sessions:			
Epilepsy Prevention *Plenary Hall 2* 11:00-12:30	**Global Campaign Against Epilepsy** *Room 104* 11:00-12:30	**SUDEP** *Room 106* 11:00-12:30	**Genetics of Large Populations** *Room 109* 11:00-12:30	**Tuberous Sclerosis and Epilepsy** *Room 110* 11:00-12:30

Lunch & Posters
12.30-13.30

Satellite Symposium:
UCB -
Management of Inadequately Controlled Epilepsy: Evaluating the Evidence
Plenary Hall 2
13:30-15:00

Platform Sessions:

Paediatric Epileptology *Room 103* 15:00-16:00	**Surgery and Neuroimaging** *Room 104* 15:00-16:00	**Social and Economic Issues** *Room 106* 15:00-16:00	**General Epileptology** *Room109* 15:00-16:00	**Basic Science** *Room 110* 15:00-16:00

Coffee Break
16:00 - 16:30

Practical Sessions:

Video: Is This an Epileptic Seizure? *Room106* 16:30-17:30	**Debate: Surgery is the Best Way Forward in Refractory Focal Epilepsy** *Room109* 16:30-17:30	**Workshop: Management Guidelines in Epilepsy** *Room 110* 16:30-17:30

Platform Sessions:

Translational Research *Room 104* 17:30-18:30	**Genetics** *Room 106* 17:30-18:30	**AED Issues** *Room 109* 17:30-18:30	**Surgery** *Room 110* 17:30-18:30

Sunday 24th October

ASEPA Didactic Lecture:

Surgery
Room 106
08:00-08:45

Awards Ceremony
Room 106

Main Session:
Psychological Wellbeing in Epilepsy
Room 106
09:00-10:30

Coffee Break
10.30-11.00

Post Main Session:	Parallel Sessions:	
Social and Economic Aspects of Epilepsy *Room 106* 11:00-12:30	**Absence Epilepsies** *Room 109* 11:00-12:30	**Adverse Effects of Antiepileptic Drugs** *Room 110* 11:00-12:30

SCIENTIFIC PROGRAMME

Wednesday 20th October

Room 109	Room 110
ASEPA Workshop: The Use of Animal Models for Epilepsy Research *13:00-18:00*	**ASEPA Workshop:** Epilepsy Surgery In Neocortical Epilepsy *13:30-17:30*

SCIENTIFIC PROGRAMME

Wednesday 20th October

13:00 · 18:00 ASEPA Workshop: Basic Science Room 109

The use of animal models for epilepsy research
Chairs: FRENCH, C (Australia) / O'BRIEN, T (Australia)

Animal models of genetic and acquired epilepsies: how good are they?
BERTRAM, E H (USA)

Acute animal models: surprising contributions to epilepsies
WILLOUGHBY, J (Australia)

Novel disease mechanisms revealed by mouse models of familial epilepsy syndromes
PETROU, S (Australia)

Use of gene expression systems to study cellular effects of human epilepsy mutations
KAZUAKI, K (Japan)

Transgenic rat models for epilepsy
HIROSE, S (Japan)

An animal model for Dravet syndrome: molecular and cellular basis of the disease
YAMAKAWA, K (Japan)

The use of animal models to investigate neuropsychiatric and neurocognitive co-morbidities
JONES, N (Australia)

Putting it all together: molecules to epilepsy
HUGUENARD, J (USA)

General discussion
O'BRIEN, T (Australia) / FRENCH, C (Australia)

SCIENTIFIC PROGRAMME

Wednesday 20th October

13:30 - 17:30 ASEPA Workshop: Clinical Epilepsy Room 110

Epilepsy surgery in neocortical epilepsy
Chairs: BLEASEL, A (Australia) / CHINVARUN, Y (Thailand)

Effect of lesion status on management and surgical outcome of neocortical epilepsy
RADHAKRISHNAN, K (India)

Identification and characterisation of lesions in intractable neocortical epilepsy
LOCHARERNKUL, C (Thailand)

Magnetoencephalographic evaluation of neocortical epilepsy and eloquent cortical function
NAKASATO, N (Japan)

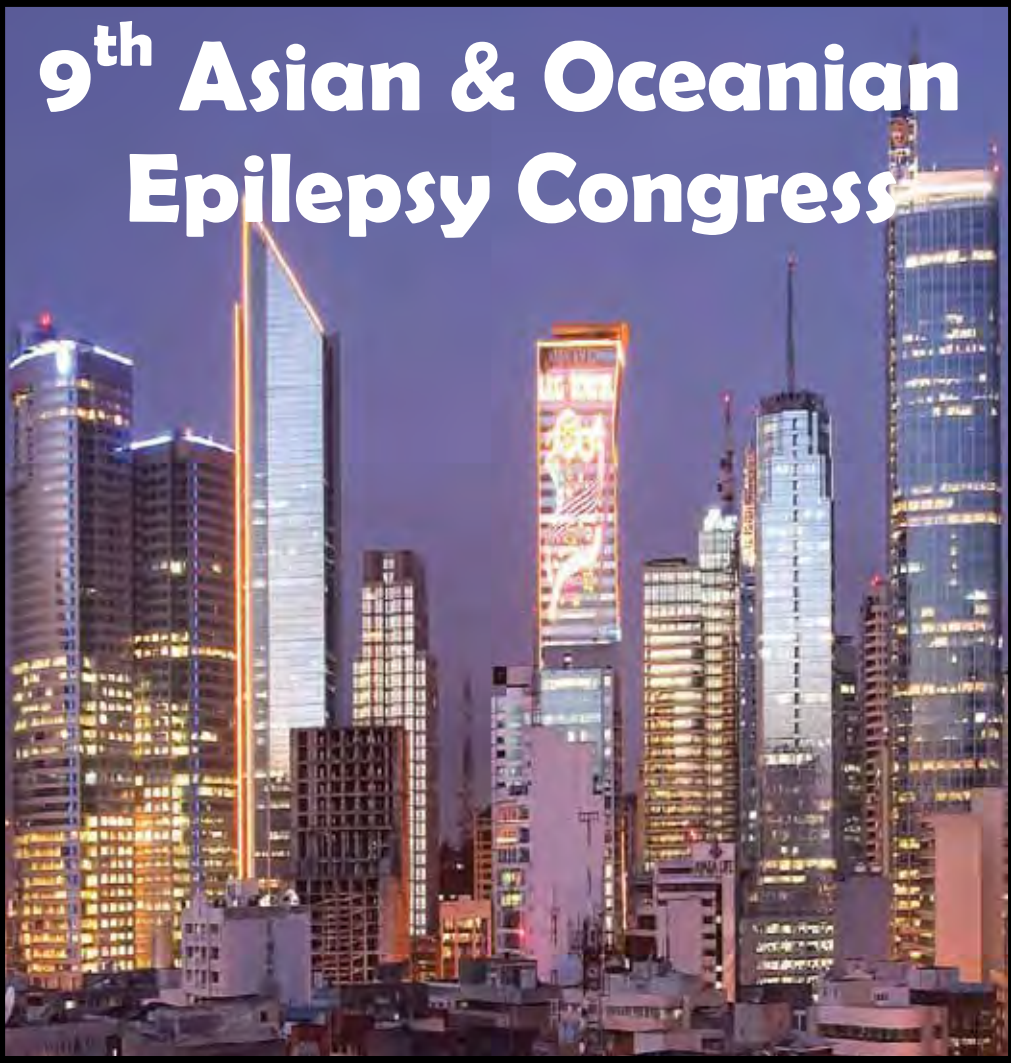
Case presentation and discussion: lesional neocortical epilepsy
CHINVARUN, Y (Thailand)

Defining the epileptogenic zone in non-lesional neocortical epilepsy, non-invasively
BLEASEL, A (Australia)

Surgical approaches in non-lesional neocortical epilepsy
LEE, S K (South Korea)

Case presentation and discussion: non-lesional neocortical epilepsy
LUAN, G (China)

9th Asian & Oceanian Epilepsy Congress



Manila, Philippines

March 2012

IBE

INTERNATIONAL LEAGUE AGAINST EPILEPSY ILAE

www.epilepsycongress.org

SCIENTIFIC PROGRAMME

Thursday 21st October

<table>
<tr><th>Plenary Hall 2</th><th>Room 103</th><th>Room 104</th><th>Room 106</th></tr>
<tr><td></td><td></td><td></td><td></td></tr>
<tr><td rowspan="6">Chairman's Symposium:
Epilepsy Surgery - Who and When?
14:00-15:30</td><td rowspan="3">Epilepsy & Society Programme :
Break-Out Room
11:00-16:30</td><td rowspan="3">Epilepsy & Society Programme :
Break-Out Room
11:00-16:30</td><td rowspan="5">Epilepsy & Society Programme
08:45-18:00</td></tr>
<tr></tr>
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<tr><td>Masakazu Seino Memorial Lecture
15:30-16:30</td></tr>
<tr><td>Collaborative Research Proposals
16:30-18:00</td><td rowspan="3"></td><td rowspan="3"></td></tr>
<tr><td></td></tr>
<tr><td>Welcome Ceremony
18:30-19:30</td><td></td></tr>
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SCIENTIFIC PROGRAMME

Thursday 21st October

Room 107	Room 109	Room 110
CAOA & ASEPA Business Meetings *08:00-11:00*		**ASEPA Workshop:** Epilepsy Memory and Epilepsy Surgery *08:30-12:00*
	ILAE/CAOA Chapter Convention *11:15-13:45*	

SCIENTIFIC PROGRAMME

Thursday 21st October

08:30 · 12:00 ASEPA Workshop: Clinical Epilepsy — Room 110

Epilepsy, memory and epilepsy surgery
Chairs: KRISHNAMOORTHY, E S (India) / SALING, M (Australia)

Neuropsychological assessment of memory in epilepsy
SALING, M (Australia)

Memory problems in patients with medial TLE
INOUE, Y (Japan)

Effect of psychiatric, psychosocial and other cognitive factors on memory
KRISHNAMOORTHY, E S (India)

Intracarotid amytal testing in medial TLE
KANNER, A M (USA)

Case presentation and discussion
LAWN, N (Australia) / BOWDEN, S (Australia)

11:15 · 13:45 ILAE/ CAOA Chapter Convention — Room 109

14:00 · 15:30 Chairmans Symposium — Plenary Hall 2

Epilepsy surgery: who and when?
Chairs: HARVEY, S (Australia) / LIM, S (Singapore)

Seizure outcomes following epilepsy surgery
WIEBE, S (Canada)

Epilepsy surgery with limited resources
RADHAKRISHNAN, K (India)

Lesional epilepsy: how long should you wait?
LEE, B-I (South Korea)

Non-lesional, extra temporal epilepsy: when is surgery justified?
BLEASEL, A (Australia)

15:30 · 16:30 Special Lecture — Plenary Hall 2

Masakazu Seino memorial lecture
Chair: TAN, C-T (Malaysia)

Heredity as the cause of epilepsy
SHORVON, S (United Kingdom)

SCIENTIFIC PROGRAMME

Thursday 21st October

16:30 · 18:00 Special Session — Plenary Hall 2

Collaborative research proposals in the Asia-Oceanian region
Chairs: D'SOUZA, W (Australia) / KWAN, P (Hong Kong)

Genetics
BERKOVIC, S F (Australia)

Community based epidemiology
D'SOUZA, W (Australia)

Antiepileptic drug-induced Stevens Johnson syndrome
KWAN, P (Hong Kong)

Stigma
LIM, K S (Malaysia)

Paediatric epileptology
SCHEFFER, I (Australia)

The EpiNet project
BERGIN, P (New Zealand)

Epilepsy and Society Programme (IBE)

08:45 · 09:00 Welcome — Room 106
SHEARS, G (Australia) / GLYNN, M (Ireland)

09:00 · 10:30 Main Session — Room 106

Epilepsy and stigma
Chairs: LAI, S (Taiwan) / SAXENA, V (India)

Stigma and QOL
SCAMBLER, G (United Kingdom)

My journey
LEWIS, W (Australia)

SCIENTIFIC PROGRAMME

Thursday 21st October

Epilepsy and Society Programme (IBE)

11:00 · 12:30 Parallel Discussion Group Room 106

Epilepsy and memory
Chair: CUMMINS, J (Australia)

The nature of memory dysfunction in epilepsy and its neurological basis
WEINTROB, D (Australia)

Practical ways to live with memory difficulties
BELLON, M (Australia)

11:00 · 12:30 Parallel Discussion Group Room 104

Epilepsy and sexuality
Chair: WALKER, C (Australia)

Changes in sexual functioning after epilepsy surgery
WILSON, S (Australia)

Women's reproductive health and epilepsy
VOLLENHOVEN, B (Australia)

13:30 · 15:00 Parallel Session Room 106

Employment and advocacy
Chairs: COLE, R (Australia) / SRINIVAS, H V (India)

Epilepsy and employment
TAN, G (Singapore)

The need of advocacy programmes for public awareness in the developing countries
MEHNDIRATTA, M (India)

Workplace inclusion
PFEIFFER, W (Australia)

13:30 · 15:00 Parallel Session Room 104

SUDEP
Chairs: CHAPMAN, D (Australia) / TSENG, Y-F (Taiwan)

What is it and who seems to be at risk?
SO, E (USA)

Global, local and individual perspectives
PANELLI, R (Australia)

Panel discussion: the global conversation
SO, E (USA), PANELLI, R (Australia), PRESTON, J (United Kingdom)

SCIENTIFIC PROGRAMME

Thursday 21st October

Epilepsy and Society Programme (IBE)

15:30 · 16:30	Parallel Discussion Group	Room 106
	Practical ways to manage depression *Chair: WHITEHEAD, H (Australia)*	
	The association between epilepsy and depression *SALZBERG, M (Australia)*	
	An overview of treatment options for depression in people with epilepsy *JONES, S (United Kingdom)*	
15:30 · 16:30	Parallel Discussion Group	Room 104
	Epilepsy and art *Chair: SHEARS, G (Australia)*	
	The influences of epilepsy in visual art *CHAMBLISS, J (Australia)*	
	Caligraphy *JANARDHAN, K (India)*	
15:30 · 16:30	Parallel Discussion Group	Room 103
	Epilepsy, sport and exercise *Chair: COLE, R (Australia)*	
	Epilepsy, diet and AEDs *CLIGNET, M (France)*	
	Understanding the risks *SOMERVILLE, E (Australia)*	
16:30 · 17:15	Book Launch	Room 106
	Social Epileptology, edited by Jaya Pinikahana and Christine Walker	
	Epilepsy in the Family, a new title by Suzanne Yanko	
17:15 · 18:00	Outstanding Persons Awards	Room 106

SCIENTIFIC PROGRAMME

Friday 22nd October

Plenary Hall 2	Room 103	Room 104	Room 106
ASEPA Didactic Lecture: Defining Drug Resistant Epilepsy *07:30-08:15*			
ASEPA Didactic Lecture: Basics of the EEG; Always Needed *08:15-09:00*	**ASEPA EEG Certification Exams Part 1** *08:30-11:30*		
Main Session: Outcomes in Newly Diagnosed Epilepsy *09:00-10:30*			
Coffee Break - Exhibition Area		**Coffee Break - Exhibition Area**	
Post Main Session: Management of First Seizures *11:00-12:30*			**Parallel Session:** Frontiers of MRI in Epilepsy *11:00-12:30*
Lunch: Sponsored by Eisai Co. Ltd. & Dainippon Sumitomo Pharma Co. Ltd - Exhibition Area			
Satellite Symposium: Eisai Co. Ltd. & Dainippon Sumitomo Pharma Co. Ltd - Refractory Zone: Dealing with Uncertainties *13:30-15:00*			
		Platform Session: Neuroimaging *15:00-16:00*	**Platform Session:** Clinical Neurophysiology and Seizure Semiology *15:00-16:00*
Coffee Break - Exhibition Area			
			Video: Sleep and Epilepsy *16:30-17:30*
Satellite Symposium: Sanofi Aventis - New Approaches in Treatment and Management in Epilepsy *17:30-19:00*			

SCIENTIFIC PROGRAMME

Friday 22nd October

<table>
<tr><th>Room 109</th><th>Room 110</th><th>Poster Area</th></tr>
<tr><td></td><td></td><td></td></tr>
<tr><td></td><td></td><td>Poster Set-Up
07:30-09:00</td></tr>
<tr><td></td><td></td><td rowspan="2">Posters on Display
09:00-12:30</td></tr>
<tr><td colspan="2">Coffee Break - Exhibition Area</td></tr>
<tr><td>Parallel Session:
Epilepsy and Driving
11:00-12:30</td><td>Parallel Session:
Epilepsy and Intellectual Impairment
11:00-12:30</td><td></td></tr>
<tr><td colspan="2">Lunch: Sponsored by Eisai Co. Ltd. & Dainippon Sumitomo Pharma Co. Ltd - Exhibition Area</td><td>Poster Presentations
12:30-13:30</td></tr>
<tr><td></td><td></td><td rowspan="3">Posters on Display
13:30-17:00</td></tr>
<tr><td>Platform Session:
Psychiatry and Neuropsychology
15:00-16:00</td><td>Platform Session:
Neurobiology
15:00-16:00</td></tr>
<tr><td colspan="2">Coffee Break - Exhibition Area</td></tr>
<tr><td>Debate:
All Seizures are Focal
16:30-17:30</td><td>Workshop:
Neurostimulation in Epilepsy
16:30-17:30</td><td></td></tr>
<tr><td></td><td></td><td></td></tr>
</table>

SCIENTIFIC PROGRAMME

Friday 22nd October

07:30 · 08:15 ASEPA Didactic Lecture — Plenary Hall 2

Drug treatment
Chair: CABRAL LIM, L (Philippines)

Defining drug resistant epilepsy
KWAN, P (Hong Kong)

08:15 · 09:00 ASEPA Didactic Lecture — Plenary Hall 2

Basic science
Chair: GAMAGE, R (Sri Lanka)

Basics of the EEG: always needed
MOSHÉ, S L (USA)

09:00 · 10:30 Main Session — Plenary Hall 2

Outcome in newly-diagnosed epilepsy
Chairs: DUNNE, J (Australia) / LAI, S (Taiwan)

Predictors of outcome in newly-diagnosed epilepsy: clinical, EEG and imaging
SO, E (USA)

Pharmacogenomics: predicting seizure response and adverse events
KWAN, P (Hong Kong)

Psychosocial outcomes of newly-diagnosed epilepsy
WILSON, S (Australia)

Attitudes to epilepsy and their impact on outcome: insights from rural China
WANG, W (China)

SCIENTIFIC PROGRAMME

Friday 22nd October

11:00 · 12:30 Post Main Session — Plenary Hall 2

Management of first seizures
Chairs: LAI, S (Taiwan) / TAKAHASHI, T (Japan)

Do you need a second seizure for diagnosis of epilepsy?
LIM, S (Singapore)

Management decisions in children: investigation, treatment and counselling
VISUDTIBHAN, A (Thailand)

Management decisions in adults and the elderly: investigation, treatment and counselling
LAWN, N (Australia)

Discussion panel: *SO, E (USA), KWAN, P (Hong Kong), WILSON, S (Australia), WANG, W (China), LIM, S (Singapore), VISUDTIBHAN, A (Thailand), LAWN, N (Australia)*

11:00 · 12:30 Parallel Session — Room 106

Frontiers of MRI in epilepsy
Chairs: JACKSON, G (Australia) / LEE, B-I (South Korea)

EEG-fMRI and epilepsy networks
JACKSON, G (Australia)

Tractography: applications in diagnosis and treatment of epilepsy
LEE, S-K (South Korea)

White matter fibre tracking: dealing with crossing fibres and super resolution imaging
CONNELLY, A (Australia)

11:00 · 12:30 Parallel Session — Room 109

Epilepsy and driving
Chairs: GLYNN, M (Ireland) / SOMERVILLE, E (Australia)

Driving and epilepsy: a risk assessment
DUNNE, J (Australia)

Regional differences in certification of fitness to drive
TSAI, J-J (Taiwan)

Legal and ethical issues for patients and doctors
SOMERVILLE, E (Australia)

SCIENTIFIC PROGRAMME

Friday 22nd October

11:00 · 12:30	Parallel Session	Room 110
	Epilepsy and intellectual impairment *Chairs: ORTIZ, M (Philippines) / SCHEFFER, I (Australia)*	
	Syndromes of epilepsy and intellectual disability *SCHEFFER, I (Australia)*	
	New genetic and microchromosomal diagnostic technologies *LEE, Y-M (South Korea)*	
	Principles and practice of behaviour management *WAKAMOTO, H (Japan)*	
13:30 · 15:00	Satellite Symposium · Eisai Co. Ltd. & Dainippon Sumitomo Pharma Co. Ltd.	Plenary Hall 2
	Refractory zone: dealing with uncertainties *Chairs: BLEASEL, A (Australia) / KANEKO, S (Japan)*	
	Challenge in management of partial seizures *MOHAMED, A (Australia)*	
	Zonisamide in refractory partial seizures *LEPPIK, I (USA)*	
	Learning from two decades of experience *OSAWA, M (Japan)*	
15:00 · 16:00	Platform Session	Room 104
Refer to page 56	**Neuroimaging** *Chairs: LOCHARERNKUL, C (Thailand) / COOK, M (Australia)*	
15:00 · 16:00	Platform Session	Room 106
Refer to page 56	**Clinical neurophysiology and seizure semiology** *Chairs: GUTIERREZ, J (Philippines) / TSAI, J-J (Taiwan)*	
15:00 · 16:00	Platform Session	Room 109
Refer to page 57	**Psychiatry and neuropsychology** *Chairs: KRISHNAMOORTHY, E S (India) / KANNER, A M (USA)*	

SCIENTIFIC PROGRAMME

Friday 22nd October

15:00 · 16:00 Platform Session — Room 110

Refer to page 57

Neurobiology
Chairs: REID, C (Australia) / HUGUENARD, J (USA)

16:30 · 17:30 Practical Session: Video — Room 106

Sleep and epilepsy
Chairs: GILL, D (Australia) / IKEDA, A (Japan)

Sleep and epilepsy: presentation 1
IKEDA, A (Japan)

Sleep and epilepsy: presentation 2
GILL, D (Australia)

16:30 · 17:30 Practical Session: Debate — Room 109

All seizures are focal
Chair: BERKOVIC, S F (Australia)

All seizures are focal: For
JACKSON, G (Australia)

All seizures are focal: Against
SHORVON, S (United Kingdom)

16:30 · 17:30 Practical Session: Workshop — Room 110

Neurostimulation in epilepsy
Chairs: LEE, B-I (South Korea) / WANG, Y (China)

Clinical trials and experience with open-loop neurostimulation
CHO, Y-J (South Korea)

Development and clinical trials of closed-loop neurostimulation
COOK, M (Australia)

Repetitive transcranial magnetic stimulation in drug-resistant epilepsy
WANG, Y (China)

SCIENTIFIC PROGRAMME

Friday 22nd October

17:30 · 19:00 Satellite Symposium · Sanofi Aventis Plenary Hall 2

New approaches in treatment and management in epilepsy
Chair: O'BRIEN, T (Australia)

The Australian pregnancy register of antiepileptic drugs:
Its contributions over the decade
VAJDA, F (Australia)

A review of intravenous antiepileptic drugs
COOK, M (Australia)

Melbourne welcomes delegates from the 8th Asian & Oceanian Epilepsy Congress

Melbourne is celebrated as Australia's home to the arts, culture, sport and shopping. Yet just a 1 to 2 hour drive takes you worlds away – take your pick from 100 local vineyards, rest and relax at an award-winning day spa, tee-off at a world-class golf course, snowboard in the mountains during winter, or get up close and personal with wildlife and penguins at the Phillip Island Nature Park.

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VICTORIA AUSTRALIA

SCIENTIFIC PROGRAMME

Saturday 23rd October

Plenary Hall 2	Room 103	Room 104	Room 106	Room 109
ASEPA Didactic Lecture: Genetics of the Epilepsies; an Update *07:30-08:15*				
ASEPA Didactic Lecture: Neurobiologic Aspects of Depression in Epilepsy: Do they Explain the High Comorbidity of the Two Disorders? *08:15-09:00*				
Main Session: Prevention of Symptomatic Epilepsy *09:00-10:30*				
Coffee Break - Exhibition Area				
Post Main Session: Epilepsy Prevention *11:00-12:30*		**Parallel Session:** Global Campaign Against Epilepsy *11:00-12:30*	**Parallel Session:** SUDEP *11:00-12:30*	**Parallel Session:** Genetics of Large Populations *11:00-12:30*
Lunch: Sponsored by UCB - Exhibition Area				
Satellite Symposium: UCB - Management of Inadequately Controlled Epilepsy; Evaluating the Evidence *13:30-15:00*				
	Platform Session: Paediatric Epileptology *15:00-16:00*	**Platform Session:** Surgery and Neuroimaging *15:00-16:00*	**Platform Session:** Social and Economic Issues *15:00-16:00*	**Platform Session:** General Epileptology *15:00-16:00*
Coffee Break - Exhibition Area				
			Video: Is This an Epileptic Seizure? *16:30-17:30*	**Debate:** Surgery is the Best Way Forward in Refractory Focal Epilepsy *16:30-17:30*
		Platform Session: Translational Research *17:30-18:30*	**Platform Session:** Genetics *17:30-18:30*	**Platform Session:** AED Issues *17:30-18:30*

SCIENTIFIC PROGRAMME

Saturday 23rd October

Room 110	Poster Area
	Posters On Display *09:00-12:30*
Coffee Break - Exhibition Area	
Parallel Session: Tuberous Scierosis and Epilepsy *11:00-12:30*	
Lunch: Sponsored by UCB - Exhibition Area	**Poster Presentations** *12:30-13:30*
	Posters On Display *13:30-17:00*
Platform Session: Basic Science *15:00-16:00*	
Coffee Break - Exhibition Area	
Workshop: Management Guidelines in Epilepsy *16:30-17:30*	**Poster Removal** *17:00-18:00*
Platform Session: Surgery *17:30-18:30*	

SCIENTIFIC PROGRAMME

Saturday 23rd October

07:30 · 08:15	ASEPA Didactic Lecture	Plenary Hall 2
	Genetics *Chair: MANNAN, M (Bangladesh)*	
	Genetics of the epilepsies: an update *BERKOVIC, S F (Australia)*	
08:15 · 09:00	ASEPA Didactic Lecture	Plenary Hall 2
	Psychiatric issues *Chair: AGRAWAL, J P (Nepal)*	
	Neurobiologic aspects of depression in epilepsy: do they explain the high comorbidity of the two disorders? *KANNER, A M (USA)*	
09:00 · 10:30	Main Session	Plenary Hall 2
	Prevention of symptomatic epilepsy *Chairs: SAXENA, V (India) / TAN, C-T (Malaysia)*	
	Epilepsy due to perinatal brain injury *UDANI, V (India)*	
	Infections, immunisation and epilepsy *SINGH, G (India)*	
	Head injury and post-traumatic epilepsy *SELLADURAI, B (Malaysia)*	
	Can epilepsy be prevented after the brain insult? *MOSHÉ, S L (USA)*	

SCIENTIFIC PROGRAMME

Saturday 23rd October

11:00 · 12:30 Post Main Session — Plenary Hall 2

Epilepsy prevention
Chairs: JAIN, S (India) / TAN, C-T (Malaysia)

Consanguinity and inherited epilepsies
JAIN, S (India)

Interaction of genetic and acquired factors in epilepsies
BERKOVIC, S F (Australia)

Clinical trials in antiepileptogenesis: the evidence
O'BRIEN, T (Australia)

Discussion panel: *UDANI, V (India), SINGH, G (India), SELLADURAI, B (Malaysia), MOSHÉ, S L (USA), BERKOVIC, S F (Australia), O'BRIEN, T (Australia)*

11:00 · 12:30 Parallel Session — Room 104

Global Campaign Against Epilepsy
Chairs: LEE, B-I (South Korea) / SOMERVILLE, E (Australia)

The Francophone Epilepsy Project in Laos
BARENNES, H (Lao People's Democratic Republic)

The China Demonstration Project: lessons learned
WU, J (China)

The East Timor Epilepsy Project: not as easy as it sounds
SOMERVILLE, E (Australia)

The Global Campaign Against Epilepsy: a global perspective
PERUCCA, E (Italy)

SCIENTIFIC PROGRAMME

Saturday 23rd October

11:00 · 12:30 Parallel Session Room 106

SUDEP
Chairs: LEUNG, H (Hong Kong) / SHEARS, G (Australia)

SUDEP: epidemiology and risk factors
SO, E (USA)

Seizures and the cardiorespiratory system
LEUNG, H (Hong Kong)

SUDEP counselling: how and when?
PANELLI, R (Australia)

11:00 · 12:30 Parallel Session Room 109

Genetics of large populations
Chairs: KWAN, P (Hong Kong) / MULLEN, S (Australia)

Association studies: trials and tribulations
TAN, N (Singapore)

Genetic variants associated with epilepsy causation
HIROSE, S (Japan)

Genome-wide association studies
MULLEN, S (Australia)

11:00 · 12:30 Parallel Session Room 110

Tuberous sclerosis and epilepsy
Chairs: UDANI, V (India) / HARVEY, S (Australia)

Epilepsy and related neurological disability in tuberous sclerosis
ORTIZ, M (Philippines)

Tuberous sclerosis and the mTOR pathway
KIM, K J (South Korea)

Epilepsy surgery for tuberous sclerosis
HARVEY, S (Australia)

SCIENTIFIC PROGRAMME

Saturday 23rd October

13:30 · 15:00 Satellite Symposium – UCB — Plenary Hall 2

Management of inadequately controlled epilepsy - evaluating the evidence
Chair: SOMERVILLE, E (Australia)

My journey: from uncontrolled epilepsy to control
CLIGNET, M (France)

Uncontrolled epilepsy – evidence-based decision making in clinical practice
BERKOVIC, S F (Australia)

What are the chances of the newer AEDs controlling seizures in chronic epilepsy?
SHORVON, S (United Kingdom)

Newer AEDs: what can we learn from post-marketing experience?
O'BRIEN, T (Australia)

15:00 · 16:00 Platform Session — Room 103

Refer to page 58

Paediatric epileptology
Chairs: VISUDTIBHAN, A (Thailand) / BYE, A (Australia)

15:00 · 16:00 Platform Session — Room 104

Refer to page 58

Surgery and neuroimaging
Chairs: JAIN, S (India) / CHINVARUN, Y (Thailand)

15:00 · 16:00 Platform Session — Room 106

Refer to page 59

Social and economic issues
Chairs: TAN, G (Singapore) / ORTIZ, M (Philippines)

15:00 · 16:00 Platform Session — Room 109

Refer to page 59

General epileptology
Chairs: GOURIE-DEVI, M (India) / GUNADARMA, S (Indonesia)

15:00 · 16:00 Platform Session — Room 110

Refer to page 60

Basic science
Chairs: FRENCH, C (Australia) / YAMAKAWA, K (Japan)

SCIENTIFIC PROGRAMME

Saturday 23rd October

16:30 · 17:30 Practical Session: Video Room 106

Is this an epileptic seizure?
Chairs: CHINVARUN, Y (Thailand) / WALKER, E (New Zealand)

Epileptic and non-epileptic events: presentation 1
WALKER, E (New Zealand)

Epileptic and non-epileptic events: presentation 2
CHINVARUN, Y (Thailand)

16:30 · 17:30 Practical Session: Debate Room 109

Surgery is the best way forward in refractory focal epilepsy
Chair: PERUCCA, E (Italy)

Surgery is the best way forward in refractory focal epilepsy: For
COOK, M (Australia)

Surgery is the best way forward in refractory focal epilepsy: Against
WIEBE, S (Canada)

16:30 · 17:30 Practical Session: Workshop Room 110

Management guidelines in epilepsy
Chairs: WU, J (China) / SAXENA, V (India)

The role of management guidelines in a developing country
JAIN, S (India)

The development of guidelines for epilepsy management in China
WU, J (China)

The development of Japanese guidelines through multisociety collaboration
AKAMATSU, N (Japan)

17:30 · 18:30 Platform Session Room 104

Refer to page 60

Translational research
Chairs: PETROU, S (Australia) / TANAKA, T (Japan)

SCIENTIFIC PROGRAMME

Saturday 23rd October

17:30 · 18:30 Platform Session Room 106

Refer to page 61

Genetics
Chairs: TAN, N (Singapore) / LIAO, W (China)

17:30 · 18:30 Platform Session Room 109

Refer to page 61

AED issues
Chairs: BERGIN, P (New Zealand) / RADHAKRISHNAN, K (India)

17:30 · 18:30 Platform Session Room 110

Refer to page 62

Surgery
Chairs: OTSUKI, T (Japan) / MURPHY, M (Australia)

SCIENTIFIC PROGRAMME

Sunday 24th October

Room 106	Room 109	Room 110
ASEPA Didactic Lecture: Surgical Treatment of Occipital Lobe Epilepsy: Basic and Clinical Approach *08:00- 08:45*		
Awards Ceremony *08:45-09:00*		
Main Session: Psychological Wellbeing in Epilepsy *09:00-10:30*		
Coffee Break - Exhibition Area *10:30-11:00*		
Post Main Session: Social and Economic Aspects of Epilepsy *11:00-12:30*	**Parallel Session:** Absence Epilepsies *11:00-12:30*	**Parallel Session:** Adverse Effects of Antiepileptic Drugs *11:00-12:30*

SCIENTIFIC PROGRAMME

Sunday 24th October

08:00 · 08:45	ASEPA Didactic Lecture	Room 106
	Surgery *Chair: AZIZ, H (Pakistan)*	
	Surgical treatment of occipital lobe epilepsy: basic and clinical approach *TANAKA, T (Japan)*	
08:45 · 09:00	Awards Ceremony	Room 106
09:00 · 10:30	Main Session	Room 106
	Psychological wellbeing in epilepsy *Chairs: GLYNN, M (Ireland) / O'BRIEN, T (Australia)*	
	Prevalence and nature of psychological disorders in epilepsy *INOUE, Y (Japan)*	
	Neurobiological links between epilepsy and mood disorders *SALZBERG, M (Australia)*	
	The effects of treatments on seizures and depression *KANNER, A M (USA)*	
	Interventions to reduce stigma in clinical practice *SCAMBLER, G (United Kingdom)*	

SCIENTIFIC PROGRAMME

Sunday 24th October

11:00 - 12:30 Post Main Session Room 106

Social and economic aspects of epilepsy
Chairs: GLYNN, M (Ireland) / TAN, G (Singapore)

Economic consequences of newly-diagnosed epilepsy
MARTINIUK, A (Australia)

Epilepsy and the workplace: risks, discrimination, insurance and disclosure
GLYNN, M (Ireland)

Social impacts of depression in epilepsy
SAXENA, V (India)

Discussion Panel: *INOUE, Y (Japan), SALZBERG, M (Australia), KANNER, A M (USA), SCAMBLER, G (United Kingdom), MARTINIUK, A (Australia), SAXENA, V (India)*

11:00 - 12:30 Parallel Session Room 109

Absence epilepsies
Chairs: LIAO, W (China) / VISUDTIBHAN, A (Thailand)

Epilepsies with absence seizures
SADLEIR, L (New Zealand)

Genetics of absence epilepsies
LIAO, W (China)

Drug treatment of absence epilepsies
PERUCCA, E (Italy)

11:00 - 12:30 Parallel Session Room 110

Adverse effects of antiepileptic drugs
Chairs: O'BRIEN, T (Australia) / GUNAWAN, D (Indonesia)

Bone health and fracture risk with antiepileptic drugs
PETTY, S (Australia)

Adverse effects of antiepileptic drugs on the unborn child
THOMAS, S V (India)

Hypersensitivity to antiepileptic drugs
LIM, K S (Malaysia)

The impact on cognition by phenobarbital in epilepsy treatment
HONG, Z (China)

SPEAKER INDEX

NAME	DATE	TIME	ROOM	SESSION TYPE	CHAIR/SPEAKER
AGRAWAL JP (NEPAL)	23/10/2010	08:15	Plenary Hall 2	ASEPA Didactic Lecture	Chair
AKAMATSU N (JAPAN)	23/10/2010	16:30	Room 110	Practical Session: Workshop	Speaker
ALEXANDER A (INDIA)	22/10/2010	15:00	Room 109	Platform	Speaker
ANDERSON C (AUSTRALIA)	23/10/2010	15:00	Room 109	Platform	Speaker
ARAVIND KUMAR R (INDIA)	23/10/2010	15:00	Room 109	Platform	Speaker
AZIZ H (PAKISTAN)	24/10/2010	08:00	Room 106	ASEPA Didactic Lecture	Chair
BABA H (JAPAN)	23/10/2010	17:30	Room 110	Platform	Speaker
BARENNES H (LAO PEOPLE'S DEMOCRATIC REPUBLIC)	23/10/2010	11:00	Room 104	Parallel Session	Speaker
BELLON M (AUSTRALIA)	21/10/2010	11:00	Room 106	Parallel Discussion Group	Speaker
BERGIN P (NEW ZEALAND)	21/10/2010	16:30	Plenary Hall 2	Special Session	Speaker
BERGIN P (NEW ZEALAND)	23/10/2010	15:00	Room 109	Platform	Speaker
BERGIN P (NEW ZEALAND)	23/10/2010	17:30	Room 109	Platform	Chair
BERKOVIC SF (AUSTRALIA)	21/10/2010	16:30	Plenary Hall 2	Special Session	Speaker
BERKOVIC SF (AUSTRALIA)	22/10/2010	16:30	Room 109	Practical Session: Debate	Chair
BERKOVIC SF (AUSTRALIA)	23/10/2010	07:30	Plenary Hall 2	ASEPA Didactic Lecture	Speaker
BERKOVIC SF (AUSTRALIA)	23/10/2010	11:00	Plenary Hall 2	Post Main Session	Speaker
BERKOVIC SF (AUSTRALIA)	23/10/2010	13:30	Plenary Hall 2	Satellite Symposium	Speaker
BERTRAM E (USA)	20/10/2010	13:00	Room 109	ASEPA Workshop: Basic Science	Speaker
BLEASEL A (AUSTRALIA)	20/10/2010	13:30	Room 110	ASEPA Workshop: Clinical Epilepsy	Chair
BLEASEL A (AUSTRALIA)	20/10/2010	13:30	Room 110	ASEPA Workshop: Clinical Epilepsy	Speaker
BLEASEL A (AUSTRALIA)	21/10/2010	14:00	Plenary Hall 2	Chairmans Symposium	Speaker
BLEASEL A (AUSTRALIA)	22/10/2010	13:30	Plenary Hall 2	Satellite Symposium	Chair
BOWDEN S (AUSTRALIA)	21/10/2010	08:30	Room 110	ASEPA Workshop: Clinical Epilepsy	Speaker
BYE A (AUSTRALIA)	23/10/2010	15:00	Room 103	Platform	Chair
CABRAL LIM L (PHILIPPINES)	22/10/2010	07:30	Plenary Hall 2	ASEPA Didactic Lecture	Chair
CANUET L (JAPAN)	22/10/2010	15:00	Room 109	Platform	Speaker
CHAMBLISS J (AUSTRALIA)	21/10/2010	15:30	Room 104	Parallel Discussion Group	Speaker
CHANG W-L (CHINA)	23/10/2010	17:30	Room 104	Platform	Speaker
CHAPMAN D (AUSTRALIA)	21/10/2010	13:30	Room 104	Parallel Session	Chair
CHEN L (AUSTRALIA)	22/10/2010	15:00	Room 106	Platform	Speaker
CHINVARUN Y (THAILAND)	20/10/2010	13:30	Room 110	ASEPA Workshop: Clinical Epilepsy	Chair
CHINVARUN Y (THAILAND)	20/10/2010	13:30	Room 110	ASEPA Workshop: Clinical Epilepsy	Speaker
CHINVARUN Y (THAILAND)	23/10/2010	15:00	Room 104	Platform	Chair
CHINVARUN Y (THAILAND)	23/10/2010	16:30	Room 106	Practical Session: Video	Chair
CHINVARUN Y (THAILAND)	23/10/2010	16:30	Room 106	Practical Session: Video	Presenter
CHO Y (SOUTH KOREA)	22/10/2010	16:30	Room 110	Practical Session: Workshop	Speaker
CLARKE A (AUSTRALIA)	23/10/2010	15:00	Room 106	Platform	Speaker
CLIGNET M (FRANCE)	21/10/2010	15:30	Room 103	Parallel Discussion Group	Speaker
CLIGNET M (FRANCE)	23/10/2010	13:30	Plenary Hall 2	Satellite Symposium	Speaker
COLE R (AUSTRALIA)	21/10/2010	13:30	Room 106	Parallel Session	Chair
COLE R (AUSTRALIA)	21/10/2010	15:30	Room 103	Parallel Discussion Group	Chair
CONNELLY A (AUSTRALIA)	22/10/2010	11:00	Room 106	Parallel Session	Speaker
COOK M (AUSTRALIA)	22/10/2010	15:00	Room 104	Platform	Chair
COOK M (AUSTRALIA)	22/10/2010	16:30	Room 110	Practical Session: Workshop	Speaker
COOK M (AUSTRALIA)	22/10/2010	17:30	Plenary Hall 2	Satellite Symposium	Speaker
COOK M (AUSTRALIA)	23/10/2010	16:30	Room 109	Practical Session: Debate	Speaker
CUMMINS J (AUSTRALIA)	21/10/2010	11:00	Room 106	Parallel Discussion Group	Chair
D'SOUZA W (AUSTRALIA)	21/10/2010	16:30	Plenary Hall 2	Special Session	Chair
D'SOUZA W (AUSTRALIA)	21/10/2010	16:30	Plenary Hall 2	Special Session	Speaker
DU P (CHINA)	23/10/2010	17:30	Room 104	Platform	Speaker
DUNNE J (AUSTRALIA)	22/10/2010	09:00	Plenary Hall 2	Main Session	Chair
DUNNE J (AUSTRALIA)	22/10/2010	11:00	Room 109	Parallel Session	Speaker
FAULKNER HJ (AUSTRALIA)	22/10/2010	15:00	Room 106	Platform	Speaker
FEDERICO P (CANADA)	22/10/2010	15:00	Room 104	Platform	Speaker
FRENCH C (AUSTRALIA)	20/10/2010	13:00	Room 109	ASEPA Workshop: Basic Science	Chair
FRENCH C (AUSTRALIA)	20/10/2010	13:00	Room 109	ASEPA Workshop: Basic Science	Speaker
FRENCH C (AUSTRALIA)	23/10/2010	15:00	Room 110	Platform	Chair
GAMAGE R (SRI LANKA)	22/10/2010	08:15	Plenary Hall 2	ASEPA Didactic Lecture	Chair
GARCIA PG (AUSTRALIA)	23/10/2010	15:00	Room 110	Platform	Speaker
GHAEMI K (IRAN)	23/10/2010	17:30	Room 110	Platform	Speaker
GILL D (AUSTRALIA)	22/10/2010	16:30	Room 106	Practical Session: Video	Chair

SPEAKER INDEX

NAME	DATE	TIME	ROOM	SESSION TYPE	CHAIR/SPEAKER
GILL D (AUSTRALIA)	22/10/2010	16:30	Room 106	Practical Session: Video	Presenter
GLYNN M (IRELAND)	21/10/2010	08:45	Room 106	Welcome	Chair
GLYNN M (IRELAND)	22/10/2010	11:00	Room 109	Parallel Session	Chair
GLYNN M (IRELAND)	24/10/2010	09:00	Room 106	Main Session	Chair
GLYNN M (IRELAND)	24/10/2010	11:00	Room 106	Post Main Session	Chair
GLYNN M (IRELAND)	24/10/2010	11:00	Room 106	Post Main Session	Speaker
GOEL D (INDIA)	23/10/2010	15:00	Room 109	Platform	Speaker
GOURIE-DEVI M (INDIA)	23/10/2010	15:00	Room 109	Platform	Chair
GUNADARMA S (INDONESIA)	23/10/2010	15:00	Room 109	Platform	Chair
GUNAWAN D (INDONESIA)	24/10/2010	11:00	Room 110	Parallel Session	Chair
GUTIERREZ J (PHILIPPINES)	22/10/2010	15:00	Room 106	Platform	Chair
HARVEY S (AUSTRALIA)	21/10/2010	14:00	Plenary Hall 2	Chairmans Symposium	Chair
HARVEY S (AUSTRALIA)	23/10/2010	11:00	Room 110	Parallel Session	Chair
HARVEY S (AUSTRALIA)	23/10/2010	11:00	Room 110	Parallel Session	Speaker
HERON SE (AUSTRALIA)	23/10/2010	17:30	Room 106	Platform	Speaker
HIRANO Y (JAPAN)	23/10/2010	15:00	Room 103	Platform	Speaker
HIROSE S (JAPAN)	20/10/2010	13:00	Room 109	ASEPA Workshop: Basic Science	Speaker
HIROSE S (JAPAN)	23/10/2010	11:00	Room 109	Parallel Session	Speaker
HONG Z (CHINA)	23/10/2010	15:00	Room 106	Platform	Speaker
HONG Z (CHINA)	24/10/2010	11:00	Room 110	Parallel Session	Speaker
HUGUENARD J (USA)	20/10/2010	13:00	Room 109	ASEPA Workshop: Basic Science	Speaker
HUGUENARD J (USA)	22/10/2010	15:00	Room 110	Platform	Chair
HUGUENARD J (USA)	22/10/2010	15:00	Room 110	Platform	Speaker
IKEDA A (JAPAN)	22/10/2010	16:30	Room 106	Practical Session: Video	Chair
IKEDA A (JAPAN)	22/10/2010	16:30	Room 106	Practical Session: Video	Presenter
INOUE Y (JAPAN)	21/10/2010	08:30	Room 110	ASEPA Workshop: Clinical Epilepsy	Speaker
INOUE Y (JAPAN)	24/10/2010	09:00	Room 106	Main Session	Speaker
JACKSON G (AUSTRALIA)	22/10/2010	11:00	Room 106	Parallel Session	Chair
JACKSON G (AUSTRALIA)	22/10/2010	11:00	Room 106	Parallel Session	Speaker
JACKSON G (AUSTRALIA)	22/10/2010	16:30	Room 109	Practical Session: Debate	Speaker
JAIN S (INDIA)	23/10/2010	11:00	Plenary Hall 2	Post Main Session	Chair
JAIN S (INDIA)	23/10/2010	11:00	Plenary Hall 2	Post Main Session	Speaker
JAIN S (INDIA)	23/10/2010	15:00	Room 104	Platform	Chair
JAIN S (INDIA)	23/10/2010	16:30	Room 110	Practical Session: Workshop	Speaker
JANARDHAN K (INDIA)	21/10/2010	15:30	Room 104	Parallel Discussion Group	Speaker
JIANG W (CHINA)	22/10/2010	15:00	Room 110	Platform	Speaker
JIN L (CHINA)	22/10/2010	15:00	Room 106	Platform	Speaker
JONES N (AUSTRALIA)	20/10/2010	13:00	Room 109	ASEPA Workshop: Basic Science	Speaker
JONES S (UNITED KINGDOM)	21/10/2010	15:30	Room 106	Parallel Discussion Group	Speaker
KANEKO S (JAPAN)	22/10/2010	13:30	Plenary Hall 2	Satellite Symposium	Chair
KANNER AM (USA)	21/10/2010	08:30	Room 110	ASEPA Workshop: Clinical Epilepsy	Speaker
KANNER AM (USA)	22/10/2010	15:00	Room 109	Platform	Chair
KANNER AM (USA)	23/10/2010	08:15	Plenary Hall 2	ASEPA Didactic Lecture	Speaker
KANNER AM (USA)	24/10/2010	09:00	Room 106	Main Session	Speaker
KATO AM (JAPAN)	23/10/2010	17:30	Room 106	Platform	Speaker
KAZUAKI K (JAPAN)	20/10/2010	13:00	Room 109	ASEPA Workshop: Basic Science	Speaker
KENNARD JT (AUSTRALIA)	22/10/2010	15:00	Room 110	Platform	Speaker
KIM KJ (SOUTH KOREA)	23/10/2010	11:00	Room 110	Parallel Session	Speaker
KLEIN KM (AUSTRALIA)	23/10/2010	17:30	Room 106	Platform	Speaker
KOE AS (AUSTRALIA)	23/10/2010	17:30	Room 104	Platform	Speaker
KRISHNAMOORTHY ES (INDIA)	21/10/2010	08:30	Room 110	ASEPA Workshop: Clinical Epilepsy	Chair
KRISHNAMOORTHY ES (INDIA)	21/10/2010	08:30	Room 110	ASEPA Workshop: Clinical Epilepsy	Speaker
KRISHNAMOORTHY ES (INDIA)	22/10/2010	15:00	Room 109	Platform	Chair
KWAN P (HONG KONG)	21/10/2010	16:30	Plenary Hall 2	Special Session	Chair
KWAN P (HONG KONG)	21/10/2010	16:30	Plenary Hall 2	Special Session	Speaker
KWAN P (HONG KONG)	22/10/2010	07:30	Plenary Hall 2	ASEPA Didactic Lecture	Speaker
KWAN P (HONG KONG)	22/10/2010	09:00	Plenary Hall 2	Main Session	Speaker
KWAN P (HONG KONG)	23/10/2010	11:00	Room 109	Parallel Session	Chair
LAI S (TAIWAN)	21/10/2010	09:00	Room 106	Main Session	Chair
LAI S (TAIWAN)	22/10/2010	09:00	Plenary Hall 2	Main Session	Chair
LAI S (TAIWAN)	22/10/2010	11:00	Plenary Hall 2	Post Main Session	Chair
LAWN N (AUSTRALIA)	21/10/2010	08:30	Room 110	ASEPA Workshop: Clinical Epilepsy	Speaker

SPEAKER INDEX

NAME	DATE	TIME	ROOM	SESSION TYPE	CHAIR/SPEAKER
LAWN N (AUSTRALIA)	22/10/2010	11:00	Plenary Hall 2	Post Main Session	Speaker
LEE B-I (SOUTH KOREA)	21/10/2010	14:00	Plenary Hall 2	Chairmans Symposium	Speaker
LEE B-I (SOUTH KOREA)	22/10/2010	11:00	Room 106	Parallel Session	Chair
LEE B-I (SOUTH KOREA)	22/10/2010	16:30	Room 110	Practical Session: Workshop	Chair
LEE B-I (SOUTH KOREA)	23/10/2010	11:00	Room 104	Parallel Session	Chair
LEE SK (SOUTH KOREA)	20/10/2010	13:30	Room 110	ASEPA Workshop: Clinical Epilepsy	Speaker
LEE SK (SOUTH KOREA)	22/10/2010	11:00	Room 106	Parallel Session	Speaker
LEE S-A (SOUTH KOREA)	23/10/2010	17:30	Room 109	Platform	Speaker
LEE Y-M (SOUTH KOREA)	22/10/2010	11:00	Room 110	Parallel Session	Speaker
LEPPIK I (USA)	22/10/2010	13:30	Plenary Hall 2	Satellite Symposium	Speaker
LEUNG H (HONG KONG)	22/10/2010	15:00	Room 104	Platform	Speaker
LEUNG H (HONG KONG)	23/10/2010	11:00	Room 106	Parallel Session	Chair
LEUNG H (HONG KONG)	23/10/2010	11:00	Room 106	Parallel Session	Speaker
LEWIS W (AUSTRALIA)	21/10/2010	09:00	Room 106	Main Session	Speaker
LIAO W (CHINA)	23/10/2010	17:30	Room 106	Platform	Chair
LIAO W (CHINA)	24/10/2010	11:00	Room 109	Parallel Session	Chair
LIAO W (CHINA)	24/10/2010	11:00	Room 109	Parallel Session	Speaker
LIM KS (MALAYSIA)	21/10/2010	16:30	Plenary Hall 2	Special Session	Speaker
LIM KS (MALAYSIA)	22/10/2010	15:00	Room 109	Platform	Speaker
LIM KS (MALAYSIA)	24/10/2010	11:00	Room 110	Parallel Session	Speaker
LIM SH (SINGAPORE)	21/10/2010	14:00	Plenary Hall 2	Chairmans Symposium	Chair
LIM SH (SINGAPORE)	22/10/2010	11:00	Plenary Hall 2	Post Main Session	Speaker
LOCHARERNKUL C (THAILAND)	20/10/2010	13:30	Room 110	ASEPA Workshop: Clinical Epilepsy	Speaker
LOCHARERNKUL C (THAILAND)	22/10/2010	15:00	Room 104	Platform	Chair
LUAN G (CHINA)	20/10/2010	13:30	Room 110	ASEPA Workshop: Clinical Epilepsy	Speaker
LUAN G (CHINA)	23/10/2010	17:30	Room 110	Platform	Speaker
MAHDAVI N (AUSTRALIA)	22/10/2010	15:00	Room 109	Platform	Speaker
MALLA BR (INDIA)	23/10/2010	17:30	Room 110	Platform	Speaker
MANNAN M (BANGLADESH)	23/10/2010	07:30	Plenary Hall 2	ASEPA Didactic Lecture	Chair
MARTINIUK A (AUSTRALIA)	24/10/2010	11:00	Room 106	Post Main Session	Speaker
MEHNDIRATTA M (INDIA)	21/10/2010	13:30	Room 106	Parallel Session	Speaker
MENON RN (INDIA)	23/10/2010	17:30	Room 110	Platform	Speaker
MOHAMED AR (AUSTRALIA)	22/10/2010	13:30	Plenary Hall 2	Satellite Symposium	Speaker
MOHAMED AR (AUSTRALIA)	23/10/2010	15:00	Room 104	Platform	Speaker
MOSHÉ SL (USA)	22/10/2010	08:15	Plenary Hall 2	ASEPA Didactic Lecture	Speaker
MOSHÉ SL (USA)	23/10/2010	09:00	Plenary Hall 2	Main Session	Speaker
MU J (CHINA)	23/10/2010	15:00	Room 109	Platform	Speaker
MULLEN S (AUSTRALIA)	23/10/2010	11:00	Room 109	Parallel Session	Chair
MULLEN S (AUSTRALIA)	23/10/2010	11:00	Room 109	Parallel Session	Speaker
MURAKAMI H (JAPAN)	23/10/2010	15:00	Room 104	Platform	Speaker
MURPHY M (AUSTRALIA)	23/10/2010	17:30	Room 110	Platform	Chair
NADEBAUM C (AUSTRALIA)	23/10/2010	17:30	Room 109	Platform	Speaker
NAKAGAWA E (JAPAN)	23/10/2010	17:30	Room 106	Platform	Speaker
NAKASATO N (JAPAN)	20/10/2010	13:30	Room 110	ASEPA Workshop: Clinical Epilepsy	Speaker
NG C (AUSTRALIA)	23/10/2010	15:00	Room 110	Platform	Speaker
NITTA N (JAPAN)	23/10/2010	15:00	Room 110	Platform	Speaker
O'BRIEN T (AUSTRALIA)	20/10/2010	13:00	Room 109	ASEPA Workshop: Basic Science	Chair
O'BRIEN T (AUSTRALIA)	20/10/2010	13:00	Room 109	ASEPA Workshop: Basic Science	Speaker
O'BRIEN T (AUSTRALIA)	22/10/2010	17:30	Plenary Hall 2	Satellite Symposium	Chair
O'BRIEN T (AUSTRALIA)	23/10/2010	11:00	Plenary Hall 2	Post Main Session	Speaker
O'BRIEN T (AUSTRALIA)	23/10/2010	13:30	Plenary Hall 2	Satellite Symposium	Speaker
O'BRIEN T (AUSTRALIA)	24/10/2010	09:00	Room 106	Main Session	Chair
O'BRIEN T (AUSTRALIA)	24/10/2010	11:00	Room 110	Parallel Session	Chair
OGUNI H (JAPAN)	23/10/2010	15:00	Room 103	Platform	Speaker
ORTIZ M (PHILIPPINES)	22/10/2010	11:00	Room 110	Parallel Session	Chair
ORTIZ M (PHILIPPINES)	23/10/2010	11:00	Room 110	Parallel Session	Speaker
ORTIZ M (PHILIPPINES)	23/10/2010	15:00	Room 106	Platform	Chair
OSAWA M (JAPAN)	22/10/2010	13:30	Plenary Hall 2	Satellite Symposium	Speaker
OTSUKI T (JAPAN)	23/10/2010	17:30	Room 110	Platform	Chair
PANELLI R (AUSTRALIA)	21/10/2010	13:30	Room 104	Parallel Session	Speaker
PANELLI R (AUSTRALIA)	23/10/2010	11:00	Room 106	Parallel Session	Speaker
PERUCCA E (ITALY)	23/10/2010	11:00	Room 104	Parallel Session	Speaker

SPEAKER INDEX

NAME	DATE	TIME	ROOM	SESSION TYPE	CHAIR/SPEAKER
PERUCCA E (ITALY)	23/10/2010	16:30	Room 109	Practical Session: Debate	Chair
PERUCCA E (ITALY)	24/10/2010	11:00	Room 109	Parallel Session	Speaker
PETROU S (AUSTRALIA)	20/10/2010	13:00	Room 109	ASEPA Workshop: Basic Science	Speaker
PETROU S (AUSTRALIA)	23/10/2010	17:30	Room 104	Platform	Chair
PETTY S (AUSTRALIA)	24/10/2010	11:00	Room 110	Parallel Session	Speaker
PFEIFFER W (AUSTRALIA)	21/10/2010	13:30	Room 106	Parallel Session	Speaker
PINIKAHANA J (AUSTRALIA)	23/10/2010	15:00	Room 106	Platform	Speaker
PRESTON J (UNITED KINGDOM)	21/10/2010	13:30	Room 104	Parallel Session	Speaker
PRIMARDI A (INDONESIA)	23/10/2010	15:00	Room 106	Platform	Speaker
RADHAKRISHNAN A (INDIA)	22/10/2010	15:00	Room 104	Platform	Speaker
RADHAKRISHNAN K (INDIA)	20/10/2010	13:30	Room 110	ASEPA Workshop: Clinical Epilepsy	Speaker
RADHAKRISHNAN K (INDIA)	21/10/2010	14:00	Plenary Hall 2	Chairmans Symposium	Speaker
RADHAKRISHNAN K (INDIA)	23/10/2010	17:30	Room 109	Platform	Chair
RAMESHA KN (JAPAN)	23/10/2010	15:00	Room 104	Platform	Speaker
RAYNER G (AUSTRALIA)	22/10/2010	15:00	Room 109	Platform	Speaker
REID C (AUSTRALIA)	22/10/2010	15:00	Room 110	Platform	Chair
REID C (AUSTRALIA)	23/10/2010	17:30	Room 104	Platform	Speaker
RINEY K (AUSTRALIA)	23/10/2010	15:00	Room 103	Platform	Speaker
ROSEMERGY I (NEW ZEALAND)	22/10/2010	15:00	Room 106	Platform	Speaker
SADLEIR L (NEW ZEALAND)	24/10/2010	11:00	Room 109	Parallel Session	Speaker
SALING M (AUSTRALIA)	21/10/2010	08:30	Room 110	ASEPA Workshop: Clinical Epilepsy	Chair
SALING M (AUSTRALIA)	21/10/2010	08:30	Room 110	ASEPA Workshop: Clinical Epilepsy	Speaker
SALZBERG M (AUSTRALIA)	21/10/2010	15:30	Room 106	Parallel Discussion Group	Speaker
SALZBERG M (AUSTRALIA)	24/10/2010	09:00	Room 106	Main Session	Speaker
SANTOS RP (PHILIPPINES)	23/10/2010	17:30	Room 109	Platform	Speaker
SAXENA V (INDIA)	21/10/2010	09:00	Room 106	Main Session	Chair
SAXENA V (INDIA)	23/10/2010	09:00	Plenary Hall 2	Main Session	Chair
SAXENA V (INDIA)	23/10/2010	16:30	Room 110	Practical Session: Workshop	Chair
SAXENA V (INDIA)	24/10/2010	11:00	Room 106	Post Main Session	Speaker
SCAMBLER G (UNITED KINGDOM)	21/10/2010	09:00	Room 106	Main Session	Speaker
SCAMBLER G (UNITED KINGDOM)	24/10/2010	09:00	Room 106	Main Session	Speaker
SCHEFFER I (AUSTRALIA)	21/10/2010	16:30	Plenary Hall 2	Special Session	Speaker
SCHEFFER I (AUSTRALIA)	22/10/2010	11:00	Room 110	Parallel Session	Chair
SCHEFFER I (AUSTRALIA)	22/10/2010	11:00	Room 110	Parallel Session	Speaker
SELLADURAI B (MALAYSIA)	23/10/2010	09:00	Plenary Hall 2	Main Session	Speaker
SHEARS G (AUSTRALIA)	21/10/2010	08:45	Room 106	Welcome	Chair
SHEARS G (AUSTRALIA)	21/10/2010	15:30	Room 104	Parallel Discussion Group	Chair
SHEARS G (AUSTRALIA)	23/10/2010	11:00	Room 106	Parallel Session	Chair
SHORVON S (UNITED KINGDOM)	21/10/2010	15:30	Plenary Hall 2	Special Lecture	Speaker
SHORVON S (UNITED KINGDOM)	22/10/2010	16:30	Room 109	Practical Session: Debate	Speaker
SHORVON S (UNITED KINGDOM)	23/10/2010	13:30	Plenary Hall 2	Satellite Symposium	Speaker
SHU H-F (CHINA)	22/10/2010	15:00	Room 110	Platform	Speaker
SINGH A (INDIA)	23/10/2010	15:00	Room 104	Platform	Speaker
SINGH G (INDIA)	23/10/2010	09:00	Plenary Hall 2	Main Session	Speaker
SO E (USA)	21/10/2010	13:30	Room 104	Parallel Session	Speaker
SO E (USA)	22/10/2010	09:00	Plenary Hall 2	Main Session	Speaker
SO E (USA)	23/10/2010	11:00	Room 106	Parallel Session	Speaker
SOMERVILLE E (AUSTRALIA)	21/10/2010	15:30	Room 103	Parallel Discussion Group	Speaker
SOMERVILLE E (AUSTRALIA)	22/10/2010	11:00	Room 109	Parallel Session	Chair
SOMERVILLE E (AUSTRALIA)	22/10/2010	11:00	Room 109	Parallel Session	Speaker
SOMERVILLE E (AUSTRALIA)	23/10/2010	11:00	Room 104	Parallel Session	Chair
SOMERVILLE E (AUSTRALIA)	23/10/2010	11:00	Room 104	Parallel Session	Speaker
SOMERVILLE E (AUSTRALIA)	23/10/2010	13:30	Plenary Hall 2	Satellite Symposium	Chair
SRINIVAS H (INDIA)	21/10/2010	13:30	Room 106	Parallel Session	Chair
SUGAI K (JAPAN)	23/10/2010	15:00	Room 103	Platform	Speaker
SUKIGARA S (JAPAN)	22/10/2010	15:00	Room 104	Platform	Speaker
SUHR C (AUSTRALIA)	23/10/2010	17:30	Room 109	Platform	Speaker
TAKAHASHI T (JAPAN)	22/10/2010	11:00	Plenary Hall 2	Post Main Session	Chair
TAN C-T (MALAYSIA)	21/10/2010	15:30	Plenary Hall 2	Special Lecture	Chair
TAN C-T (MALAYSIA)	23/10/2010	09:00	Plenary Hall 2	Main Session	Chair
TAN C-T (MALAYSIA)	23/10/2010	11:00	Plenary Hall 2	Post Main Session	Chair
TAN G (SINGAPORE)	21/10/2010	13:30	Room 106	Parallel Session	Speaker

SPEAKER INDEX

NAME	DATE	TIME	ROOM	SESSION TYPE	CHAIR/SPEAKER
TAN G (SINGAPORE)	23/10/2010	15:00	Room 106	Platform	Chair
TAN G (SINGAPORE)	24/10/2010	11:00	Room 106	Post Main Session	Chair
TAN N (SINGAPORE)	23/10/2010	11:00	Room 109	Parallel Session	Speaker
TAN N (SINGAPORE)	23/10/2010	17:30	Room 106	Platform	Chair
TANAKA T (JAPAN)	23/10/2010	17:30	Room 104	Platform	Chair
TANAKA T (JAPAN)	24/10/2010	08:00	Room 106	ASEPA Didactic Lecture	Speaker
THOMAS SV (INDIA)	24/10/2010	11:00	Room 110	Parallel Session	Speaker
TIAMKAO S (THAILAND)	23/10/2010	17:30	Room 106	Platform	Speaker
TODARO M (AUSTRALIA)	23/10/2010	17:30	Room 109	Platform	Speaker
TOHYAMA J (JAPAN)	23/10/2010	15:00	Room 103	Platform	Speaker
TSAI J-J (TAIWAN)	22/10/2010	11:00	Room 109	Parallel Session	Speaker
TSAI J-J (TAIWAN)	22/10/2010	15:00	Room 106	Platform	Chair
TSENG Y-F (TAIWAN)	21/10/2010	13:30	Room 104	Parallel Session	Chair
UDANI V (INDIA)	23/10/2010	09:00	Plenary Hall 2	Main Session	Speaker
UDANI V (INDIA)	23/10/2010	11:00	Room 110	Parallel Session	Chair
VAJDA F (AUSTRALIA)	22/10/2010	17:30	Plenary Hall 2	Satellite Symposium	Speaker
VAN RAAY L (AUSTRALIA)	23/10/2010	17:30	Room 104	Platform	Speaker
VISUDTIBHAN A (THAILAND)	22/10/2010	11:00	Plenary Hall 2	Post Main Session	Speaker
VISUDTIBHAN A (THAILAND)	23/10/2010	15:00	Room 103	Platform	Chair
VISUDTIBHAN A (THAILAND)	24/10/2010	11:00	Room 109	Parallel Session	Chair
VOGRIN SJ (AUSTRALIA)	22/10/2010	15:00	Room 104	Platform	Speaker
VOLLENHOVEN B (AUSTRALIA)	21/10/2010	11:00	Room 104	Parallel Discussion Group	Speaker
WAKAMOTO H (JAPAN)	22/10/2010	11:00	Room 110	Parallel Session	Speaker
WALKER C (AUSTRALIA)	21/10/2010	11:00	Room 104	Parallel Discussion Group	Chair
WALKER E (NEW ZEALAND)	23/10/2010	16:30	Room 106	Practical Session: Video	Chair
WALKER E (NEW ZEALAND)	23/10/2010	16:30	Room 106	Practical Session: Video	Presenter
WANG W (CHINA)	22/10/2010	09:00	Plenary Hall 2	Main Session	Speaker
WANG Y (CHINA)	22/10/2010	16:30	Room 110	Practical Session: Workshop	Chair
WANG Y (CHINA)	22/10/2010	16:30	Room 110	Practical Session: Workshop	Speaker
WEINTROB D (AUSTRALIA)	21/10/2010	11:00	Room 106	Parallel Discussion Group	Speaker
WHITEHEAD H (AUSTRALIA)	21/10/2010	15:30	Room 106	Parallel Discussion Group	Chair
WIEBE S (CANADA)	21/10/2010	14:00	Plenary Hall 2	Chairmans Symposium	Speaker
WIEBE S (CANADA)	23/10/2010	16:30	Room 109	Practical Session: Debate	Speaker
WILLOUGHBY J (AUSTRALIA)	20/10/2010	13:00	Room 109	ASEPA Workshop: Basic Science	Speaker
WILSON S (AUSTRALIA)	21/10/2010	11:00	Room 104	Parallel Discussion Group	Speaker
WILSON S (AUSTRALIA)	22/10/2010	09:00	Plenary Hall 2	Main Session	Speaker
WU J (CHINA)	23/10/2010	11:00	Room 104	Parallel Session	Speaker
WU J (CHINA)	23/10/2010	16:30	Room 110	Practical Session: Workshop	Chair
WU J (CHINA)	23/10/2010	16:30	Room 110	Practical Session: Workshop	Speaker
YAMAKAWA K (JAPAN)	20/10/2010	13:00	Room 109	ASEPA Workshop: Basic Science	Speaker
YAMAKAWA K (JAPAN)	23/10/2010	15:00	Room 110	Platform	Chair
YU P (CHINA)	23/10/2010	15:00	Room 106	Platform	Speaker
ZHANG C-Q (CHINA)	23/10/2010	15:00	Room 110	Platform	Speaker
ZENG Z (AUSTRALIA)	23/10/2010	15:00	Room 110	Platform	Speaker

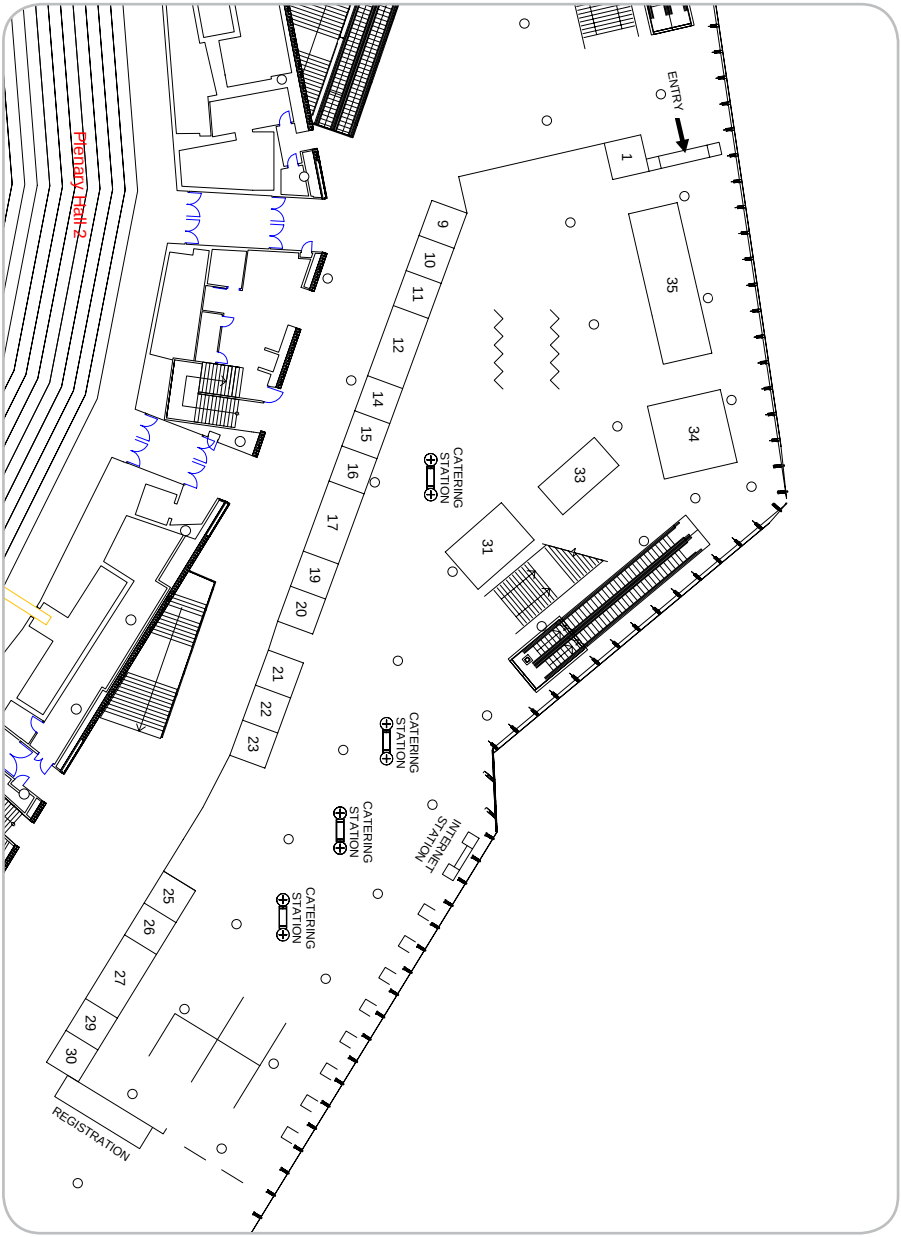
EXHIBITION INFORMATION

Exhibition Opening Hours

Friday 22nd October	**09:00 - 17:00**
Saturday 23rd October	**09:00 - 17:00**
Sunday 24th October	**09:00 - 12:00**

EXHIBITOR	STAND NO.
Ad Tech	9
13th Asian Oceanian Congress of Neurology	19
Aurora BioScience	21
Cadwell	15
Carefusion	17
Compumedics	12
Congress Recording · Medical Update	29
EGI	1
Eisai Co. Ltd & Dainippon Sumitomo Pharma Co. Ltd	33
EPI-Assist	16
Future Meetings	27
Global Campaign Against Epilepsy	35
International Bureau For Epilepsy	35
International Leauge Against Epilepsy	35
Janssen-Cilag	10
Joint Epilepsy Council of Australia	25
Melbourne Convention + Visitors Bureau	11
Nihon Kohden	20
Novartis	14
Pharmigene	26
PMT	23
Sanofi-Aventis	31
Tours	30
UCB	34

EXHIBITION INFORMATION



PLATFORM SESSIONS

Neuroimaging
Friday 22nd October
15:00-16:00
Room 104
Refer to pages 84-86 for abstracts

001 **Source localization of IEDs from simultaneous high-density EEG and fMRI in focal epilepsy**
VOGRIN SJ[1], LAU S[1,2], KUHLMANN L[1], D'SOUZA W[1], HAUEISEN J[2], COOK MJ[1] ([1]Australia, [2]Germany)

002 **Pre- and post-surgical investigation of cerebral blood flow using NIRS in patients with hemispherical cortical dysplasia**
SUKIGARA S, NAKAGAWA E, AIZAKI K, ISHIYAMA A, TAKESHITA E, OKAZAKI T, HIYANE M, FUKUMURA S, SAKUMA H, SAITO Y, KOMAKI H, SUGAI K, SASAKI M, TAKAHASHI A, OTSUKI T, KOBAYASHI I (Japan)

003 **Voxel-based morphometric study of white matter tracts in patients with non-lesional temporal lobe epilepsy**
LEUNG H, WANG RD, SHI L, LAM WW, KWAN P, WONG KS, MOK V (Hong Kong)

004 **Voxel-based optimized morphometry (VBM) in the identification of focal cortical dysplasias (FCDs) in patients with refractory seizures**
RADHAKRISHNAN A, JAMES JS, KESAVADAS C, THOMAS B, RADHAKRISHNAN K (India)

005 **Intracranial EEG-fMRI study of focal epileptiform discharges in humans: evidence of a default state**
CUNNINGHAM C, GOODYEAR B, BADAWY R, PITTMAN D, FEDERICO P (Canada)

Clinical neurophysiology and seizure semiology
Friday 22nd October
15:00-16:00
Room 106
Refer to pages 86-88 for abstracts

006 **Use of post ictal respiratory pattern to discriminate between convulsive non-epileptic seizures and generalised tonic clonic seizures**
ROSEMERGY I, FRITH R, HERATH S, WALKER E (New Zealand)

007 **Lateralizing value of ictal head deviation in mesial temporal lobe epilepsy**
JIN L, WU L (China)

008 **Latency to first interictal epileptiform discharge in epilepsy with outpatient ambulatory EEG recording**
FAULKNER HJ, MOHAMED A (Australia)

009 **The utility of one day video EEG (VEEG) recording in the diagnosis of non-epileptic seizures**
CHEN L, KHUN TT, WONG TH, BLYTH K, BLEASEL A (Australia)

PLATFORM SESSIONS

Psychiatry and neuropsychology
Friday 22nd October
15:00-16:00
Room 109
Refer to pages 88-91 for abstracts

010 **Differential contributions of objective memory and mood to subjective memory complaints in refractory focal epilepsy**
RAYNER G, WRENCH JM, WILSON SJ (Australia)

011 **Working memory-induced activation and severity of psychopathology in schizophrenia-like psychosis of epilepsy: evidence from magnetoencephalography**
CANUET L[1], ISHII R[1], IWASE M[1], IKEZAWA K[1], KURIMOTO R[1], TAKAHASHI H[1], CURRAIS A[2], AZECHI M[1], AOKI Y[1], NAKAHACHI T[1], SORIANO S[3], TAKEDA M[1] ([1]Japan, [2]United Kingdom, [3]USA)

012 **Memory changes in individuals with childhood-onset lesional TLE: a follow-up study**
MAHDAVI N, GONZALEZ L, ANDERSON V (Australia)

013 **Is there 'phantom of dementia' in elderly who have undergone ATL for refractory MTLE?**
ALEXANDER A, RADHAKRISHNAN A, RADHAKRISHNAN K (India)

014 **Psychogenic non-epileptic seizures (PNES): can electro-clinical features aid in distinguishing cases with psychopathology?**
LIM KS[1,2], SARAH W[1], SCHEFFER I[1], BERKOVIC S[1] ([1]Australia, [2]Malaysia)

Neurobiology
Friday 22nd October
15:00-16:00
Room 110
Refer to pages 91-93 for abstracts

015 **No GAD67-positive GABAergic neurogenesis in the dentate gyrus of adult mice in experimental epilepsy models**
JIANG W, YANG F, WANG Y, QUAN Q-Y, CHEN J, WU S-X (China)

016 **Astrocytic uptake of GABA critically regulates thalamic activity in generalized absence epilepsy**
BEENHAKKER M, HUGUENARD J (USA)

017 **The involvement of SVZ-derived neural precursor cells in the epileptogenicity of focal cortical dysplasia**
SHU H-F, YANG H (China)

018 **Increased stargazin and AMPA receptor expression in somatosensory cortex of genetic absence epilepsy rats from Strasbourg**
KENNARD JT, BARMANRAY R, SAMPURNO S, REID CA, PARADISO L, D'ABACO G, KAYE AH, FOOTE SJ, O'BRIEN TJ, POWELL KL (Australia)

PLATFORM SESSIONS

Paediatric epileptology
Saturday 23rd October
15:00-16:00
Room 103
Refer to pages 93-96 for abstracts

019 **A new clinical epileptic syndrome caused by SPTAN1 mutation**
TOHYAMA J, SAITSU H, AKASAKA N, OSAKA H, MIYATA R, OHASHI T, KOBAYASHI Y, KATO M, MATSUMOTO N (Japan)

020 **Re-evaluation of ketogenic diet therapy for refractory epilepsy in childhood Tokyo women's medical university's experience over 40 years**
OGUNI H, OGUNI M, ITO S, ITO Y, OSAWA M (Japan)

021 **Symptomatic focal epilepsy (SFE) sharing the clinico-electrical characteristics with Panayiotopoulos syndrome (PS)**
HIRANO Y, OGUNI H, HIRASAWA K, OSAWA M (Japan)

022 **A five-year review of childhood deaths due to SUDEP in Queensland**
CLARK D, RINEY K (Australia)

023 **Intractability of chronic non-idiopathic partial epilepsies: how many antiepileptic drugs might be tried?**
SUGAI K, NAKAGAWA E, KOMAKI H, SAITO Y, SAKUMA H, SASAKI M (Japan)

Surgery and neuroimaging
Saturday 23rd October
15:00-16:00
Room 104
Refer to pages 96-98 for abstracts

024 **Effect of anterior temporal lobectomy (ATL) on sexuality in male patients with refractory temporal lobe epilepsy-hippocampal sclerosis (TLE-HS): a case control study**
RAMESHA KN, ASHALATHA R, SEBASTIAN P, SARMA PS, UNNIKRISHNAN JP, RADHAKRISHNAN K (India)

025 **Limitations of ictal scalp EEG and SPECT in seizure localization in tuberous sclerosis with epileptic spasms**
MOHAMED AR, FREEMAN JL, BAILEY CA, MAIXNER WJ, HARVEY AS (Australia)

026 **MRI-guided stereotactic radiofrequency thermocoagulation for pediatric intractable epilepsy patients with hypothalamic hamartoma**
MURAKAMI H, KAMEYAMA S, MASUDA H (Japan)

027 **Medically refractory epilepsy associated with posterior cortex gliosis: clinical, electrophysiologic, imaging and pathologic characteristics and seizure outcome**
SINGH A, SAINI J, RATHORE C, RADHAKRISHNAN A, RADHAKRISHNAN VV, CHANDRASEKARAN K, RADHAKRISHNAN K (India)

PLATFORM SESSIONS

Social and economic issues
Saturday 23rd October
15:00-16:00
Room 106
Refer to pages 98-100 for abstracts

028 **The role of optimism, hope, and family support on quality of life in epilepsy**
PRIMARDI A, HADJAM NR (Indonesia)

029 **The lived experience of initial symptoms of epileptic seizures: an Australian study**
PINIKAHANA J (Australia)

030 **The impact of anxiety and depression on health related quality of life of young people with epilepsy**
CLARKE A, CRITCHLEY C (Australia)

031 **Economic burden of epilepsy in a developing country: a retrospective cost analysis in China**
HONG Z, WU X, YANG T, ZHANG Q, ZHOU D (China)

032 **Factors associated with quality of life in people with epilepsy and the variations between men and women, younger and older people**
YU P, HONG Z, YUE L, WU X, ZHU G (China)

General epileptology
Saturday 23rd October
15:00-16:00
Room 109
Refer to pages 100-103 for abstracts

033 **Prevalence of acute symptomatic seizures in community based study from Northern India**
GOEL D, SAXENA V (India)

034 **International variability in use of EEG when withdrawing anti-epileptic drugs (AEDs)**
BERGIN PS[1], BEGHI E[2], LEGROS B[3], RICHARDSON M[4], TRIPATHI M[5], D'SOUZA W[6], BURNEO J[7], RAYMOND AA[8], MOGAL Z[9] ([1]New Zealand, [2]Italy, [3]Belgium, [4]United Kingdom, [5]India, [6]Australia, [7]Canada, [8]Malaysia, [9]Pakistan)

035 **The Sydney Epilepsy Incidence Study to Measure Illness Consequences (SEISMIC): rationale, design and preliminary results**
ANDERSON C, BLEASEL A, GARDNER G, GHOUGASSIAN D, GLOZIER N, HACKETT M, HASSAN B, IRELAND C, JAN S, LAWSON J, MARTINIUK A, MOHAMED A, SAMPAIO H, SOMERVILLE E, TODD L (Australia)

036 **Behavioural and electrophysiological evidence of (central) auditory processing disorder in patients with refractory mesial temporal lobe epilepsy and mesial temporal lobe sclerosis**
ARAVIND KUMAR R, SHIVASHANKAR N, SATISHCHANDRA P, SINHA S, SUBBAKRISHNA DK (India)

037 **Causes of deaths among people with convulsive epilepsy in rural West China: a prospective study**
MU J, LIU L, ZHOU D (China)

PLATFORM SESSIONS

Basic science
Saturday 23rd October
15:00-16:00
Room 110
Refer to pages 103-105 for abstracts

038 **The GAERS Cav3.2 T-type calcium channel mutation affects the expression ratio of the channel splice variants**
NG C[1], POWELL KL[1], TYSON JR[2], REID CA[1], CAIN SM[2], SNUTCH TP[2], O'BRIEN TJ[1] ([1]Australia, [2]Canada)

039 **The activation of mammalian target of rapamycin signaling pathway is related to the gliosis in hippocampal sclerosis in a mouse model of mesio-temporal lobe epilepsy**
NITTA N, NOZAKI K (Japan)

040 **The modulatory effects of VEGF-C on NMDA receptor function in dysplasia cortex from an animal model of malformations of cortical development**
ZHANG C-Q, SHU H-F, ZHANG B, AN N, ZHAO B-Y, SONG Y-C, LIU S-Y, ZHOU Z, YANG H (China)

041 **The effects of strontium on outward currents in the postsynaptic membrane**
GARCIA PG, FRENCH C (Australia)

042 **The modulation of sodium conductance by phenytoin is mediated by slow rather than fast inactivation processes**
ZENG Z, FRENCH CR (Australia)

Translational research
Saturday 23rd October
17:30-18:30
Room 104
Refer to pages 106-108 for abstracts

043 **The anticonvulsive and antioxidant effects of curcumin on pilocarpine-induced seizure in rats**
DU P, TANG H, LI X, LIN H, PENG W, MA Y, FAN W, WANG X (China)

044 **Action potential dysfunction in pyramidal neurons of a Scn1b mutant mouse model of Dravet syndrome**
REID C[1], HILL E[1], THOMAS EA[1], RICHARDS K[1], WIMMER V[1], LERCHE H[2], SCHEFFER I[1], BERKOVIC S[1], PETROU S[1] ([1]Australia, [2]Germany)

045 **Pharmacogenomics of carbamazepine: from kinetics to dynamics**
CHANG W-L, LIOU H-H, HUNG C-C (Taiwan, Republic of China)

046 **The enduring effects of early-life stress on limbic epileptogenesis in rodents are mediated by seizure-induced corticosterone release**
KOE AS, JONES NC, SALZBERG MR, O'BRIEN TJ (Australia)

047 **Bilateral focal neuropeptide Y injections in the somatosensory cortex maximally suppresses absences seizures in a genetic rat model**
VAN RAAY L, JOVANOVSKA V, MORRIS MJ, O'BRIEN TJ (Australia)

PLATFORM SESSIONS

Genetics
Saturday 23rd October
17:30-18:30
Room 106
Refer to pages 108-111 for abstracts

048 **Is HLA-B*1502 associated with carbamazepine/ phenytoin-induced severe cutaneous adverse reactions in a Thai population**
TIAMKAO S (Thailand)

049 **Autosomal dominant vasovagal syncope: clinical and linkage results**
KLEIN KM, BROMHEAD C, SMITH K, O'CALLAGHAN CJ, SCHEFFER IE, MCMAHON JM, LAWRENCE KM, DIBBENS LM, BAHLO M, BERKOVIC SF (Australia)

050 **Haploinsufficiency of STXBP1 is an important cause for Ohtahara syndrome, but not for cryptogenic West syndrome**
KATO M, SAITSU H, OKADA I, ORII KE, HIGUCHI T, HOSHINO H, KUBOTA M, ARAI H, TAGAWA T, KIMURA S, SUDO A, MIYAMA S, TAKAMI Y, WATANABE T, NISHIMURA A, NISHIYAMA K, MIYAKE N, WADA T, OSAKA H, KONDO N, HAYASAKA K, MATSUMOTO N (Japan)

051 **Benign familial neonatal seizures: the mutational spectrum, clinical overlap with benign familial neonatal-infantile seizures and evidence for an additional locus**
HERON SE[1], GRINTON BE[1], PELEKANOS J[1], CARRANZA D[1], ZUBERI SM[2], LUNAN R[2], KIVITY S[3], AFAWI Z[3], SCHEFFER IE[1], BERKOVIC SF[1], MULLEY JC[1]
([1]Australia, [2]United Kingdom, [3]Israel)

052 **Genetic analysis in the patients of epilepsy with mental retardation**
NAKAGAWA E, TAKESHITA E, GOTO Y-I (Japan)

AED issues
Saturday 23rd October
17:30-18:30
Room 109
Refer to pages 111-113 for abstracts

053 **Antiepileptic drug use in pregnancy: impact on brain function of exposed Australian children**
NADEBAUM C, ANDERSON VA, VAJDA FJE, REUTENS D, BARTON S, WOOD AG (Australia)

054 **Effect of seizures on cognition, behavior, and quality of life during carbamazepine or lamotrigine monotherapy in patients with newly diagnosed or untreated partial epilepsy**
LEE S-A, KIM M-J, LEE H-W, HEO K, SHIN D-J, SONG H-K, KIM O-J, LEE S-M, KIM S-O, LEE B-I (Korea, Republic of)

055 **KONQUEST: Keppra versus older AEDS and neuropsychiatric, neurocognitive and quality of life outcomes in treatment of epilepsy as substitution**
HAKAMI T, TODARO M, ROTEN A, BRIGHT T, GERMAINE D, PETROVSKI S, MACGREGOR L, GASKO H, MATKOVIC Z, YERRA R, O'BRIEN TJ (Australia)

056 **Cognitive effects of antiepileptic drugs: a meta-analysis**
SANTOS RP (Philippines)

057 **Biodegradable polymers as drug delivery devices for the treatment of epilepsy**
SUHR C, MOULTON S, HALLIDAY A, MCLEAN K, WALLACE G, COOK M (Australia)

PLATFORM SESSIONS

Surgery
Saturday 23rd October
17:30-18:30
Room 110
Refer to pages 114-116 for abstracts

058 **Vagus nerve stimulation: outcome and predictors of seizure freedom in long-term follow-up**
GHAEMI K (Iran, Islamic Republic of)

059 **Avoidance of complications in epilepsy surgery**
MALLA BR (India)

060 **Long-term outcome of corpus callosotomy for West syndrome: second report**
BABA H, TODA K, ONO K (Japan)

061 **Surgical treatment of the patients with Rasmussen's encephalitis (20 cases)**
LUAN G, GUAN Y (China)

062 **Feasibility and safety of antiepileptic drug withdrawal following resective extratemporal surgery**
MENON RN, RATHORE C, RADHAKRISHNAN K (India)

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POSTER SESSIONS

Basic science
Refer to pages 118-129 for abstracts

p063 **Levetiracetam-releasing biodegradable polymer sheets effective at reducing seizures in the tetanus toxin model of chronic mesial temporal lobe epilepsy in rats**
HALLIDAY AJ, CAMPBELL TE, NELSON TS, MCLEAN KJ, SUHR CL, COOK MJ, WALLACE GG (Australia)

p064 **Novel oxazolidinones as potential anticonvulsant agents: in vitro testing**
KOMBIAN SB, ANANTHALAKSHMI KVV, PHILLIPS OA (Kuwait)

p065 **MicroRNA profiles involved in the hippocampal epileptogenesis**
LEE J-J, PARK K-I, CHU K, LEE S-T, JUNG K-H, LEE SK, ROH J-K (Korea, Republic of)

p066 **Reorganization of neural circuits of reuniens thalamic nucleus in the mouse brain after pilocarpine induced temporal lobe epilepsy**
MA DL, TANG FR, GOH E (Singapore)

p067 **Susceptibility of adult rats to pentylentetrazol induced epileptiform convulsions after fetal and infantile exposure to noise stress and hyperthermic-induced seizures**
MOGHIMI A, ZOBEIRI M, MORADPOOR M, GHASSEMZADEH F, BEHNAM RASOULI M (Iran, Islamic Republic of)

p068 **FFT analysis on kindling-induced afterdischarge in the rabbit hippocampus**
ANG YF, TSUCHIYA K, KOGURE S (Japan)

p069 **Frequency analysis on the afterdischarge induced by bilateral or alternate-site kindling of the rabbit hippocampi**
TSUCHIYA K, KAKUNO M, MURAYAMA N, KONDOU T, KOGURE S (Japan)

p070 **Decreased antioxidant levels and oxidative stress in patients with epilepsy**
BINDU M, RAMALINGAM K, VINOTHKUMAR R, LOKESH B (India)

p071 **Differential DNA methylation correlates with differential expression of neuronal cell adhesion molecule in human epilepsy**
WANG L, XI Z (China)

p072 **Can structural or functional changes following traumatic brain injury in the rat predict the epileptic outcome?**
CARDAMONE L[1], BOUILLERET V[1,2], LIU YR[1], GREGOIRE MC[1], HICKS RJ[1], WILLIAMS JP[1], JONES NC[1], MYERS DE[1], O'BRIEN TJ[1] ([1]Australia, [2]France)

p073 **Experimental study on the effects of EPO, C-EPO on the protein expression of Jak2/ STAT5 and PI3K/akt in hippocampus of epilepsy rats induced by kainic acid**
WANG Z, LIN WH, JIANG HY, WU SS (China)

p074 **The role of GABA neurons in the pathogenesis of epileptic FMR1 KO mice**
ZENG Y, XU M, YU M, SHI Y, LONG Y, YI Y, LIAO W (China)

POSTER SESSIONS

p075 **Linking the brain and heart: alterations in cardiac function and HCN channel expression in genetically epileptic rats**
NG C[1], URMALIYA V[1], POWELL KL[1], KENNARD JT[1], JONES NC[1], MEGATIA I[1], REID CA[1], PINAULT D[2], WHITE P[1], O'BRIEN TJ[1] ([1]Australia, [2]France)

p076 **A Cav3.2 T-type calcium channel mutation (R1584P) associates with altered neuronal firing pattern in the thalamic reticular nucleus (TRN) of genetic absence epilepsy rats from Strasbourg**
VAN RAAY L[1], ABDULLAH ANN[1], JONES NC[1], ÇARÇAK2 N[2], ONAT F[2], FRENCH C[1], PINAULT D[3], O'BRIEN TJ[1], POWELL KL[1] ([1]Australia, [2]Turkey, [3]France)

p077 **Ethosuximide treatment restricts epileptogenesis and alleviates behavioural co-morbidities in the GAERS model of absence epilepsy**
DEZSI G[1], BLUMENFELD H[2], SALZBERG MR[1], O'BRIEN TJ[1], JONES NC[1] ([1]Australia, [2]USA)

p078 **Supression of absence-like seizures in the rat by neuropeptide Y is associated with increased neuronal firing in the reticular thalamus**
GANDRATHI AK, O' BRIEN T, FRENCH C, MORRIS M (Australia)

p079 **Viral therapy for idiopathic generalised epilepsy: neuropeptide Y overexpression in ventrolateral thalamus suppresses absence-like seizures in a genetic rat model**
JOVANOVSKA V[1], KRISHNASAMY N[1], VAN RAAY L[1], BLAND R[2], DURING MJ[2], O'BRIEN TJ[1], MORRIS MJ[1] ([1]Australia, [2]USA)

p080 **Seizure control with very high frequency electrical stimulation in the GAERS model of epilepsy**
NELSON T, SUHR C, COOK M (Australia)

p081 **Environmental enrichment leads to delayed onset of limbic epilepsy, improved neuropsychiatric behavioural profile and neuroanatomical alterations**
YANG M, SALZBERG MR, REES SM, O'BRIEN TJ, JONES NC (Australia)

p082 **Experimental study of lamotrigine intervention for epileptogenesis in the model of post-traumatic epilepsy**
SUN MZ[1], GUO ZZ[1], WANG M[1], VAN LUIJTELAAR ELJM[2] ([1]China, [2]Netherlands)

Neurobiology
Refer to pages 129-135 for abstracts

p083 **Inhibition mechanism of focal epileptic seizure by thumb pressing method**
KUSUMASTUTI K (Indonesia)

p084 **Cell recognition molecules may trigger the epileptogenesis during development in the hippocampus of the epileptic mutant EL**
MURASHIMA YL, YOSHII M, INOUE N (Japan)

p085 **Initiation and propagation of epileptiform activity in hippocampal slice based on multilectrode array recording**
LIU JS, GONG XW, LI JB, ZHANG PM, LIANG PJ, LU QC (China)

POSTER SESSIONS

p086 **Low blood glucose increases absence seizure susceptibility**
KIM TH, REID CA, BERKOVIC SF, PETROU S (Australia)

p087 **Gender effect on the amygdala volume in temporal lobe epilepsy**
PINTO ML, SILVA I, LIN K, JACKOWSKI AP, CENTENO RS, JUNIOR HC, YACUBIAN EM, AMADO D (Brazil)

p088 **Enduring effects of early life stress on neuronal firing patterns in thalamic reticular nucleus: implications for limbic epilepsy**
ALI I[1], SALZBERG M[1], JONES NC[1], O'BRIEN P[1], FRENCH C[1], PINAULT D[2], MORRIS MJ[1], O'BRIEN TJ[1] ([1]Australia, [2]France)

p089 **The impact of tissue plasminogen activator (tPA) expression on limbic epileptogenesis in mice**
NG AQ, SASHINDRANATH M, MEDCALF R, COOK A, HAMILTON J, O'BRIEN TJ, JONES NC (Australia)

p090 **Rats with epilepsy induced by pilocarpine present a pineal gland lesion and reduced nocturnal melatonin levels**
LIMA E, PERES R, BARATELI V, ROSA R, AFECHE S, CAVALHEIRO E, NAFFAH-MAZZACORATTI MDG, AMADO D, CIPOLLA-NETO J (Brazil)

p091 **MEMRI evaluation of coriaria lactone-induced neuronal activation in rhesus hippocampus**
YANG T, HONG Z, YAN B, LI H, GONG Q, ZHOU D (China)

p092 **Precursor rhythmic neuronal activities in S2 somatosensory and insular cortices during the initiation of genetically determined absence-related spike-and-wave discharges**
ZHENG TW[1,2], MORRIS MJ[1], JOVANOVSKA V[1], VAN RAAY L[1], GANDRATHI AK[1], REID CA[1], O'BRIEN TJ[1], PINAULT D[2] ([1]Australia, [2]France)

p093 **Further evidence of GABRG2 variants in severe myoclonic epilepsy of infancy**
CHUNG S-K (United Kingdom)

Genetics

Refer to pages 135-146 for abstracts

p094 **Molecular diagnosis of generalized epilepsy with febrile seizure plus (gefs+): novel mutations of SCN1A gene detected in malaysian patients**
TAN EH, RAZAK SA, ABDULLAH JM, YUSOFF AAM (Malaysia)

p095 **The GAT3 gene is associated with epileptogenesis**
KIM YO, KIM MK, JANG SY (Korea, Republic of)

p096 **Association of variants in CYP2C19 and ABCB1 with phenytoin maintenance doses in Thai patients with epilepsy**
INSARO M, CHINVARUN Y, YODNOPKLAO P, NILAYON D, TANTISIRA M, KIJSANAYOTIN P (Thailand)

p097 **Base substitution spectrum of SCN1A point mutation in epilepsy**
HE N, GUO J, LIAO W-P (China)

POSTER SESSIONS

p098 **Genetic localization of familial cortical tremor with epilepsy gene to chromosome 8q23.3-24.22**
LI N, JIANG H, SHEN L, LI X, HUANG X, WANG J, WU Y, CHEN C, GUO J, HU Z, XIA K, ZI X, TANG B (China)

p099 **Novel mutation of SCN1A in familial generalized epilepsy with febrile seizures plus**
TANG B, LI N, ZHANG J, GUO J, YAN X, XIA K (China)

p100 **Impact of genetic polymorphisms on pharmacokinetic and pharmacodynamic pathways of valproic acid**
HO J-L, LIOU H-H, HUNG C-C (Taiwan, Republic of China)

p101 **Having an answer: does knowing your epilepsy gene really help families?**
VEARS DF, DUNN K, WAKE SA, SCHEFFER IE (Australia)

p102 **Coexistence gene mutation in SMEI are identified by using DNA array**
SUGAWARA T, YOSHIDA S, WADA K, HIROSE S, KANEKO S (Japan)

p103 **Severe infantile multi-focal epilepsy: a focal epileptic encephalopathy due to SCN1A mutations**
BELLOWS ST[1], ARSOV T[1], MCMAHON JM[1], GILL D[1], DAMIANO J[1], ZUBERI SM[2], MULLEY JC[1], BERKOVIC SF[1], SCHEFFER IE[1] ([1]Australia, [2]United Kingdom)

p104 **Acetylcholine receptor gene alpha 7 (CHRNA7) in idiopathic generalised epilepsy (IGE): mutation detection**
DAMIANO JA, BERKOVIC SF, LAWRENCE KM, MULLEN SA, ARSOV T (Australia)

p105 **Epilepsy-migraine syndrome due to a missense mutation in SCN1A**
OLIVER K[1], ARSOV T[1], KIVITY S[2], AFAWI Z[2], DAMIANO J[1], KORCZYN A[2], BERKOVIC S[1] ([1]Australia, [2]Israel)

p106 **Familial adult myoclonic epilepsy (FAME): recognition of mild phenotypes and refinement of the 2q locus**
CROMPTON DE[1,2], LAWRENCE KM[1], BELLOWS ST[1], SADLEIR LG[3], BAHLO M[1], HARTY R[3], DUNNE JW[1], ARSOV T[1], BERKOVIC SF[1], SCHEFFER IE[1] ([1]Australia, [2]United Kingdom, [3]New Zealand)

p107 **The contribution of SCARB2 and PRICKLE1 gene mutations in progressive myoclonus epilepsies**
DIBBENS LM[1], BAYLY MA[1], ANDERMANN E[2], VEARS DF[1], MACPHERSON J[3], ANDERMANN F[2], COLE AJ[2], LEACH JP[3], GORRIE G[3], MICHELI F[4], LEHESJOKI A-E[5], BERKOVIC SF[1] ([1]Australia, [2]Canada, [3]United Kingdom, [4]Argentina, [5]Finland)

p108 **Mutation of channel SCN3A in a patient with severe myoclonic epilepsy of infancy and autism**
LI B-M, WANG P, SHI Y-W, YU M-J, DENG W-Y, GAO M-M, LIU X-R, DENG Y-H, YI Y-H, LIAO W-P (China)

p109 **PCDH19 mutations cause epilepsy and mental retardation in females (EFMR)**
DESAI T[1], DIBBENS LM[1], BAYLY MA[1], ARSOV T[1], KNEEN R[2], GIBBS J[2], SISODIYA S[2], GECZ J[1], MCKENZIE F[1], BERKOVIC SF[1], MULLEY JC[1], VON SPICZAK S[3], RONAN A[1], SCHEFFER IE[1] ([1]Australia, [2]United Kingdom, [3]Germany)

POSTER SESSIONS

p110 **Epilepsy with occipital features due to SCN1A missense mutations**
ARSOV T[1], KIVITY S[2], DAMIANO J[1], AFAWI Z[2], OLIVER K[1], MCMAHON J[1], LAWRENCE K[1], SCHEFFER IE[1], BERKOVIC SF[1] ([1]Australia, [2]Israel)

p111 **Frequencies of the polymorphisms in ABCB1 and association of these polymorphisms with multidrug-resistant epilepsy**
HONG Z, YUE L, ZHU G, YU P, WU X (China)

p112 **Skewed X-inactivation may mask inheritance of a CDKL5 mutation**
YENDLE SC[1], HARVEY AS[1], BIENVENU T[2], SCHEFFER IE[1] ([1]Australia, [2]France)

Translational research
Refer to pages 146-147 for abstracts

p113 **Coupling of cerebral blood flow and metabolism during focal hypothermia**
FUJIOKA H, FUJII M, KOIZUMI H, NOMURA S, IMOTO H, SUEHIRO E, OWADA Y, SAITO T, YAMAKAWA T, SUZUKI M (Japan)

p114 **A prospective study of the use of HLA-B*1502 genotyping in preventing carbamazepine-induced Stevens-Johnson syndrome and toxic epidermal necrolysis**
CHEN P, SHEN C-Y, LIN J-J, ONG C-T, WU S-L, TSAI P-J, CHEN W-H, CHUNG W-H, HUNG S-I, CHANG C-F, CHEN C-H, LIN S-Y, CHUAN H-P, HUANG L-C, CHEN Y-J, CHEN Y-T (Taiwan, Republic of China)

Women and pregnancy
Refer to pages 147-150 for abstracts

p115 **Risk factor related with reproductive endocrinology disorder in Chinese women of child-bearing age with Epilepsy Department of Neurology**
ZHOU J-Q, CHEN L-J, ZHOU L-M, CHEN Z-Y (China)

p116 **The effect of fetal valproate exposure on memory functioning in school aged children**
BARTON S, NADEBAUM C, ANDERSON VA, VAJDA FJE, REUTENS D, WOOD AG (Australia)

p117 **Report on the second generation of antiepileptic drugs (AEDs) from the database of the Australian pregnancy register (APR)**
VAJDA F, GRAHAM J, ROTEN A, LANDER C, O'BRIEN T, EADIE M (Australia)

p118 **Status epilepticus and posterior reversible encephalopathy syndrome (PRES)**
NARAYANA RV, PATI R, KUMAR S, MAHATO A (India)

p119 **Assessment of knowledge of women's issues and epilepsy: a survey of obstetrician in Kerala, South India**
BHAT M, NIRMALA C, THOMAS SV (India)

p120 **Early cognitive and behavioural outcomes following prenatal exposure to antiepileptic drugs**
HARTMANN P, ANDERSON V, VAJDA F, REUTENS D, HOLDEN M, WOOD A (Australia)

POSTER SESSIONS

Status epilepticus

Refer to pages 151-153 for abstracts

p121 **A case of status epilepticus in a patient with ginkgo nut intoxication**
PARK H-K, YANG K-I, OH H-G (Korea, Republic of)

p122 **Neurodegenerations as etiologic factors of status epilepticus at infancy and early childhood**
KHOLIN A, MIKHAILOVA S, IL'INA E, LEMESHKO I, VORONKOVA K, MUKHIN K, PETRUKHIN A (Russian Federation)

p123 **A retrospective analysis of children admitted with convulsive status epilepticus to Children's Hospital at Westmead**
GUPTA S, GILL DA, DALE RC (Australia)

p124 **In search of neuroprotective drugs against experimentally induced status epilepticus in young rats**
AHMAD M, AL DEEB S, TARIQ M (Saudi Arabia)

p125 **A case of lingual epilepsia partialis continua persisted through two and a half years**
CHO Y-J, LEE MK, LEE BI, HEO K (Korea, Republic of)

Adult epileptology

Refer to pages 153-158 for abstracts

p126 **Convulsive syncope: diagnostic accuracy and prognosis**
CHAN J, DUNNE JW, LEE JC, LAWN ND (Australia)

p127 **Risk factors for the development of seizures and epilepsy among post stroke patients in a Philippine tertiary hospital: a retrospective cohort study**
FABIANA NL (Philippines)

p128 **Symptomatic seizures following acute Japanese and herpes simplex encephalitis: experience from a tertiary care centre in South India**
SINHA S, RAO S, RAVI V, NAGARATHNA S (India)

p129 **Quality of life for Mongolian patients with epilepsy**
BAYARMAA D, ARIUNAA J (Mongolia)

p130 **Intracranial EEG ictal onset: which frequency, high or low?**
CHUNG JM, TSUCHIYA N, TUDUSCIUC O, MAMALAK A, ELIASHIV D (USA)

p131 **Early ictal SPECT injection in the localisation of the epileptogenic zone**
WONG TH, BIRKETT J, MCANALLY L, BARTLEY M, THEA KHUN T, LARCOS G, BLEASEL A (Australia)

p132 **Presence of human herpes virus-6 DNA exclusively in temporal lobe epilepsy brain tissue of patients with history of encephalitis**
NIEHUSMANN P, MITTELSTAEDT T, BIEN CG, DREXLER JF, GROTE A, SCHOCH S, BECKER AJ (Germany)

p133 **Electrical status of slow wave sleep in adults**
MCCONNELL H (Australia)

POSTER SESSIONS

p134 **Cerebral venous thrombosis as a cause of new-onset seizures in young adults**
KIM J (Korea, Republic of)

p135 **Surveillance of epilepsy in catchment area of Dhulikhel Hospital**
PANT P, GAUTAM N, UPRETY S, KOJU R, GURUNG R, POKHAREL B, BEDI TRS (Nepal)

Epidemiology
Refer to pages 158-163 for abstracts

p136 **Seizure-related injuries in newly diagnosed childhood epilepsy**
TING D, KWONG LK (Hong Kong)

p137 **Epidemiology of epilepsy in children less than five years of age in a developing nation**
SUJATHA SG, THORA S, DUBEY R (India)

p138 **A study of newly diagnosed epileptic patients in Karachi**
HEREKAR AD (Pakistan)

p139 **Prevalence of post-stroke seizures in Srinagarind hospital**
TIAMKAO S (Thailand)

p140 **Sociodemographic and clinical characteristics of patients with epilepsy from Eastern Nepal**
ROKA YB, BISTA BK, AHMAD SW, PARAJULI M (Nepal)

p141 **Traditional health practitioners in Jhenaidah district of Bangladesh: their knowledge on epilepsy, malaria, insomnia, and sexually transmitted diseases and possible role in primary health care**
MOLLIK MAH (Bangladesh)

p142 **Incidence of epilepsy in US nursing homes by predisposing conditions**
LEPPIK IE, EBERLY L, BIRNBAUM A, HARMS S, SVENDSEN K, GARRARD J, VIRNIG B (USA)

p143 **Prevalence of epilepsy in Korea: nationwide study from the national health insurance statistics**
LEE S-Y, JUNG K-Y, KIM DW (Korea, Republic of)

p144 **The prevalence of seizure-related injury, near drowning and vehicular crashes in a community sample of patients with epilepsy**
TAN M[1], FICKER D[2], O'BRIEN TJ[1], COOK MJ[1], PEARCE N[3], D'SOUZA WJ[1,3]
([1]Australia, [2]USA, [3]New Zealand)

p145 **Prescription patterns of antiepileptic drugs in Taiwan: a nationwide population-based study in the years 2003-2007**
HSIEH L-P, HUANG C-Y (Taiwan, Republic of China)

POSTER SESSIONS

Prognosis
Refer to pages 163-165 for abstracts

p146 **Predictors of outcome in the resective surgical treatment of epilepsy**
YANG X, LU Q (China)

p147 **Does early electroencephalography (EEG) finding of clinical neonatal seizures have a predictive value to long-term outcome?**
SANG T, JIANG Y, YANG Y, LIU X, QIN J (China)

p148 **A study of predictive factors for post stroke seizures**
DHAMIJA RK, KAINTURA A, KUMAR M (India)

Paediatric epileptology
Refer to pages 165-185 for abstracts

p149 **Adolescent methamphetamine administration impairs spatial memory in rats with neonatal status epilepticus**
HUANG L-T, WANG J-Y (Taiwan, Republic of China)

p150 **Mozart K.448 acts as an add-on therapy in children with electrophysiologically or clinically refractory epilepsy**
LIN L-C, YANG R-C (Taiwan, Republic of China)

p151 **ACTH therapy for two cases of epileptic spasms without hypsarrhythmia**
SHIMONO M, ISHII M, SENJU A, SHIOTA N, KUSUHARA K (Japan)

p152 **A clinical case study of hypomelanosis of Ito and epilepsy**
ZHANG J (China)

p153 **Utility of prolonged video EEG recording in assessment of refractory epilepsies**
FUNG L-WE, CHONG J, TSUNG L-YL (Hong Kong)

p154 **Oxcarbazepine oral suspension in epilepsy: our experience in Taiwanese infants and children, and a review of the literature**
WEI S-H, FAN P-C (Taiwan, Republic of China)

p155 **Study about prevalence of idiopathic generalized epilepsies in children**
KUMAR A, PRAKASH S, KUMAR A, KUMAR R (India)

p156 **Neuropsychological change over time in children with benign rolandic epilepsy**
KWON S, SEO HE, BYUN JC (Korea, Republic of)

p157 **Malignant epilepsies starting in the neonatal period and suppression-burst EEG pattern**
YAMAMOTO H, OKUMURA A (Japan)

p158 **Quest for a pragmatic, patient oriented approach: modified ILAE classification in a pediatric epilepsy clinic**
SRIVASTAVA K, RAJADHYAKSHA S (India)

POSTER SESSIONS

p159 **The expression of TRPV1 in cortical lesions of tuberous sclerosis complex and focal cortical dysplasias type IIb**
SHU H-F, ZHANG C-Q, YU S-X, SONG Y-C, LIU S-Y, YANG H, YIN Q (China)

p160 **Clinical characteristics of patients with West syndrome associated with occipital focal cortical dysplasia**
AKASAKA N, TOHYAMA J, OHASHI T, KOBAYASHI Y, SASAGAWA M, MURAKAMI H, MASUDA H, KAMEYAMA S (Japan)

p161 **Electroclinical features of late-onset spasms: a report of 9 cases**
HWANG YS, HWANG H, LIM BC, KIM KJ, CHAE JH (Korea, Republic of)

p162 **Clinical experience with levetiracetam monotherapy in pediatric epilepsy in Chungbuk, Korea**
KIM WS, SIM JY, PARK HJ (Korea, Republic of)

p163 **Rapid onset obesity, hypothalamic dysfunction, hypoventilation, and autonomic dysregulation - a clinical syndrome with autonomic crisis masquerading as epilepsy and encephalopathy: a report of two cases**
TAJUL ARIFIN T, KHOO TB, KHOO PC, HUSSAIN IHMI, DAYANG SR, ASIAH K, NORZILA MZ (Malaysia)

p164 **Impact of pediatric epilepsy surgery on quality of life: a case controlled study from Northern India**
SHUKLA G, MOHAMMED A, GOYAL V, SRIVASTAVA A, GUPTA A, SURI A, CHANDRA PS, GARG A, GAIKWAD S, BAL CS, BEHARI M (India)

p165 **Remission of symptomatic epilepsy in post-liver transplant children in Singapore**
LAINO AGG, ONG HT, AW M, QUAK SH (Singapore)

p166 **Infantile spasm control and developmental progression following short course therapy with steroids/vigabatrin**
WANIGASINGHE J, JAYASENA YAA, DILANKA S (Sri Lanka)

p167 **A child who falls with a tap on the head**
GOVINDARAJAN R (USA)

p168 **Devastating epileptic encephalopathy following generalized febrile status epilepticus**
JADHAV VM (India)

p169 **Comparison of melatonin and sleep-deprivation for induction of sleep during paediatric sleep EEG acquisition**
SINGH H, BRASS J, RINEY K (Australia)

p170 **Febrile infection-related epilepsy syndrome (FIRES) does not respond to rituximab or vagus nerve stimulation: a case report**
HOWELL KB, HARVEY AS, BAILEY CA, MAIXNER W, FREEMAN JL (Australia)

p171 **Developmental GABAA receptor-mediated depolarization/excitation may contribute to immediate and delayed adverse effects of neonatal anesthesia**
MARTYNYUK A, CAO W, SHAH H, EDWARDS D, GRAVENSTEIN N, SEUBERT C (USA)

POSTER SESSIONS

p172 **Vigabatrin-related neurotoxicity in an infant: first reported human case detailing clinical, histopathological and radiological findings**
MALONE SA, KAMPPI H, WALSH M, WALLACE G, DEXTER M, LAWSON J, SUGO E, BYE A (Australia)

p173 **Assessment of the visual field defect associated with vigabatrin therapy in epileptic children**
CHUNG S (Korea, Republic of)

p174 **Eight cases of malignant epilepsies with intractable epileptic seizures and mental retardation**
FUKUDA M, YAMAMOTO H (Japan)

p175 **Prevalence of genetic and chromosomal syndromes and congenital brain malformations among epilepsy patients**
TYSYACHINA MD, KUZINA NY, MUKHIN KY, PETRUKHIN AS, MELIKYAN EG (Russian Federation)

p176 **Vascular endothelial growth factor C (VEGF-C) system expression in cortical tubers of the tuberous sclerosis complex**
ZHANG C-Q, SHU H-F, YIN Q, AN N, ZHAO B-Y, SONG Y-C, LIU S-Y, YANG H (China)

p177 **Clinical outcome and long term prognosis of childhood epilepsy**
YUNG A, WONG V (Hong Kong)

p178 **Predictors of intractable epilepsy in childhood**
MARTHATI MB (India)

p179 **Epileptic encephalopathies related to mutations of the SCN1A gene: a single center experience**
PILLAI S, DOUGLAS L, GILL D (Australia)

p180 **Study of the health-related quality of life in children with epilepsy**
MELIKYAN EG, MUKHIN KY (Russian Federation)

p181 **Seizure and cognitive outcome in Thai children with benign Roland epilepsy**
VISUDTIBHAN A, SAKPICHAISAKUN K, KHONGKHATITHUM C, THAMPRATANKUL L, CHIEMCHANYA S, VISUDHIPHAN P (Thailand)

p182 **Characteristics of centrotemporal spikes in Panayiotopoulos syndrome**
TOMONOH Y, FUJITA T, IHARA Y, NAKAMURA N, NIMOMIYA S, KODAMA R, IDEGUCHI H, INOUE T, YASUMOTO S, HIROSE S (Japan)

p183 **Steroid pulse therapy for a case of frontal lobe epilepsy with antibodies to glutamate receptor (GluR)**
IHARA Y, TOMONOU Y, FUJITA T, IDEGUCHI H, INOUE T, YASUMOTO S, TAKAHASHI Y, HIROSE S (Japan)

p184 **Drug withdrawal during inpatient evaluation for pediatric epilepsy surgery: outcomes in a newly established service**
SINGH H, CLARK D, GURR S, RICHMOND J, MALONE SA (Australia)

POSTER SESSIONS

p185 **Clinical profile of 13 children with severe myoclonic epilepsy of infancy in Hong Kong**
FUNG C-W, WONG V (Hong Kong)

p186 **Sub-acute sclerotic panencephalitis (SSPE) is still prevalent in under-developed countries like Pakistan**
HEREKAR AD (Pakistan)

p187 **Early identification and evaluation of neonatal seizures including neurodevelopmental impairments in the hospital admitted newborns**
BANU SH, AKHTER R, KHAN NZ (Bangladesh)

p188 **Effects of topiramate on the problem solving abilities and language in newly diagnosed pediatric epileptic patients**
KIM SJ, CHOI YM (Korea, Republic of)

AED issues
Refer to pages 186-194 for abstracts

p189 **A comparison of buccal midazolam and intravenous diazepam for the acute treatment of seizures in children and adolescents**
MAHVELATI SHAMSABADI F, TONEKABONI SH, ANVARI SS
(Iran, Islamic Republic of)

p190 **Pharmacokinetics of carbamazepine in Mongolia for people with partial epilepsy**
UURIINTUYA M, ULZIIBAYAR D, BATHUYAG P, NARAN G, BAYARMAA D (Mongolia)

p191 **What happens to seizures in patients with MIE when the number of AEDs is reduced?**
TRIPATHI M, AGRAWAL V, GOEL M, VASANTHA P, BHATIA R, SINGH M,
PRASAD K (India)

p192 **In vitro transport profile of carbamazepine and its analogs by human P-glycoprotein**
ZHANG C, ZUO Z, BAUM L, KWAN P (Hong Kong)

p193 **Efficacy and safety of carbamazepine, phenobarbital, phenytoin, valproic acid, or topiramate as initial monotherapy in patients with newly diagnosed epilepsy: a long-term, head-to-head comparison in Southwest China**
HU Y, LV Y, YU W, WANG X (China)

p194 **Idiopathic generalized epilepsy and choice of antiepileptic drugs**
AL HAMMADI F, TAHA H, AL NEAIMI O, AL SAADI T (United Arab Emirates)

p195 **Do short-term clinical trials predict long-term response? The SKATE Australia follow-up study**
SOMERVILLE ER (Australia)

p196 **Side effects and tolerability of IV levetiracetam vs. IV phenytoin and follow-on oral regimens in a neurosurgical patient population: a prospective randomised study**
FULLER KL, WANG YY, COOK MJ, MURPHY MA, D'SOUZA WJ (Australia)

POSTER SESSIONS

p197 **Lacosamide as adjunctive therapy with a broad range of AEDs: a pooled analysis of lacosamide clinical trial data by concomitant AED mechanism of action**
DE BACKER M[1], ISOJARVI J[2], HEBERT D[2], DOTY P[2], ZACKHEIM J[2], DAVIES K[2], EGGERT-FORMELLA A[2], SAKE J-K[1] ([1]Belgium, [2]USA)

p198 **Screening for risk of osteoporosis in patients with refractory epilepsy attending epilepsy camp by the use of portable BMD machine study of 61 patients**
NADKARNI V (India)

p199 **Response to lamotrigine is associated with genetic variants of ABCB1 and SCN1A**
KIM SE, HWANG TG, SHIN JG (Korea, Republic of)

p200 **The effect of topiramate monotherapy on bone mineral density and markers of bone and mineral metabolism in premenopausal women with epilepsy**
HEO K, RHEE Y, LEE BI, LEE HW, LEE SA, SHIN DJ, KIM W-J, SONG H-K, SONG K (Korea, Republic of)

p201 **Lack of association of lamotrigine-induced cutaneous adverse reactions with HLA-B*1502 in a Han Chinese population**
AN D[1], WU X[1], HU F[1], YAN B[1], STEFAN H[2], ZHOU D[1] ([1]China, [2]Germany)

p202 **Prospective HLA-B*1502 genotyping in preventing carbamazepine-induced Stevens-Johnson syndrome: one medical center experience**
HUNG K-L, LIAO H-T, CHIANG T-R, CHEN Y-T (Taiwan, Republic of China)

p203 **The effect of levetiracetam monotherapy on subjective sleep quality and objective sleep parameters in patients with epilepsy: compared with the effect of carbamazepine-CR monotherapy**
CHO YW[1], YI SD[1], KIM DH[1], MOTAMEDI GK[2] ([1]Korea, Republic of, [2]USA)

p204 **UGT2B7 polymorphisms on carbamazeine pharmacokinetics in epileptic patients developing adverse drug reactions and toxicity**
VIVEKANANDHAN S, MURALI M, TRIPATI M, BEHARI M, JAIN DC (India)

Clinical neurophysiology
Refer to pages 194-200 for abstracts

p205 **Cortical hyper-excitability in migraine and epilepsy: is this the link?**
BADAWY RAB, JACKSON GD (Australia)

p206 **Suppressive effect of low-frequency electric cortical stimulation on the seizure onset zone relative to the cortico-cortical connectivity**
KINOSHITA M, MATSUMOTO R, MATSUHASHI M, MITSUEDA-ONO T, HITOMI T, TAKI J, MIKUNI N, FUKUYAMA H, TAKAHASHI R, IKEDA A (Japan)

p207 **Effects of chronic epilepsy on heart rate variability upon the seizure frequency**
HARNOD T (Taiwan, Republic of China)

p208 **Surgery of focal and multifocal temporal epilepsy and neurophysiology of early and late epileptogenesis**
BERSNEV V, STEPANOVA T, KASUMOV V, KASUMOV R, KRAVTSOVA S, SEBELEV K (Russian Federation)

POSTER SESSIONS

p209 **The effect of hyperventilation on enhancing epileptic discharge using a simultaneous measurement by electroencephalography and near infra-red spectroscopy**
NISHIBAYASHI H, ITAKURA T (Japan)

p210 **Localization and extent of the intracranial irritative zone in patients who have no detectable interictal epileptiform activity on scalp EEG: a case-control study**
UNNWONGSE K[1,2], WEHNER T[3], ALEXOPOULOS A[1] ([1]USA, [2]Thailand, [3]Germany)

p211 **Gamma EEG in epilepsy and other CNS disorders**
WHITHAM E, POPE KJ, LEWIS TW, FITZGIBBON SP, WILLOUGHBY JO (Australia)

p212 **Frequency analysis of ictal neuromagnetic signals in presurgical evaluation of patients with intractable epilepsy**
IIDA K, HASHIZUME A, KAGAWA K, NISHIMOTO T, KIURA Y, HANAYA R, ARITA K, KURISU K (Japan)

p213 **The signal detection ability of the scalp dense array electroencephalogram: spike comparison with the simultaneous subdural electrodes**
FUJIMOTO A, YAMAZOE T, ENOKI H, OKANISHI T, YOKOTA T, TOYODA G, YAMAZAKI M, YAMAMOTO T (Japan)

p214 **Clinical, neuroimaging and electrophysiological characteristics of patients with refractory eating epilepsy (EE)**
CHERIAN A, BAHETI NN, MENON RN, RADHAKRISHNAN A, RADHAKRISHNAN K (India)

p215 **Changes of cortical excitability after epileptic seizure and non-epileptic seizure**
SHIN WC, CHO YW, SUNG MJ, RHEE HY (Korea, Republic of)

EEG

Refer to pages 200-211 for abstracts

p216 **Simultaneous recording of electroencephalography and electromyography enhances the diagnosis of paroxysmal events during video-electroencephalography monitoring**
HILL A, BRIGGS B, SENEVIRATNE U (Australia)

p217 **EEG characteristics and surgical treatment of epileptic spasms**
ZHOU W (China)

p218 **BESA epilepsy software: fast clinical evaluation of interictal spikes in long-term EEG**
SCHERG M[1], ILLE N[1], WECKESSER D[1], EBERT A[1], OSTENDORF A[1], LARSSON PG[2], HENNING O[2], BOPPEL T[1], BAST T[1] ([1]Germany, [2]Norway)

p219 **The utility of outpatient short-term video-EEG monitoring in the diagnosis of paroxysmal disorders**
SENEVIRATNE U, RAHMAN Z, DIAMOND A, BRUSCO M (Australia)

p220 **Comparative study of ictal SPECT and video EEG in localization of epileptogenic foci for intractable epilepsy**
YANG M-H, HUANG Y, YANG H-A, LIU S-Y, YANG H, AN N, HUANG T, LIU L-H, SHI X-J, CAI F-C, ZHANG Q (China)

POSTER SESSIONS

p221 **An imPRESsive EEG: novel EEG features of posterior reversible encephalopathy syndrome**
BOISSÉ L, BOYD JG, BRUNET DG (Canada)

p222 **Automatic abnormal waves detection from the electroencephalograms of epilepsy cases to sort out the spikes, the sharps, the polyphase based on their zerocrossing and wavelet transformation**
NOERTJAHJANI S (Indonesia)

p223 **Electrocorticography (ECoG) patterns of mesial temporal lobe epilepsy and neocortical temporal lobe epilepsy**
DONGMING W (China)

p224 **Bemegride-induced test in EEG recording for preoperative localization of temporal lobe epilepsy**
LI A, LIANG S, LIANG S, SUN Y (China)

p225 **Electroencephalographic findings among patients with complex febrile seizure: a 5-year review**
ADAJAR JA (Philippines)

p226 **Use of EEG in cerebrovascular disorder (Moya-Moya disease)**
COLEBATCH JG, MALHOTRA RC (Australia)

p227 **EEG findings in children with anti-N-methyl-D-aspartate receptor (NMDAR) encephalitis**
PILLAI SC, DALE RC, GILL D (Australia)

p228 **Electroencephalographic patterns in intensive care unit patients**
CINAR N, SAHIN S, OKLUOGLU T, TELLIOGLU N, KARSIDAG S (Turkey)

p229 **Serial electroencephalographic findings in patients diagnosed with subacute sclerosing panencephalitis given intraventricular ribavirin**
VICTA EAT, SANCHEZ-GAN BC, LUKBAN MB (Philippines)

p230 **Minimally-invasive ECoG recording using the subdural microelectrode array guided by the shape memory alloy wire**
YAMAKAWA T, YAMAKAWA T, AOU S, ISHIZUKA S, FUJII M, SUZUKI M, AOKI T (Japan)

p231 **Developing electro-encephalopgram (EEG) service in the management of childhood epilepsy in Bangladesh: an overview**
BANU SH, HOSSAIN M, ISLAM F, RANI S, KHAN NZ (Bangladesh)

p232 **Measuring cortical excitability using iEEG responses to an electrical probing stimulus**
FREESTONE DR, GRAYDEN DB, NELSON TS, LAI A, BURKITT AN, VOGRIN S, KUHLMANN L, COOK MJ (Australia)

p233 **Pre-tapering of antiepileptic drugs before video EEG monitoring to reduce the monitoring time**
KHARBANDA P, PRABHAKAR S, KAUR S (India)

POSTER SESSIONS

p234 **A simple change to photic stimulation markedly improves EEG diagnosis of first seizures**
MULLEN S, BERKOVIC S (Australia)

p235 **Correlation between contrast-enhanced cranial MRI and long-term ictal EEG in determining focus for surgery in patients with temporal lobe epilepsy**
BINTORO AC, KUSTIOWATI E (Indonesia)

p236 **EEG features of idiopathic and symptomatic epilepsies**
BAYASGALAN B, SUVD R (Mongolia)

Seizure semiology

Refer to pages 211-212 for abstracts

p237 **A gut feeling about insula seizures**
DIONISIO S, SOMERVILLE ER, KOENIG A (Australia)

p238 **To determine localization value of hypersalivation**
GUL G, KORKUT E, KUŞCU DY, BEHREM N, KİRBAŞ D (Turkey)

p239 **The effects of morphine and naloxon on spontaneuse seizure activity in hippocampal slices**
ALIJARAHI A, ESMAEILI MH, AMINI M (Iran, Islamic Republic of)

Psychiatry

Refer to pages 212-213 for abstracts

p240 **Do disturbances in hypothalamic-pituitary-adrenal axis and autonomic function play a role in the comorbidity of depression in patients with epilepsy?**
SOH LJH, TURNBULL C, TAN M, LOU N, JACKSON B, ADAMS S, VELAKOULIS D, O'BRIEN T, SALZBERG M (Australia)

p241 **Co-morbidity of autistic spectrum disorder with epilepsy in Chinese children**
HAN Y, QIN J, CHEN J, JI X, JIANG Y, LIN Q (China)

Neuropsychology

Refer to pages 214-217 for abstracts

p242 **The relationship between fatigue and C-reactive protein in epilepsy patients**
KIM K (Korea, Republic of)

p243 **The cognitive effects of age at seizure onset in childhood symptomatic focal epilepsy**
EMBULDENIYA U, GONZALEZ LM, WOOD AG, HARVEY S, WRENNALL J, TESTA R (Australia)

p244 **Evaluation of the material-specific amnesia model in mesial temporal lobe epilepsy**
GIUMMARRA J, BOWDEN SC, MURPHY M, COOK M (Australia)

p245 **Cognitive function in the newly diagnosed juvenile myoclonic epilepsy**
WANG T (China)

POSTER SESSIONS

p246 **Subjective well-being (SWB) in men with adult onset epileptic seizures (AOES) during the pre-ictal stage of a complex partial seizure (CPS)**
RAFFAELE M (Australia)

p247 **Evaluation of cognitive function in untreated patients with generalized tonic-clonic seizure in rural China**
DING D[1], HONG Z[1], WANG W[1], WU J[1], DE BOER HM[2], SANDER JW[3] ([1]China, [2]Netherlands, United Kingdom[3])

p248 **Memory functions of epilepsy patients with temporal lesions before and after epilepsy surgery**
KWOK FY, CHAK WK (Hong Kong)

Social issues

Refer to pages 217-220 for abstracts

p249 **Knowledge, attitude and practices on epilepsy among care-takers of Myanmar children with epilepsy**
MIN AYE AM, LINN K (Myanmar)

p250 **Knowledge, attitudes and practice toward epilepsy among epilepsy patients in Ladakh, a rural, mountaineous region of Himalayas, India**
SRIVASTAVA PV, MOHD G, THINLEY T, NORBOO T (India)

p251 **Parental cognitive appraisals and coping behaviours following child´s epilepsy diagnosis: a qualitative study**
NGUYEN S, KETTLER L, PERTINI M, FRANCIS M (Australia)

p252 **Adventure therapy: 'a life changing experience'**
CLARKE L, TODD L, MARCHINTON T (Australia)

p253 **North indian women with epilepsy: a survey of medical and social concerns**
KAUSHAL S, TANDON S, TANEJA A, SINGH G (India)

p254 **Evaluating a specialist epilepsy bereavement service**
PRESTON J, HANNA J (United Kingdom)

Surgery

Refer to pages 220-228 for abstracts

p255 **Psychiatric symptoms changes after corticoamygdalohippocampectomy in medial temporal lobe epilepsy patients through Symptom Checklist-90-Revised**
GUANGMING Z, WENJING Z, HUANCONG Z, FUQUAN Z, YAN Z, ZHAOHUI S (China)

p256 **Pre-surgical evaluation and surgical treatment of epilepsies in adults**
SITNIKOV AR, GRIGORYAN Y, MISHNYAKOVA L (Russian Federation)

p257 **Para-insular hemispherotomy: a new modification of peri-insular technique**
CHANDRA S, GARG A, GAIKWAD S, BAL C, SARKAR C, TRIPATHI M (India)

POSTER SESSIONS

p258 **Paediatric epilepsy surgery program in New Territories West Cluster, HKSAR: audit of seizure outcome from a tertiary referral center**
CHAK WK, FONG D, YAM KY, LAI TT, WONG YC, KWOK F, YIP R (Hong Kong)

p259 **One stage surgery of tuberous sclerosis patients with multiple epileptic foci**
LIU S-Y, AN N, YANG H, YANG M-H, HOU Z, LIAO W, ZHANG Q, CAI F-C (China)

p260 **Predictors and outcomes following surgery in childhood temporal lobe epilepsy**
JOHNSON AM, CONNOLLY AM, SUGO E, LAWSON JA, MALONE SA, BOYLE RC, BYE AME (Australia)

p261 **Staged total callosotomy for Lennox-Gastaut syndrome by surgical section or gamma-knife: two case reports**
KO T-S, LEE E-H, JEONG M-H, YUM M-S, HONG S-H, LEE J-K (Korea, Republic of)

p262 **Epilepsy surgery in tuberous sclerosis complex: emphasis on surgical candidate and neuropsychology**
LIANG S, LI A, JIANG H, DING C (China)

p263 **Supratentorial pilocyitic astrocytoma-associated epileptic intractability related with tumor location and morphology**
NISHIMOTO T, IIDA K, HASHIZUME A, KAGAWA K, HANAYA R, SUGIYAMA K, ARITA K, KURISU K (Japan)

p264 **Intracranial electroencephalographic findings and histopathological features in epilepsy patients with dysembryoplastic neuroepithelial tumor (DNT) and DNT-like lesions**
KAGAWA K, IIDA K, HASHIZUME A, NISHIMOTO T, KIURA Y, HANAYA R, SUGIYAMA K, ARITA K, KURISU K (Japan)

p265 **Epilepsy surgery In Indonesia: how to start the program in limited situation**
MUTTAQIN Z (Indonesia)

p266 **3 cases of disconnection syndrome after staged total callosotomy for intractable epilepsy**
TODA K, BABA H (Japan)

p267 **Sectioning of the corpus callosum for refractory epilepsy**
ZHOU J, LUAN G (China)

p268 **Dense array EEG for the tuberous sclerosis complex with epileptic seizure and intraventricle tumor**
TOYODA G, FUJIMOTO A, YAMAZOE T, ENOKI H, OKANISHI T, YOKOTA T, YAMAZAKI M, YAMAMOTO T (Japan)

p269 **Epilepsy surgery for extratemporal epilepsy, reasons for surgical failure**
WONG TH, WONG CH, BIRKETT J, MCANALLY L, DEXTER M, SOMERVILLE E, BLEASEL A (Australia)

POSTER SESSIONS

Neuroimaging
Refer to pages 228-236 for abstracts

p270 **Methionine positron emission tomography (Met-PET) in symptomatic epilepsy**
OTSUBO T, FUJITA S, HANAYA R, SUGATA S, ARITA K, YAMASAKI I, YATSUSHIRO K, NAKAMURA K, FUJIMOTO T (Japan)

p271 **EEG-fMRI identifies focal cortical BOLD signal change in some individuals with childhood absence epilepsy**
CARNEY PW, FLANAGAN D, SCHEFFER IE, BERKOVIC SF, JACKSON GD (Australia)

p272 **3-Tesla 1H MRS in Chinese temporal lobe epilepsy patients**
ZHAO Y, LU Q (China)

p273 **[18F]-Flumazenil-PET for the localisation of medically refractory focal epilepsy using without need for arterial blood sampling: a pilot study**
VIVASH L, GREGOIRE M-C, LAU EW, WARE RE, ROSELT P, BINNS D, BOUILLERET V, KATSIFIS A, MYERS DE, HICKS RJ, O'BRIEN TJ (Australia)

p274 **GABA-A receptor density abnormalities associated with seizure outcomes in epileptogenic cavernous angioma**
KONDO A, MATSUDA K, BABA K, TOTTORI T, UMEOKA S, NAKAMURA F, TERADA K, USUI K, MIHARA T, AGARI T, DATE I, INOUE Y (Japan)

p275 **The influence of the morphological brain changes on electrogenesis and pharmacoresistance in epileptic patients**
KHORSHEV SK, KORSAKOVA EA, POLYAKOV YI (Russian Federation)

p276 **FDG PET and Iomazenil SPECT study of late onset medial temporal lobe epilepsy (mTLE) patients with subtle amygdalar enlargement and slight increased signal on MRI**
MATSUDA K, BABA K, USUI N, UMEOKA S, KONDO A, TOTTORI T, MIHARA T, INOUE Y (Japan)

p277 **Imaging mouse models of brain network disorders**
KOLBE S, GAVRILESCU M, WRIGHT D, JOHNSTON L, O'BRIEN TJ, EGAN G, HILL EL (Australia)

p278 **Ictal SPECT in paediatric epilepsy of temporal lobe origin: correlation of perfusion patterns with surgical outcome**
PUSHPALATHA SUDHAKAR L, SITA JS, MANAS P, SUNDARAM C, PRABHAKAR RAO VVS (India)

p279 **Abnormal MRI finding in focal cortical dysplasia: correlation with surgical outcome and pathologic finding**
KIM S, KIM DW, CHU K, LEE SK, CHUNG CK (Korea, Republic of)

p280 **In vivo MRS glutamate/glutamine levels in brain tumour associated epilepsy**
LIUBINAS SV, YUEN T, MOFFAT B, STEWARD C, D'ABACO G, PARADISO L, KOKKINOS C, ROSENTHAL M, DESMOND P, DRUMMOND KJ, KAYE AH, O'BRIEN TJ, MOROKOFF AP (Australia)

POSTER SESSIONS

p281 **Hyperactive putamen in patients with paroxysmal kinesigenic choreoathetosis: a resting-state functional magnetic resonance imaging study**
ZHOU B, CHEN Q, ZHANG Q, CHEN L, GONG Q, SHANG H, TANG H, ZHOU D (China)

p282 **Investigation of ictal NIRS in children with intractable epilepsy**
ISHIYAMA A, NAKAGAWA E, SUKIGARA S, OKAZAKI T, HIYANE M, FUKUMURA S, AIZAKI K, TAKESHITA E, SAKUMA H, SAITO Y, KOMAKI H, SUGAI K, SASAKI M, KOBAYASHI I (Japan)

p283 **Cortical reorganization and reduced efficiency of visual word recognition in right temporal lobe epilepsy: a functional MRI study**
JENSEN E, HARGREAVES I, PEXMAN P, GOODYEAR B, FEDERICO P (Canada)

p284 **Single-subject voxel-based T2 relaxometry in focal epilepsy of uncertain origin**
KOSIOR R, SHARKEY R, FRAYNE R, FEDERICO P (Canada)

Miscellaneous

Refer to pages 236-247 for abstracts

p285 **Epilepsy education at the "grass root level" using simple tools - a prototype model: an Indian perspective**
KULKARNI C (India)

p286 **The effects of opiate receptor agonists and antagonists on spontaneuse seizure activity in hippocampal slices**
ALIJARAHI A, AMINI M (Iran, Islamic Republic of)

p287 **Knowledge of epilepsy among paediatric nurses in an acute regional hospital**
LEE WC, YIP LH (Hong Kong)

p288 **First pediatric epilepsy service in Myanmar**
LINN K, MIN AYE AM (Myanmar)

p289 **Effect of valproic acid on carotid artery intima-media thickness in epileptic patients**
ZHAO J, ZHANG M, LENG Y, DENG L, LUO X, GONG L (China)

p290 **Treating epilepsy on board a train in India: can this be a sustainable and successful model for epilepsy care delivery in India?**
SINGH MB, PRASAD K, PADMA MV, BHATIA R, BEHARI M (India)

p291 **Naturalistic study on the efficacy of topiramate, as monotherapy in high doses: a clinical trial on 18 cases of resistant seizures to common AED**
NAGARAJAN V (India)

p292 **Free testosteron level and sperm assessment in people with epilepsy among Indonesian population in Jakarta**
ISTIAWATI SE (Indonesia)

p293 **Characterization of the anti-epileptic properties of a complement factor 5a antagonist in mouse models of epilepsy**
THOMAS NK, BENSON M, WOODRUFF T, TAYLOR S, BORGES K (Australia)

POSTER SESSIONS

p294 **Experiences on pharmacist epilepsy counseling service in specialist outpatient clinics**
HIE SL, LEE SHD (Singapore)

p295 **Working of free epilepsy clinic in the community: 25 years experience**
CHAUDHRY HR, SHABBIR A, SHAFIQ N, USMAN S (Pakistan)

p296 **An unusual case of Hashimoto's encephalopathy associated with thyrotoxicosis**
TOMITA S, KINOSHITA M, TAHARA M, YAMAKAWA K, OEDA T, SAWADA H, KONISHI T (Japan)

p297 **Prophylactic phenytoin in cases of cerebral malaria**
BHANDARI G, MALOO JC (India)

p298 **Towards consumer directed service delivery**
IRELAND C, KAIN S, TODD L, WELSH C (Australia)

p299 **Utility of long-term video-EEG monitoring in older adults and elderly**
BAHETI NN, RADHAKRISHNAN A, RADHAKRISHNAN K (India)

p300 **Antiepileptic effects of triheptanoin in genetic absence epilepsy rats from Strasbourg (GAERS)**
SHE D, JONES N, O'BRIEN T, BORGES K (Australia)

p301 **Insights into epilepsy through visual art**
CHAMBLISS J[1], COOK M[1], BOLT B[1], WILLIAMS D[1], COSS E[2], PINIKAHANA J[1], BARDENHAGEN F[1], MEADE C[1], SIMPSON L[1] ([1]Australia, [2]Singapore)

p302 **Ketogenic diet in refractory epilepsies**
THAMMONGKOL S, VEARS D, BICKNELL-ROYLE J, NATION J, DRAFFIN K, STEWART KG, CAMERON F, SCHEFFER I, MACKAY M (Australia)

p303 **Risk of seizure recurrence after anti-epileptic drug withdrawal in neurocyticercosis treated and non-treated group**
GORKHALY MP (Nepal)

p304 **Effect of insulin and melatonin on kainic acid induced learning impairment in rats**
NIKBAKHT F, YEGANE F (Iran, Islamic Republic of)

PLATFORM SESSION ABSTRACTS

Neuroimaging
Friday 22nd October
15:00-16:00
Room 104

001
Source localization of IEDs from simultaneous high-density EEG and fMRI in focal epilepsy
VOGRIN SJ[1], LAU S[1,2,3], KUHLMANN L[2], D'SOUZA W[1], HAUEISEN J[3], COOK MJ[1]
[1]Centre for Clinical Neurosciences and Neurological Research, Fitzroy, Victoria, Australia, [2]Neuroengineering Group, Dept of Electrical & Electronic Engineering, University of Melbourne, Melbourne, Victoria, Australia, [3]Institute of Biomedical Engineering and Informatics, Ilmenau Technical University, Ilmenau, Thuringia, Germany

Purpose: A major challenge in planning and performing resective surgery in drug-resistant epilepsy is the accurate identification of the epileptogenic zone. Currently, intracranial EEG is considered to be the most reliable preresective functional localisation method. Our objective is to non-invasively localise the epileptogenic zone using simultaneous fMRI and high-density EEG source localisation (ESL).
Method: We recorded 128 or 64 channel EEG (10kHz) simultaneously to fMRI (1.5T) in patients with focal epilepsy for 35 mins to capture interictal epileptic discharges (IEDs). An additional 35 min EEG recording was performed in a shielded room. EPI artefacts in the EEG were removed with average artefact subtraction. The ballistocardiogram (BCG) was removed using PCA projection. A patient-specific realistic boundary element model (skin, skull and brain) was constructed from a 1mm isotropic MPRAGE image and a cortically constrained current density source model was applied to individual IEDs.
Results: The results show visual agreement of locations described by fMRI and ESL in the MR scanner and shielded room, respectively. The found locations are consistent with video EEG monitoring, PET and ictal SPECT (where available).
Conclusion: Simultaneous high-density EEG and fMRI acquisition non-invasively provides added localising information in a multimodal context. Single event analysis facilitates resolving multiple epileptogenic foci. Concordant simultaneous ESL and fMRI localisation shows promise in improving non-invasive presurgical evaluation of epilepsy.

002
Pre- and post-surgical investigation of cerebral blood flow using NIRS in patients with hemispherical cortical dysplasia
SUKIGARA S[1], NAKAGAWA E[1], AIZAKI K[1], ISHIYAMA A[1], TAKESHITA E[1], OKAZAKI T[1], HIYANE M[1], FUKUMURA S[1], SAKUMA H[1], SAITO Y[1], KOMAKI H[1], SUGAI K[1], SASAKI M[1], TAKAHASHI A[2], OTSUKI T[2], KOBAYASHI I[3]
[1]Department of Child Neurology, National Center Hospital of Neurology and Psychiatry, Tokyo, Japan, [2]Department of Neurosurgery, National Center Hospital of Neurology and Psychiatry, Tokyo, Japan, [3]Center for the Research and Support of Educational Practice, Tokyo Gakugei University, Tokyo, Japan

Purpose: This study aimed to confirm the utility of near-infrared spectroscopy (NIRS) in determining pre- and post-surgical changes in cerebral blood flow (CBF) and cerebral blood oxygenation (CBO) in patients with diffuse hemispherical cortical dysplasia by comparing findings with those of single photon emission computed tomography (SPECT). A further aim was to determine whether any correlation exists between CBO patterns on NIRS and seizure outcome post-surgery.
Method: Subjects were 8 patients with hemispherical cortical dysplasia admitted at some point between January 2008 and May 2010. Hemodynamic patterns of oxy- and deoxy-hemoglobin after photic stimulation during sleep were evaluated before and after hemispherotomy using the ETG 4000 (Hitachi Medical Corp.) and compared with ECD-SPECT findings. Seizure outcome was investigated from medical records. Five of the 8 cases were analyzed (3 were excluded due to body motion artifacts).

PLATFORM SESSION ABSTRACTS

Results: Pre-surgically, like SPECT, NIRS showed CBF was increased in the affected hemisphere and decreased in the unaffected hemisphere in all patients. Post-surgically, 4 patients who were seizure-free showed CBF was decreased in the operated hemisphere and increased in the unaffected hemisphere compared with before hemispherotomy. However, one case with recurrent seizure showed no significant CBF increase in the unaffected hemisphere, as seen on SPECT. NIRS additionally revealed interesting hemodynamic patterns—arrhythmic or asynchronous changes in oxy- and deoxy-hemoglobin—in the unaffected hemisphere.
Conclusion: NIRS is a useful, non-invasive tool to confirm hemodynamic patterns in patients with diffuse hemispherical cortical dysplasia. It has better temporal resolution than SPECT and can predict post-surgical seizure prognosis.

003
Voxel-based morphometric study of white matter tracts in patients with non-lesional temporal lobe epilepsy
LEUNG H[1], WANG RD[2], SHI L[1], LAM WW[3], KWAN P[1], WONG KS[1], MOK V[1]
[1]Chinese University of Hong Kong, Shatin, Hong Kong, [2]Department of Radiology, Chinese University of Hong Kong, Shatin, Hong Kong, [3]Hong Kong Health Check, Kowloon, Hong Kong

Purpose: Diffusion tensor imaging (DTI) is playing an increasing role in the presurgical evaluation of refractory temporal lobe epilepsy (TLE). We hypothesized that DTI may be able to identify abnormalities among patients with non-lesional TLE.
Method: Fourteen patients (mean-age 43, 40% male) with refractory non-lesional TLE and unilateral ictal electrographic onsets were recruited. Sixteen subjects(mean-age 50, 56% male) served as normal controls. The imaging protocol consisted of 1.5T magnetic resonance imaging (MRI) using 31 diffusion-gradient directions. Fractional anisotropy(FA) was calculated from the diffusion tensors. Voxel-based morphometry (VBM) and tract-based spatial statistics (TBSS) were used in the analysis of white matter tracts.
Results: With two-sample unpaired t-test examining any difference between the left TLE group and the normal subjects in terms of lower FA values, an extensive network of white matter tracts in both cerebral hemispheres was found ($p < 0.001$). A similar TBSS between the right TLE group and the normal subjects revealed a similar network ($p < 0.001$). The magnitude of FA reduction was pronounced in inferior longitudinal fasciculi and uncinate fasciculi with the mean reduction of FA in contralateral fibres (0.073) being marginally greater than the mean reduction of FA in ipsilateral fibres (0.069).
Conclusion: The use of VBM and TBSS in DTI may form part of the multi-modal investigations of patients with refractory non-lesional TLE. They may demonstrate abnormalities among white matter tracts which might otherwise be reported as normal on conventional MRI.

004
Voxel-based optimized morphometry (VBM) in the identification of focal cortical dysplasias (FCDs) in patients with refractory seizures
RADHAKRISHNAN A, JAMES JS, KESAVADAS C, THOMAS B, RADHAKRISHNAN K
Sree Chitra Tirunal Institute for Medical Sciences and Technology, Trivandrum, India

Purpose: Identification of indistinct and ill-defined FCDs responsible for refractory seizures is difficult even with the high-resolution magnetic resonance imaging (MRI) techniques and sequences. Detection of such FDCs will help the epileptologist to proceed with presurgical evaluation and surgery. Aim of the present study was to use VBM to identify such subtle FCDs not well defined in 1.5 T MRI.

PLATFORM SESSION ABSTRACTS

Method: Sixteen patients with refractory seizures and ill-defined FCDs suspected in T1W and 3D FLAIR MRI from September 2006-September 2009 were chosen (mean age 34.6 +9.7 years; Male/Female-10/6; temporal: extra temporal 10:6). 15 normal controls (mean age 35.2 + 11.2 years; Male: female-10/5) were also studied. MRI was acquired on 1.5 T (Siemens, Magnetom, Avanto, Germany) system. T1W 3D-FLASH images with TR/TE/FOV/Flip angle of 11s/4.94s/23cm/15^0, matrix-size 256 x 224, slice thickness 1.5mm, pixel spacing 0.89mm were used. Normalization, segmentation, smoothing and voxel-wise statistical analysis were done. Data were analyzed using SPM -5 running MATLAB 6.1 version.
Results: By VBM, 13/16 (80.6%) of the lesions could be identified by scientists blinded to MRI findings. Six of them underwent lesion resection, who had concordant clinical and EEG correlates and is seizure-free at a mean follow-up of 1.5 years (mean 1-3.8years). Pathology revealed FCD Type I and II in 4 and 2 patients respectively. None of the controls had statistically significant gray-white differences by VBM.
Conclusion: VBM is a novel MRI technique to detect ill-defined FCDs accurately in majority of patients with refractory seizures which may go undetected in a high-resolution 1.5T MRI.

005
Intracranial EEG-fMRI study of focal epileptiform discharges in humans: evidence of a default state
CUNNINGHAM C, GOODYEAR B, BADAWY R, PITTMAN D, FEDERICO P
University of Calgary, Calgary, Canada

Background: Combining intracranial EEG (ICE) with functional MRI (fMRI) is of particular as it would allow the detection of much smaller epileptiform discharges than scalp EEG-fMRI, and may help further investigate the mechanisms of seizures. To our knowledge, ICE-fMRI never been performed.
Method: fMRI was performed at 3T with concurrent intracranial EEG in two subjects who had subdural strip electrodes placed according to clinical protocol. Subject 1 was a 20-year-old female with bilateral periventricular gray matter heterotopia. Subject 2 was a 29-year-old female with independent bilateral temporal seizures and a normal MRI.
Results: Subject 1 showed spike-associated BOLD signal increases bilaterally in the parietal lobes, superior temporal gyri and anterior cingulate gyrus (105 spikes). BOLD decreases were seen bilaterally in the frontal and parietal lobes. In Subject 2, BOLD increases were seen in both mesial temporal lobes, which was greater on the side of discharge when the independent left and right spikes were modeled separately (194 left spikes, 284 right spikes). Striking BOLD decreases were also seen in the thalamus and posterior cingulate gyrus.
Conclusion: Intracranial EEG-fMRI can be performed safely at 3 Tesla. Only 5 or 10 min of ICE-fMRI was performed as part of our implementation protocol, many discharges were seen, allowing meaningful analyses. With these studies, we have shown that resting state network deactivation as well as thalamic deactivation is associated with focal epileptiform discharges. This suggests a novel mechanism through which focal interictal discharges may have widespread cortical and subcortical influences.

Clinical neurophysiology and seizure semiology
Friday 22nd October
15:00-16:00
Room 106

006
Use of post ictal respiratory pattern to discriminate between convulsive non-epileptic seizures and generalised tonic clonic seizures
ROSEMERGY I[1], FRITH R[1], HERATH S[2], WALKER E[1]
[1]Neurology Department, Auckland, New Zealand, [2]Respiratory Department, Auckland, New Zealand

PLATFORM SESSION ABSTRACTS

Three authors examined edited video recording of the post ictal phase of 72 convulsive seizures recorded in the video-telemetry monitoring unit at Auckland Hospital. The examiners were blinded to the nature of the seizure. There were 59 generalised epileptic seizures and 13 generalised convulsive non epileptic seizures (NES). Measurements of respiratory rate at zero, 2, 4, 6, and 8 minutes were recorded, as well as time to return to normal breathing pattern. Those patients who had genuine epileptic seizures had a faster initial respiratory rate than those with non-epileptic seizures, and this was statistically significant at 2 minutes and 8 minutes post seizure. Patients with non-epileptic seizures returned to normal respiratory pattern twice as fast as those with genuine seizures. The absence of snoring was highly sensitive but only moderately specific for identifying non-epileptic seizures. Post ictal respiratory rate and pattern can be a reliable discriminating factors between generalised convulsive seizures and convulsive non epileptic seizures.

007

Lateralizing value of ictal head deviation in mesial temporal lobe epilepsy

JIN L, WU L
Department of Neurology, Peking Union Medical College Hospital, Chinese Academy of Medical Sciences, Beijing, China

Purpose: To investigate the lateralizing value of various ictal head deviation(HD) in patients with mesial temporal lobe epilepsy.
Method: Presurgical videotapes of 206 complex partial seizures (CPS) with or without secondarily tonic-clonic seizures (SGTC) from 43 patients who had at least one HD and were seizure-free for at least 1 year after surgery were retrospectively reviewed. Two forms of HD, versive HD(VHD) defined as forced, sustained and unnatural head turning, and non-VHD(NVHD) defined as not forceful or sustained enough to be considered version, were identified. The correlation of HD occurring at different phases of CPS to the side of resection was investigated to determine lateralizing value by evaluating positive predictive value (PPV) of various HD.
Results: The overall frequency of HD was 42.7% of 206 CPS. VHD occurred mainly in the second half of CPS with SGTC, second half of CPS only, and first half of CPS with SGTC.VHD occurring in these three different phases was contralateral to the epileptogenic zone with the PPV of 89.4%, 100% and 100%, respectively. NVHD being ipsilateral to resected side appeared predominantly in the first and second half of CPS only, and first half of CPS with SGTC, each with PPV of 73.9%, 100% and 100%, respectively.
Conclusion: HD is a useful lateralizing sign in CPS of mesial temporal lobe origin, appearing contralateral and ipsilateral to the epileptogenic zone for VHD and NVHD, respectively. Lateralizing significance varies for each form of HD occurring at different phases of CPS with or without SGTC.

008

Latency to first interictal epileptiform discharge in epilepsy with outpatient ambulatory EEG recording

FAULKNER HJ, MOHAMED A
Royal Prince Alfred Hospital, Sydney, Australia

Purpose: The diagnosis and classification of epilepsy often relies upon the demonstration of interictal epileptiform discharges (IEDs) on EEG rather than recording ictal abnormalities. A single 20 minutes EEG is reported to have sensitivity as low as 36 % in patients with epilepsy, with repeat EEG increasing the sensitivity to a maximum of 77%. An alternate strategy to repeat EEG is the use of prolonged continuous ambulatory EEG; however, there is no data on the average latency to first IED with continuous monitoring.
Method: In this retrospective study we analysed 180 consecutive 96-hour (5 day) outpatient ambulatory EEGs, without medication withdrawal, where IEDs were recorded.

PLATFORM SESSION ABSTRACTS

Results: Mean latency to first IED was 630 minutes (range 1·4569 minutes, n=180). Mean latency to first IED in primary generalised epilepsies was 121 minutes (range 1·901 minutes, n=34). Mean latency to first IED in focal onset epilepsies was 750 minutes (range 1·4569 minutes, n=146). The difference between the latencies to interictal epileptiform discharges with primary generalised epilepsies and focal onset epilepsies was statistically significant (p=< 0.05). IEDs were recorded in 44% of patients within 4 hours, 85% within 24 hours and 95% within 48 hours of recording.
Conclusion: Our data is the first to show the latency to recording interictal epileptiform discharges with continuous EEG monitoring in outpatients without medication withdrawal. A 48·hour recording is sufficient for classification in the majority of patients; only 5% in our study required more prolonged recording for electro·clinical classification.

009
The utility of one day video EEG (VEEG) recording in the diagnosis of non-epileptic seizures
CHEN L, KHUN TT, WONG TH, BLYTH K, BLEASEL A
Westmead Hospital, Sydney, Australia

Purpose: Outpatient one day VEEG is suitable for the diagnosis of frequent paroxysmal clinical events. We hypothesised non-epileptic seizures would be more likely to occur during an outpatient VEEG than epileptic seizures regardless of the reported frequency of the events.
Method: Retrospective study of all one day VEEG between January 2005·March 2010. Patients were divided into epilepsy and non-epilepsy with medical records, EEG, and later inpatient VEEG. No placebo induction techniques were used. Chi-squared test was used to compare the observed number of seizures to that expected using the reported frequency of events for a subject. An F test was used to compare the discrepancy between observed and expected numbers of seizure by patient group.
Results: Of 222 patients with one day VEEG; 40% of 100 patients with epilepsy had events recorded (6 non-epileptic seizures) and 65% of 122 patients without epilepsy had events recorded (all non-epileptic). 113 patients reported daily seizures and 76% had clinical events recorded, 45 patients reported weekly seizures and 46.7% had clinical events, 40 reported monthly seizures and 20% had clinical events. There were significantly more seizures observed than expected on the basis of historical reports in both the non-epileptic and in the epileptic groups (p< 0.0001 for each group). The excess of observed over reported rates was significantly higher in the non-epileptic group than in the epileptic group (p< 0.0001).
Conclusion: Patients with non-epileptic seizures have a high likelihood of their events being recorded even if the events are not reported as daily.

Psychiatry and neuropsychology
Friday 22nd October
15:00-16:00
Room 109

010
Differential contributions of objective memory and mood to subjective memory complaints in refractory focal epilepsy
RAYNER G[1], WRENCH JM[1], WILSON SJ[1,2]
[1]The University of Melbourne, Parkville, Victoria, Australia, [2]Comprehensive Epilepsy Programme, Austin Health, Melbourne, Victoria, Australia

Purpose: Patients with epilepsy frequently present with bitter memory complaints. Yet the determinants of subjective memory complaint in epilepsy remain ill·defined, with research to date variously attributing these complaints to either symptoms of mood disturbance or objective memory deficits. The purpose of this study was to investigate the influence of the epileptogenic region on this variability in a sample of patients with focal epilepsy.

PLATFORM SESSION ABSTRACTS

Method: In total, 96 adults participated in the study. Of these, 60 were diagnosed with medically refractory focal epilepsy as part of pre-surgical characterization in the Comprehensive Epilepsy Program of Austin Health. Patients were grouped according to mesial temporal (MT, n=39) or non-mesial temporal (NMT, n=21) foci, and contrasted with 36 neurologically-normal controls. All participants were compared on measures of mood and subjective and objective memory functioning. Hierarchical multiple regressions were then used to examine the extent to which objective memory performance, current mood symptoms, and a history of depression predicted subjective memory complaints in the MT and NMT patients.
Results: In contrast to controls, both patient groups were highly concerned about their memory ($p < 0.001$), and were more likely to have a history of depression ($p=0.005$). Multiple regression showed that objective memory dysfunction and current depressive symptoms predicted the memory complaints of MT patients ($p=0.005$), while a history of depression predicted the complaints of NMT patients ($p=0.008$).
Conclusion: These findings suggest that patients have concerns about their memory underpinned by distinct psychological and neurobiological factors depending on the location of the epileptogenic focus.

011

Working memory-induced activation and severity of psychopathology in schizophrenia-like psychosis of epilepsy: evidence from magnetoencephalography

CANUET L[1], ISHII R[1], IWASE M[1], IKEZAWA K[1], KURIMOTO R[1], TAKAHASHI H[1], CURRAIS A[2], AZECHI M[1], AOKI Y[1], NAKAHACHI T[1], SORIANO S[3], TAKEDA M[1]
[1]Osaka University, Suita, Japan, [2]King's College London, London, United Kingdom, [3]Loma Linda University, Loma Linda, USA

Purpose: The purpose of this study was to determine the pattern of brain oscillatory activity underlying working memory (WM) dysfunction in schizophrenia-like psychosis of epilepsy (SLPE), and to assess whether neural activity in WM-compromising brain regions correlates with the severity of psychopathology in these patients.
Method: Twelve patients with SLPE and 14 with nonpsychotic epilepsy were recruited. Neuromagnetic activity was recorded using a 64-channel whole-head magnetoencephalography system while patients performed a visual-object WM task. Patients with gross organic lesions and an IQ score below 70 were excluded. The Brief Psychiatric Rating Scale (BPRS) was used to assess psychopathology. Multiple Source Beamformer and Brain-Voyager were used for neuroimaging analysis. The region of maximal activation within the anatomical areas that showed significant activation deficits were determined for correlation with clinical symptom scores.
Results: Significant differences were found exclusively in alpha frequency band: compared to nonpsychotic epilepsy controls, patients with SLPE showed greater activation, as indicated by alpha event-related desynchronization in the right dorsolateral prefrontal cortex (DLPFC), and event-related synchronization in the left inferior temporal cortex. Significant positive correlations were found between WM-induced DLPFC activation and severity of disorganization ($r=0.74$; $p=0.005$).
Conclusion: Our findings indicate that dysfunction in prefrontal areas engaged during active maintenance of information in WM may be critical to some domains of psychopathology, in particular disorganized thought processing in SLPE. These convergent cognitive abnormalities and psychopathological disturbance in the DLPFC suggests a major role for this cortical region in the pathophysiology of SLPE.

012

Memory changes in individuals with childhood-onset lesional TLE: a follow-up study

MAHDAVI N[1,2], GONZALEZ L[1,3], ANDERSON V[1,2,3], AUSTRALIAN CENTRE FOR CHILD NEUROPSYCHOLOGY STUDIES
[1]Murdoch Children's Research Institute, Parkville, Australia, [2]University of Melbourne, Parkville, Australia, [3]Royal Children's Hospital, Parkville, Australia

PLATFORM SESSION ABSTRACTS

Purpose: Our study examined changes over time in memory function in children, adolescents and young adults with lesional TLE of childhood onset. While research suggests that memory impairments in adults with TLE resembles material-specific patterns, research with children has yielded inconsistent results and little is known of memory impairments in adolescents with TLE.
Method: Twenty-four participants aged 8 to 23 years were re-assessed after an initial baseline study; 13 individuals with right TLE (RTLE) and 11 with left TLE (LTLE). Fourteen participants had undergone epilepsy surgery since the initial study and 10 were managed medically. The design included tests of verbal and non-verbal memory, immediate memory, working memory and intellectual functioning.
Results: Contrary to expectation, memory performances were more lateralised or lesion specific at follow-up with greater memory impairments present in those with LTLE. Change over time analyses yielded that the RTLE group improved in memory over time and the LTLE group remained stable or declined in memory, regardless of whether the tasks assessed visual or verbal memory skills and regardless of surgical intervention. Age of seizure onset and number of medications were predictive of memory performance at follow-up. Those with early age of seizure onset (before the 5th year of life) and LTLE were at greatest risk of verbal memory impairments.
Conclusion: This study provides a step forward in understanding the natural progression of epilepsy (in the absence of surgical intervention) and highlights differential memory trajectories arising from TLE according to laterality of lesions.

013
Is there 'phantom of dementia' in elderly who have undergone ATL for refractory MTLE?
ALEXANDER A, RADHAKRISHNAN A, RADHAKRISHNAN K
Sree Chitra Tirunal Institute for Medical Sciences and Technology, Trivandrum, India

Purpose: One of the deficits patients report after anterior temporal lobectomy (ATL) is worsening of memory. As these patients reach their sixth decade, aging can compromise their cognitive functions further. We undertook this study on patients who have undergone ATL for refractory mesial temporal lobe epilepsy who are >50 years of age during the study period to quantify their memory function and to ascertain presence of any form of dementia in them as compared to age and sex matched healthy controls.
Method: Out of the 724 patients who underwent ATL from 1995-2008, there are twenty four who are >50 years during the study period. We administered a detailed dementia protocol assessing all domains of cognition including overall cognitive ability, memory, executive, visuo-spatial functions, confrontation naming, verbal fluency, Instrument for activities of daily living, QOL, Becks depression, HADS and neuropsychiatry inventory.
Results: The mean age was 54.04+4.8 years (14 males; 10 females). On comparison with normative data, patients showed no significant impairment in most of the domains. None had any significant features of anxiety or depression. All of them were independent for ADL and had good QOL.
Conclusion: Despite the fact that patients who are subjected to ATL lack one hippocampus and part of temporal neocortex, it is evident from this study that a significant re-organization would have had occurred in their memory and neurocognitive network to compensate for it. This study emphasizes that there is no risk of cognitive decline or dementia in patients subjected to ATL as they reach older age.

014
Psychogenic non-epileptic seizures (PNES): can electro-clinical features aid in distinguishing cases with psychopathology?
LIM KS[1,2], SARAH W[1], SCHEFFER I[1], BERKOVIC S[1]
[1]Epilepsy Research Centre and Department of Medicine (Neurology), University of Melbourne, Austin Health, Melbourne, Australia, [2]University of Malaya, Kuala Lumpur, Malaysia

Purpose: To characterise a group of patients with psychogenic non-epileptic seizures (PNES) without psychopathology at Austin Comprehensive Epilepsy Program, 1997-2009.

PLATFORM SESSION ABSTRACTS

Method: Of 81 patients with habitual PNES captured on video-EEG monitoring who underwent psychiatric assessment, 12/81 (14.8%) had no evidence of psychopathology. Their electro-clinical and psychosocial variables were compared with patients with identified psychopathology.
Results: 11/12(92%) were female. The mean age of onset was 25.6 years (SD 14.7), mean age of presentation 33.8 years (SD 13.7), with a mean delay in diagnosis of 64.7 months (SD 140.1). The mean seizure frequency was 38.8 seizures/month (SD 58.9). Psychosocially, 4/9(44%) adult patients were single and 10/12(83%) lived with their family. 8/12(66%) were unemployed, and none had tertiary education. Half had asynchronous high-amplitude limb movements, with pelvic thrusting and head shaking (typical motor type), 2 had atypical motor type with minor limb movement, 2 had blank staring, and none collapsed. 5 cases had co-existing epilepsy; 2 had epileptiform discharges on EEG. MRI abnormalities were seen in 3 cases. Compared to the cohort with identified psychopathology, this cohort had a higher female ratio (91.7% vs. 69.6%), none had tertiary education (vs. 23.2%), none collapsed (vs. 15.9%), and they were less likely to have epileptiform discharges (16.7% vs. 34.8%) and MRI abnormalities (25.0% vs. 41.5%). All variables tested were, however, not significantly different.
Conclusion: Patients with PNES without identified psychopathology do not present a distinctive picture electroclinically or psychosocially from patients with psychopathology.

Neurobiology
Friday 22nd October
15:00-16:00
Room 110

015
No GAD67-positive GABAergic neurogenesis in the dentate gyrus of adult mice in experimental epilepsy models
JIANG W[1], YANG F[1], WANG Y[1], QUAN Q-Y[1], CHEN J[1], WU S-X[2]
[1]Department of Neurology, Xijing Hospital, Xi'an, China, [2]Department of Anatomy, Histology and Embryology, K. K. Leung Brain Research Centre, the Fourth Military Medical University, Xi'an, China

Purpose: We strove to investigate whether the newborn neurons in the epileptic hippocampus can differentiate into inhibitory neurons, i.e. GABAergic interneurons.
Method: We established pentylenetetrazol (PTZ)-induced chronic kindling model to mimic human generalized epilepsy, and lithium-pilocarpine-induced status epilepticus model to reproduce human partial epilepsy. By using BrdU immunocytochemistry, combined with glutamic acid decarboxylase-green fluorescence protein (GAD67-GFP) knock-in mice, in which a GFP gene was introduced into the gene for GAD67 and thus all GABAergic neurons were fluorescent, we determined the identity of newborn neurons in the hippocampus.
Results: Both types of epilepsy significantly increased the number of newborn cells in the dentate gyrus at early time-point after seizures; however, there is a significant loss of newborn cells at 2 weeks after PTZ kindling and 2 months after pilocarpine-induced seizures. About 80% of newborn dentate cells differentiated into neurons in control groups, whereas only 58% and 29% of newborn cells in the dentate gyrus differentiated into neurons in the PTZ-kindling and pilocarpine models, respectively. Double or triple immunofluorescence labeling did not reveal any newborn cells co-labeled with GFP in both intact and epileptic dentate gyrus. A significant decrease in the total number of GABAergic neurons in the dentate gyrus was detected in the pilocarpine-induced status epilepticus model but not in the PTZ-kindling model.
Conclusion: These results indicate that epileptic seizures do not produce new GAD67-positive GABAergic interneurons in the dentate gyrus; rather, prolonged seizures result in the loss of GABAergic interneurons.

PLATFORM SESSION ABSTRACTS

016

Astrocytic uptake of GABA critically regulates thalamic activity in generalized absence epilepsy

BEENHAKKER M, HUGUENARD J
Stanford University, Stanford, USA

Purpose: The inhibitory neurotransmitter GABA paces thalamic activity during generation of Spike Wave seizures of generalized absence epilepsy. High levels of GABA are released in thalamus during SWD and this promotes the seizures through a neural post-inhibitory rebound mechanism. Synaptically released GABA is recycled to maintain continuous availability of this key neurotransmitter, and disruption in GABA metabolism could exacerbate absences. Clinical observation are consistent with this -- GABAergic drugs worsen absence epilepsy.
Method: We used electrophysiological methods to assess GABA release in thalamic brain slices from rodents. $GABA_B$-mediated inhibitory postsynaptic currents (IPSCs) were recorded in thalamic relay neurons while modulators of GATS were applied to the brain slice.
Results: $GABA_B$ IPSCs were differentially regulated by two astrocytic GABA transporter subtypes, GAT1 and GAT3. GAT1 blockade increased $GABA_B$ response amplitude, while GAT3 block in addition produced a more sustained response. We incorporated experimentally determined GAT1 and GAT3 expression patterns into a computational model of synaptic GABA diffusion and showed that differential GAT localization leads to mechanisms through which GABA transients can be modulated to enable distinct $GABA_B$IPSC amplitude/kinetic changes. Perisynaptic expression of GAT1 limits peak GABA concentration near synapses. GAT3 expression is broader and includes distal extrasynaptic regions.
Conclusion: GAT3 plays a critical role in thalamic oscillations -- it limits GABA diffusion away from synapses and thus keeps $GABA_B$ responses small. Failure of GAT3 leads to enhanced diffusion away from synapses and activation of a very large pool of extrasynaptic $GABA_B$ receptors that would otherwise not be activated.

017

The involvement of SVZ-derived neural precursor cells in the epileptogenicity of focal cortical dysplasia

SHU H-F, YANG H
Department of Neurosurgery, Xinqiao Hospital, Third Military Medical University, Chongqing, China

Purpose: Focal cortical dysplasia (FCD) is a common cause of medically intractable epilepsy in pediatric patients. It has indicated that the abnormal cells within the cortical lesions of FCD, which are closely correlated with the severity of seizure, are derived in part from the neural precursor cells (NPCs) of subventricular zone (SVZ). Therefore, to observe the migration, differentiation and the electrophysiological features of the SVZ-derived cells within the cortical dysplastic lesions of FCD will help us better understand the epileptogenicity of FCD.
Method: We created an FCD rat model by focal contact of a frozen metal probe on the scalp immediately after birth, in which the SVZ-derived NPCs were labeled with DiI through intracerebroventricular injection. The neurobiological features of DiI+ cells within the freezing-induced cortical lesions were studied by morphological and electrophysiological methods.
Results: The DiI+ cells, which form a migratory stream from SVZ to cortical lesions, could be continuously observed from P10 to P90. The immunofluorescence results showed that the majority of SVZ-derived NPCs in the cortical lesions differentiated into glial cells and neurons, and to format synaptic connections with the surround cells, suggesting the possibility of these cells to influence the local neural circuit. Electrophysiological studies indicated that the SVZ-NPCs-derived neurons showed high excitability and received more excitatory input, while the feedback suppression to the presynaptic cells reduced.
Conclusion: These data suggested that the high excitability of SVZ-NPCs-derived neurons would result in the hyperexcitability of the local neural circuit, and consequently trigger the kindling of the epileptiform discharge.

PLATFORM SESSION ABSTRACTS

018

Increased stargazin and AMPA receptor expression in somatosensory cortex of genetic absence epilepsy rats from Strasbourg

KENNARD JT[1], BARMANRAY R[1], SAMPURNO S[1], REID CA[2], PARADISO L[3], D'ABACO G[3], KAYE AH[3], FOOTE SJ[4], O'BRIEN TJ[1], POWELL KL[1]

[1]Department of Medicine, University of Melbourne, Melbourne, Australia, [2]Howard Florey Institute, University of Melbourne, Melbourne, Australia, [3]Department of Surgery, University of Melbourne, Melbourne, Australia, [4]The Menzies Research Institute, Hobart, Australia

Purpose: Absence-like seizures in Genetic Absence Epilepsy Rats from Strasbourg (GAERS) are believed to arise from hyperexcitable somatosensory cortical neurons, however the mechanism of increased excitability remains unknown. We have shown that expression of the Transmembrane AMPA receptor Regulatory Protein (TARP), stargazin, is elevated in the somatosensory cortex of GAERS. Here we examined the developmental expression of stargazin and the impact this has on AMPA receptor trafficking in the GAERS model.
Method: Somatosensory cortex tissue was fractionated into cytosol and membrane fractions using the ProteoExtract Subcellular Proteome Extraction Kit and western blotting was performed using antibodies for stargazin, GluA1 and GluA2 with tubulin used as a loading control.
Results: Stargazin expression was elevated in somatosensory cortex plasma membrane fractions in adult GAERS compared to Non-Epileptic Control (NEC) rats (NEC 1.0±0.05, GAERS 1.22±0.05, $p< 0.01$). This was associated with an increase in AMPA receptor proteins, GluA1 (NEC 1.0±0.14, GAERS 1.46±0.12, $p< 0.05$) and GluA2 (NEC 1.0±0.07, GAERS 1.42±0.21, $p< 0.05$) in the same compartment. In 6 week old GAERS, just prior to the age of seizure onset, despite mRNA expression for stargazin being elevated, protein expression was not (NEC 1.0±0.08, GAERS 0.81±0.09, $p>0.05$), nor were GluA1 (NEC 1.0±0.07, GAERS 1.09±0.07, $p>0.05$) or GluA2 (NEC 1.0±0.09, GAERS 0.94±0.11, $p>0.05$).
Conclusion: These data demonstrate that stargazin and AMPA receptor membrane targeting is enhanced in adult epileptic GAERS, potentially driving hyperexcitability in somatosensory cortex. This shows a developmental time course that would suggest a pathophysiological role in the manifestation of the epilepsy phenotype.

Paediatric epileptology
Saturday 23rd October
15:00-16:00
Room 103

019

A new clinical epileptic syndrome caused by SPTAN1 mutation

TOHYAMA J[1,2], SAITSU H[3], AKASAKA N[1], OSAKA H[4], MIYATA R[5], OHASHI T[1], KOBAYASHI Y[1], KATO M[6], MATSUMOTO N[3]

[1]Department of Pediatrics, Nishi-Niigata Chuo National Hospital, Niigata, Japan, [2]Niigata University Medical and Dental Hospital, Niigata, Japan, [3]Departmen of Human Genetcs, Yokohama City University Graduate School of Medicine, Yokohama, Japan, [4]Department of Neurology, Kanagawa Children's Medical Hospital, Yokohama, Japan, [5]Department of Pediatrics, Tokyo Kita Shakai Hoken Hospital, Tokyo, Japan, [6]Department of Pediatrics, Yamagata University School of Medicine, Yamagata, Japan

Purpose: Recently, *SPTAN1* mutations have been identified in West syndrome with severe cerebral hypomyelination. We aimed to investigate the genotype-phenotype correlation in previously reported patients with *SPTAN1* aberration.

PLATFORM SESSION ABSTRACTS

Method: Among the previously reported patients of West syndrome with cerebral hypomyelination, three patients with *SPTAN1* aberration were studied. Patient 1 had a 9q33.3-q34.11 microdeletion including *STXBP1* and *SPTAN1*. Patient 2 has a *de novo* heterozygous in-frame 3-bp deletion (c.6619_6621 del, pE2207del), and Patient 3 has a *de novo* heterozygous in-frame 6 bp duplication (c.6923_6928 dup, p.R2308_M2309 dup) in *SPTAN1*, respectively. We evaluated their clinical and neuroradiological findings in association with genotype of the patients.
Results: Patient 1 showed slight psychomotor development with eye contact, but no head control, and her seizures have been well controlled. Patient 2 and 3 showed severe spastic quadriplegia, no developmental process, no visual attention, and acquired microcephaly. Epileptic spasms were resistant to various treatments. Brain MRI of Patient 2 and 3 revealed widespread brain atrophy including brainstem, hypoplasia, and/or atrophy of cerebellar hemispheres and vermis, and severe hypomyelination with strikingly reduced white matter. While Patient 1 initially showed hypomyelination of cerebral white matter at 12 months of age, she showed only slightly reduced white matter at 4 years of age.
Conclusion: In-frame *SPTAN1* mutations could cause distinct clinical conditions of West syndrome with severe cerebral hypomyelination and developmental delay. In-frame *SPTAN1* mutations cause more severe phenotypes than deletion, suggesting the dominant negative effects of the mutations.

020
Re-evaluation of ketogenic diet therapy for refractory epilepsy in childhood Tokyo women's medical university's experience over 40 years
OGUNI H, OGUNI M, ITO S, ITO Y, OSAWA M
Dept of Pediatrics, Tokyo Women's Medical University, Tokyo, Japan

Purpose: We conducted a survey on the efficacy of ketogenic diet therapy (KD) for refractory childhood epilepsy in our hospital from 1968 to 2007.
Method: We retrospectively reviewed the effectiveness of KD therapy from 1968 to 2007.
Results: Fifty-one patients underwent classical 4:1 KD therapy following a 1-week fasting period between 1968 and 1973. At 1 year after KD therapy, 8 (15.7%) and 3 (6%) children showed excellent and good responses, respectively. Myoclonic and astatic seizures were most favorably responded. Between 1973 and 1977, 39 children underwent KD therapy. An excellent response was noted in 9 (23.1%) at 1 year, and in 10 (25.6%) at 6 years after KD therapy, demonstrating that these effects persisted for a long time. From 1982 to 1984, 21 children were treated with MCT 2:1 KD therapy following a 4 day-fasting period, with excellent and good responses in 3 (14.3%) and 4 (19.0%) patients, respectively. From 1984 to 2007, 54 patients underwent the KD therapy. KD was introduced following a 2-day fasting period. As a result, 18.5 and 35% of them showed excellent and good improvements, respectively, at 1 year. Favorable responses were achieved in those with early Lennox-Gastaut syndrome, epilepsy with both focal and generalized seizures, Dravet syndrome, and idiopathic myoclonic-astatic epilepsy in this order of decrease.
Conclusion: Although various antiepileptic drugs as well as surgical interventions have been introduced in Japan, KD therapy has been utilized and shown effectiveness without a significant difference for over 40 years in our hospital.

021
Symptomatic focal epilepsy (SFE) sharing the clinico-electrical characteristics with Panayiotopoulos syndrome (PS)
HIRANO Y, OGUNI H, HIRASAWA K, OSAWA M
Department of Pediatrics, Tokyo Women's Medical University, Tokyo, Japan

Purpose: PS is an idiopathic focal epilepsy, characterized by prolonged autonomic seizures and shifting and/or multifocal epileptic EEG foci with an occipital predominance. We herein report 8 children with SFE, sharing clinico-electrical characteristics with PS.
Method: The subjects were 8 patients (boys=3, girls=5) with cerebral palsy (CP) and mental retardation (MR), who had prolonged autonomic seizures and multifocal epileptic EEG foci, and were followed up for longer than 2 years in our hospital. We retrospectively analyzed their medical records, EEGs, and treatment response.

PLATFORM SESSION ABSTRACTS

Results: There was a history of perinatal asphyxia in 6 and surgical complications in 2 cases. Their seizure onset was 39±23 months, characterized by ictal vomiting, followed by secondary GTCS (n=5) and status epilepticus (n=6). Four of them experienced more than 10 seizure recurrences. The evolutional EEG changes showed that multifocal epileptic EEG foci with frontopolar and parieto-occipital predominance improved with increasing age. The seizures initially tended to be pharmaco-resistant, but all were controlled at 46.5±26.7 months from the onset.
Conclusion: Young children with CP and MR tend to develop status epilepticus and multifocal epileptic EEG abnormality, which have been considered to be consequence of acquired brain damage. However, the close resemblance of the clinical and EEG features with PS except for the recurrences of the seizures in all 8 children suggests that a combination of both acquired brain damage and genetic PS susceptibility prevalent in this age-group causes SFE, whose epileptogenesis may subside with increasing age.

022
A five-year review of childhood deaths due to SUDEP in Queensland
CLARK D[1], RINEY K[2]
[1]Royal Children's Hospital, Melbourne, Australia, [2]Mater Children's Hospital, Brisbane, Australia

Purpose: The purpose of this study was to determine the number of child deaths from sudden unexplained death in epilepsy (SUDEP) in Queensland and to examine documentation of SUDEP on death certificates and coronial post-mortem records.
Method: Data on deaths of children with epilepsy was reviewed over a 5-year period from three sources: death certificate data, Queensland Coronial post-mortem records and data from the Queensland Children's Commissioner's Child Death Review. Data was extracted on potential child SUDEP deaths from each source and the recording of SUDEP on death certificates and coronial post-mortem reports was noted.
Results: Child Death Review data documented that a total of 61 children with epilepsy died in the study period. In 21/61, the death was sudden, in the majority (15/21) the child was found dead in the morning. Nineteen sudden deaths were reported to the Coroner, however only 35% had full post-mortem investigation sufficient to definitively establish SUDEP as the cause of death. Despite most of the 21 sudden deaths being consistent with SUDEP, the term SUDEP was not recorded on any death certificate and was listed as the cause of death on post-mortem reports in only 5/21 cases. SUDEP in childhood in Queensland could only be estimated at 0.4 deaths /1000 children with epilepsy / year (CI 0.2-3.5).
Conclusion: SUDEP in childhood is inadequately recorded on death certificates, inadequately investigated at post-mortem and inadequately reported on post-mortem reports in Queensland.

023
Intractability of chronic non-idiopathic partial epilepsies: how many antiepileptic drugs might be tried?
SUGAI K, NAKAGAWA E, KOMAKI H, SAITO Y, SAKUMA H, SASAKI M
National Center of Neurology and Psychiatry Hospital, Kodaira, Tokyo, Japan

Purpose: For newly diagnosed epilepsies, two-drug definition for intractability (Kwan P, 2000) has been widely accepted. How about intractability for chronic epilepsies? We studied intractability for chronic non-idiopathic partial epilepsies (NIPE).
Method: 218 referral cases of chronic NIPE, aged 1 to 38 years, mainly < 18 years, which had not responded to one or more antiepileptic drugs (AEDs) prior to referral, were treated with other AEDs over one year by the author (SK). They consisted of frontal lobe epilepsy, 182 cases, temporal lobe epilepsy, 26 cases, and parietal or occipital lobe epilepsy, 10 cases. New AEDs were selected based on the precise seizure symptoms, and added to or switched to the previous AEDs.
Results: At the last evaluation, one year or more seizure-freedom was obtained in 135 cases (62%). Seizure-freedom was obtained in 26 cases by the 2nd AED, 40 cases by the 3rd AED, 28 cases by the 4th AED, 15 cases by the 5th AED, 10 cases by the 6th AED, 13 cases by the 7th AED, 1 cases by the 8th AED, 2 case by the 9th AED. Cumulative seizure-free rate was 50% of the subjects by 2nd to 5th AED.

PLATFORM SESSION ABSTRACTS

Conclusion: Two-drug definition of intractability is not applicable to chronic NIPE in children or young adults, because it has developed for newly diagnosed epilepsies consisted of all kinds in adolescents and adults including the elderly. Up to 5th AED should be tried for chronic NIPE in children or young adults.

Surgery and neuroimaging
Saturday 23rd October
15:00-16:00
Room 104

024
Effect of anterior temporal lobectomy (ATL) on sexuality in male patients with refractory temporal lobe epilepsy-hippocampal sclerosis (TLE-HS): a case control study
RAMESHA KN, ASHALATHA R, SEBASTIAN P, SARMA PS, UNNIKRISHNAN JP, RADHAKRISHNAN K
Sree Chitra Tirunal Institute for Medical Sciences and Technology, Trivandrum, India

Purpose: To find out the change in sexuality after ATL in males with MTLE-HS in comparison to their pre-operative state and age, sex, religion and socio-economic status matched controls, and to know any factors which can predict the outcome.
Method: A consecutive 50 patients with a healthy living partner and with a minimum one-year postoperative follow-up were taken as cases. Both cases and controls (50 each) were administered a questionnaire (38 questions) regarding their sexual desire and satisfaction, sexual anxiety, self esteem and sexual depression, general self-esteem, and general anxiety and depression. Mann Witney test, Wilcoxon signed ranks test and spearman's rank correlation test were used for statistical analysis.
Results: Mean age of cases at evaluation was 41±6years and that of controls were 39±6years. 78% patients had good seizure outcome (Engel class I) after a median follow-up of five years. All aspects of sexuality studied were significantly better in controls than cases before surgery (p=≤0.001). Majority of the sexual aspects improved with surgery, which correlated with the seizure outcome (p=0.021), number of AEDs (p=0.002), IEDs in postoperative EEGs (p=0.016), general self esteem (p=0.002) and duration of follow up after surgery (p=0.05). The side of surgery and duration of epilepsy before surgery did not predict the sexual outcome.
Conclusion: Sexual dysfunction is significantly higher in patients with TLE, both before and after surgery than controls. Majority of the sexual function improves with ATL, which positively correlate with their seizure outcome, monotherapy, general self-esteem and absence of IEDs in the postoperative EEGs.

025
Limitations of ictal scalp EEG and SPECT in seizure localization in tuberous sclerosis with epileptic spasms
MOHAMED AR[1], FREEMAN JL[1], BAILEY CA[1], MAIXNER WJ[1], HARVEY AS[1,2,3]
[1]Royal Children's Hospital, Melbourne, Australia, [2]Murdoch Children's Research Institute, Melbourne, Australia, [3]Department of Paediatrics, University of Melbourne, Melbourne, Australia

Purpose: Epilepsy surgery in tuberous sclerosis (TS) requires localization of the epileptogenic tuber(s), this being difficult in children with epileptic spasms (ES). We assessed the accuracy of seizure localization with scalp-EEG (s-EEG) and SPECT in children with TS and ES by comparison with intracranial-EEG (i-EEG).
Method: Review of ictal s-EEG, SPECT (subtraction ictal-interictal) and i-EEG in 9 consecutive children with TS, refractory epilepsy and ES who underwent subdural EEG monitoring.

PLATFORM SESSION ABSTRACTS

Results: 16 electroclinically distinct seizures were recorded intracranially in the 9 children, 9 seizures were associated with ES. 10 seizures were focal and localized to a solitary tuber, 3 were focal but unlocalized and 3 were ES only. Ictal rhythms in the 10 localized seizures consisted of low-voltage fast activity involving only 1-4 subdural electrode contacts and lasting only 10-60 (median 25) secs; evolution to periodic spike-waves and gamma bursts at these and remote electrodes occurred in 7 seizures. Pre-operative s-EEG showed ictal rhythms in the region of the epileptogenic tuber in only 4/10 intracranially localized seizures. Pre-operative ictal SPECT showed hyperperfusion in the region of the epileptogenic tuber in only 2/10 intracranially localized seizures. 7/10 seizures were associated with significant contralateral or bilateral ictal s-EEG or SPECT changes.
Conclusion: In children with TS and ES, i-EEG shows brief, low-voltage, focal seizures referable to solitary tubers, not detectable with ictal s-EEG or SPECT in the majority. Non-invasive, pre-surgical work-up is likely to be non- or falsely localising, such that i-EEG with wide sampling should be strongly considered.

026
MRI-guided stereotactic radiofrequency thermocoagulation for pediatric intractable epilepsy patients with hypothalamic hamartoma
MURAKAMI H, KAMEYAMA S, MASUDA H
Department of Neurosurgery, Epilepsy Center, Nishi-Niigata Chuo National Hospital, Niigata, Japan

Purpose: To develop a novel method for treating intractable epileptic hypothalamic hamartoma (HH) which is characterized by gelastic seizures (GS) using stereotactic radiofrequency thermocoagulation (SRT). Direct surgery and radiotherapy have not achieved decisive results.
Method: Forty consecutive patients with HH underwent SRT in our hospital between 1997 and 2010. Twenty-six patients (65%) were under 16 years of age (mean age, 8.9 years; range, 2-15 years). All patients displayed daily GS, with other types of seizures in 23 patients (88%), precocious puberty in 6 (23%), behavioral disorder in 10 (38%), and mental retardation in 12 (46%). Mean maximum diameter of the HH was 20 mm (range, 8-50 mm). SRT (74 °C, 60 s) was used to make multiple 5-mm spherical lesions within the HH through multiple tracks via the unilateral frontal route. Ictal single-photon emission computed tomography was useful for determining laterality of propagation from HH and for deciding the approach side.
Results: Thirty-two SRT surgeries were performed, using 1-9 tracks (mean, 4) and making 1-18 lesions (mean, 8) without adaptive limitations regardless of the size or subtype. No permanent complications persisted after SRT. Observations were continued for >2 years after SRT in 21 of 26 patients. GS disappeared in all patients. Complete freedom from seizures and behavioral disorder was achieved in 24 patients (92%), along with intellectual improvement.
Conclusion: SRT is minimally invasive and appears effective, without adaptive limitations compared to other surgical procedures. This procedure should be considered before adulthood, to minimize the risk of residual seizures.

027
Medically refractory epilepsy associated with posterior cortex gliosis: clinical, electrophysiologic, imaging and pathologic characteristics and seizure outcome
SINGH A[1], SAINI J[2], RATHORE C[3], RADHAKRISHNAN A[3], RADHAKRISHNAN VV[3], CHANDRASEKARAN K[3], RADHAKRISHNAN K[3]
[1]Fortis Hospital, Noida, India, [2]NIMHANS, Bangalore, India, [3]SCTIMST, Trivandrum, India

Objective: To investigate the electro-clinical, imaging, pathologic characteristics and post- operative outcome of patients with posterior cortex gliosis associated medically refractory epilepsy.
Method: Study cohort comprised of all patients with posterior cortex gliosis who underwent VEEG monitoring at our centre from 2005 till June 2009. All underwent standard pre-surgical evaluation including long-term video-EEG and high resolution MRI, and evaluation with SPECT, PET, f-MRI, cortical stimulation-mapping and invasive monitoring when indicated. Some of the patients were operated for posterior cortex gliois with >1 year postoperative follow-up.

PLATFORM SESSION ABSTRACTS

Results: The cohort comprised of 117 patients (86 males, 31 females) with a mean age of 17.6 years and mean duration of epilepsy was 11.15 years. Nineteen patients underwent focal resective surgery and 98 patients were in the non surgical group. In the non surgical group the mean age was 17.1 years and mean duration of epilepsy was 11 years. Radiologically, 25 patients had unilateral lesion and 23 had ulegyric cortex. In the surgical group, mean age was 19.6 years and mean duration of epilepsy was 11.5 years. All of them had focal epilepsy. Sixteen patients had unilateral gliosis and 4 had ulegyric cortex. Sixteen patients (80%) were seizure free in the surgery group at one year follow up in comparison to 20 (20%) patients in the medically treated group (p=0.0004).
Conclusion: This study is the largest reported till date on epilepsy-associated with posterior cortex gliosis. The observations indicate that surgery should be offered to patients who have good electroclinical concordant data.

Social and economic issues
Saturday 23rd October
15:00-16:00
Room 106

028
The role of optimism, hope, and family support on quality of life in epilepsy
PRIMARDI A[1], HADJAM NR[2]
[1]Faculty of Psychology, Gunadarma University, Jakarta, DKI Jakarta, Indonesia, [2]Faculty of Psychology, Gadjah Mada University, Yogyakarta, DIY, Indonesia

Purpose: Persons with chronic disease such as epilepsy, where a cure is not attainable and therapy may be prolonged, quality of life (QOL) has come to be seen as an important health care outcome. The purpose of this study is to determine the interaction of optimism, hope, and family support as predictors of QOL in epilepsy case.
Method: People With Epilepsy (PWE) were recruited from Epileptic Clinic of Department of Neurology Cipto Mangunkusumo Hospital, Jakarta. The 62 patients were taken as subject in 2009. QOL in epilepsy, optimism, hope, and family support were diagnosed by using the questionnaire, and also were explored using interviews with 9 PWE.
Results: The result of regression analysis showing that optimism, hope, and family support, can become predictor of QOL in epilepsy ($R = 0{,}436$; $p < 0{,}01$). As assessed by partial correlation analysis, there are positive relationship most significantly between optimism and QOL ($r = 0{,}323$; $p < 0{,}01$), positive relationship significantly between hope and QOL ($r = 0{,}275$; $p < 0{,}05$), and positive relationship insignificantly between family support and QOL ($r = 0{,}006$; $p > 0{,}05$). In the result of interview, PWE reported their experience in medical treatment and epilepsy surgery, aura and seizure, the impact of epilepsy in daily life, family and social support, culture & social stigma, and psychological problem such as shyness, anxiety, self-confidence.
Conclusion: The role in improving QOL may not only focus heavily on the epilepsy physically, but also considering social and psychological impact of epilepsy.

029
The lived experience of initial symptoms of epileptic seizures: an Australian study
PINIKAHANA J
The Epilepsy Foundation of Victoria, Australia, Camberwell, Victoria, Australia

Purpose: To document self-perceived warning signs, initial symptoms, and triggers of epileptic seizures and techniques to control seizures for people with epilepsy, and to establish patterns in these self-reported experiences of epilepsy in relation to age, gender and seizure type.
Method: Participants were sourced from the Epilepsy Foundation of Victoria's Social Research

PLATFORM SESSION ABSTRACTS

Participant Register. Of the 338 people with epilepsy who had registered interest in participating in social research, 225 completed a self-report survey questionnaire (66% response rate) that contained information regarding demographic characteristics, living with epilepsy, and self-perceived warning signs, initial symptoms, triggers of seizures and techniques to control seizures.
Results: Of 225 respondents, 195 (86.6%) experienced at least one initial symptom prior to a seizure and 202 (89.8%) experienced at least one seizure trigger. Gender analysis of triggers revealed that females differed from males regarding seizures triggered by low blood sugar, dieting, fasting, touch and female specific triggers(menstruation, pregnancy, and ovulation). Respondents reported tiredness as the most frequent trigger (65.3%), followed by stress (64%) and sleep deprivation (55.1%). Many respondents (63.6%) reported that they could predict seizure occurrence, with 91(40.4%) also indicating that family members could predict seizure occurrence. A total of 157(69.8%) respondents had tried at least one of 12 possible seizure avoidance techniques, with resting and medication the most frequently used.
Conclusion: Finding that respondents were able to recognise warning signs, initial symptoms and triggers of seizures provides scope for developing interventions, such as promoting avoidance of high risk triggers of epileptic seizures.

030
The impact of anxiety and depression on health related quality of life of young people with epilepsy
CLARKE A[1,2], CRITCHLEY C[1]
[1]Swinburne University of Technology, Hawthorn, Australia, [2]Epilepsy ACT, Canberra, Australia

Purpose: Available data indicates that young people with epilepsy experience higher levels of anxiety and depression and lower levels of health related quality of life than the normal population. The purpose of this study was to investigate the relationship between anxiety and depression and Health Related Quality of Life (HRQOL) in young people with epilepsy.
Method: A total of 114 young people (35.1% male), ranging in age from 10 to 24 years with a mean age of 17.92 years (SD = 3.90), completed a paper (60.5%) or Internet survey (39.5%) that included demographic, medical and psychosocial questions. The survey included: demographic and medical history questionnaires, Quality of Life in Epilepsy for Adolescents Scale (QOLIE-AD-48), the Family Assessment Device (FAD-GF), Adolescent Coping Scale (ACS-SF) and Hospital and Anxiety Scale (HADS). The young people were mainly recruited from community support groups in each state of Australia and New Zealand, neurologists who specialize in treating epilepsy, and Internet support groups.
Results: Using multiple regression, demographic, medical and psychological variables were used to predict quality of life. Together the variables explained 47.8% of the variance, with only higher depression (Beta = -3.06) and anxiety scores (Beta = -2.93) significantly predicting lower HRQOL. Gender, age, duration and severity of illness, family functioning, and coping style were not significantly associated with HRQOL.
Conclusion: In addition to seizure management, clinicians can promote better quality of life for young people with epilepsy by actively screening and managing anxiety and depression symptoms.

031
Economic burden of epilepsy in a developing country: a retrospective cost analysis in China
HONG Z, WU X, YANG T, ZHANG Q, ZHOU D
West China Hospital, Sichuan University, Chengdu, China

Purpose: To study the cost of epilepsy in China, and therefore provide essential information on the burden of the disease to individuals and society.
Method: A cost-of-illness study was performed on a retrospective cohort of medically treated patients. Data on clinical characteristics, utilization of sources, and costs were collected from 289 patients in a standardized format.

PLATFORM SESSION ABSTRACTS

Results: Direct medical care costs was Chinese yuan, renminbi (RMB) 2,529 (USD 372) per year per patient, of which antiepileptic drugs (RMB 1,651 or USD 243) accounted for the major costly component. Nonmedical direct costs were much less than direct health care costs, averaging approximately RMB 756 (USD 111). Costs due to loss of productivity averaged approximately RMB 1,968 (USD 289) per patient per year. Taken together, the overall mean annual cost for epilepsy per patient in our series was approximately RMB 5,253 (USD 773), and these costs accounted for more than half of the mean annual income. Total cost was significantly associated with the disease severity and different responses to drug treatment. In addition, new antiepileptic drugs and the number of drugs taken were also closely related with the drug cost.
Conclusion: The results indicate that the economic burden of epilepsy to both Chinese patients and the nation is heavy and the composition proportions of the costs in China have many similar features and some noteworthy differences with that of other countries.

032
Factors associated with quality of life in people with epilepsy and the variations between men and women, younger and older people
YU P, HONG Z, YUE L, WU X, ZHU G
Institute of Neurology, Huashan Hospital, Fudan University, Shanghai, China

Purpose: To determine factors associated with quality of life (QOL) in people with epilepsy and the variations between men and women, younger and older people.
Method: Patients were interviewed using Quality of Life in Epilepsy-31 (QOLIE-31), Side Effect Profile (SEP), Self-rating Anxiety Scale (SAS) and Hamilton Depression Scale (HAMD). Medical as well as socio-demographic data was assembled from client files. Multivariate linear regressive analyses determined the set of best predictors of composite QOLIE-31 score.
Results: Two hundred and four patients (160 younger, 49 older; 125 men, 79 women) were included in the analysis. No statistical difference was revealed in QOLIE-31 overall score either between younger and older patients or between men and women. Among all patients, regressive analyses revealed that SEP (β=-0.395, p =0.000) and SAS (β=-0.152, p =0.016) were two strong predictors of QOLIE-31 overall score. Grouped by gender, among men, epilepsy duration (β=-0.165, p =0.028) and seizure frequency(β=-0.284, p =0.001) respectively associated with QOLIE-31 overall score and 'social function' score. Grouped by ages, seizure frequency (β=-0.284, p =0.000) and education level (β=0.203, p =0.005) predicted QOLIE-31 "social function" score only among younger patients; among older patients, a significant association were found between the number of AEDs and QOLIE-31 overall score (β=-0.363, p =0.004) .
Conclusion: Side effects of AEDs and number of AEDs exerted greater effect on QOL in women and older patients, while seizure-related variables such as epilepsy duration and seizure frequency infuence QOL only among men and younger patients.

General epileptology
Saturday 23rd October
15:00-16:00
Room 109

033
Prevalence of acute symptomatic seizures in community based study from Northern India
GOEL D, SAXENA V
HIHT University, Dehradun, India

Purpose: There are 22 Epilepsy epidemiological studies in India. Prevalence data of epilepsy is contaminated by acute symptomatic seizures due to neurocysticercosis (NCC). Very few epidemiological studies could estimate prevalence of NCC due to lack of investigation tools at community level. We have done this community based study to find out prevalence of acute symptomatic seizures and epilepsies in one community in India.

PLATFORM SESSION ABSTRACTS

Method: This community based cross-sectional study was done in Chakrata block of northern India. Total 36 villages with 14086 population were selected on a random basis from each population scale range so that, it would not affect the sampling size. As first step, door-to-door survey was done to find out cases of febrile convulsion and afebrile seizures in this population. All cases with afebrile seizures were seen be neurologist to find out true seizures cases in second step. Finally all true seizure cases were brought to our hospital for investigations.
Results: among study population 32 cases had history of febrile seizure with prevalence of 2.27 per 1000 people. Total 141 cases had afebrile seizures with prevalence of 10 per 1000. Prevalence rate of acute symptomatic seizures due to NCC was 2.5 per 1000. Finally prevalence of symptomatic, idiopathic and cryptogenic epilepsy was respectively 2.55, 1.56, and 4.1 per 1000 people.
Conclusion: Epidemiological data on epilepsy must be reviewed in view of our findings as there are high chances of over estimation of epilepsy cases in those countries where NCC is endemic.

034

International variability in use of EEG when withdrawing anti-epileptic drugs (AEDs)

BERGIN PS[1], BEGHI E[2], LEGROS B[3], RICHARDSON M[4], TRIPATHI M[5], D'SOUZA W[6], BURNEO J[7], RAYMOND AA[8], MOGAL Z[9], EPINET STUDY GROUP
[1]Auckland City Hospital, Auckland, New Zealand, [2]Mario Negri Institute, Milano, Italy, [3]Université Libre de Bruxelles, Bruxelles, Belgium, [4]Kings College Hospital, London, United Kingdom, [5]All India Institute of Medical Sciences, New Delhi, India, [6]St Vincent's Hospital, Melbourne, Australia, [7]Epilepsy Programme at the University of Western Ontario, London, Canada, [8]National University of Malaysia Medical Centre, Kuala Lumpur, Malaysia, [9]National Epilepsy Centre, Karachi, Pakistan

Purpose: To assess neurologists' use of EEG when withdrawing AEDs in seizure-free patients.
Method: An Internet-based questionnaire was circulated to adult and paediatric neurologists in New Zealand, Australia, India, Pakistan, Malaysia, Italy, Great Britain, Belgium and Canada. We asked how rapidly neurologists withdraw AEDs from seizure-free patients, how frequently they use EEG in this setting, and what factors determine its use.
Results: As of 3 July 2010, 240 responses had been received. 28% of respondents withdraw AEDs over 1 - 3 months, 30% over 3 - 6 months, and 20 % over a longer period. 53% of respondents always get an EEG when withdrawing AEDs. 15% never get an EEG when discontinuing one of several AEDs, and 10% never get an EEG when discontinuing a patient's only AED. There was a marked difference in practice between different countries. More than 75% of respondents from Pakistan (n=8), Canada (n=13), Belgium (n=38) and Italy (n=62) always get an EEG when discontinuing a patient's only AED, while fewer than 20% of respondents in New Zealand (n=29), Malaysia (n=10) or Britain (n=35) do so. When withdrawing one of several AEDs, 48% of New Zealand respondents never obtain an EEG, while fewer than 5% of respondents in India (n=27), Pakistan, Canada, Belgium and Italy never obtain an EEG.
Conclusion: There is marked variability in use of EEG in patients who are discontinuing treatment, and in the rate of AED withdrawal. Practice differs greatly both within and between countries. A multicentre trial should be considered.

035

The Sydney Epilepsy Incidence Study to Measure Illness Consequences (SEISMIC): rationale, design and preliminary results

ANDERSON C[1], BLEASEL A[2], GARDNER G[3], GHOUGASSIAN D[4], GLOZIER N[5], HACKETT M[1], HASSAN B[1], IRELAND C[3], JAN S[1], LAWSON J[6], MARTINIUK A[1], MOHAMED A[7], SAMPAIO H[6], SOMERVILLE E[4], TODD L[3], SEISMIC INVESTIGATORS
[1]The George Institute for International Health, Sydney, New South Wales, Australia, [2]Westmead Hospital, Sydney, NSW, Australia, [3]Epilepsy Action Australia, Sydney, Australia, [4]Prince of Wales Hospital, Sydney, Australia, [5]University of Sydney, Sydney, Australia, [6]Sydney Children's Hospital, Randwick, Sydney, Australia, [7]Royal Prince Alfred Hospital, Sydney, Australia

Purpose: There are limited population-based incidence and outcome data on the burden of epilepsy, worldwide.

PLATFORM SESSION ABSTRACTS

Method: The Sydney Epilepsy Incidence Study to Measure Illness Consequences (SEISMIC) is a new population-based epidemiological incidence and outcome study funded of epilepsy funded by Australian government grants (NHMRC and ARC). Following a pilot phase to establish the feasibility of case ascertainment and outcome across multiple recruitment sites from September 2008, the main phase commenced on 1 June 2010. The aims are to include all newly diagnosed cases of epilepsy who are resident of the Eastern Zone of the Sydney South West Area Health Service over a 2-year period, and to follow them up to assess a broad range of clinical and psycho-socio-economic outcomes over several years.
Results: During the pilot phase, 1000 potential patients were screened to identify 37 people with newly diagnosed epilepsy, 37 with a first unprovoked seizure, 376 with known epilepsy; 15 people declined participation. The common reasons for exclusion were: 166 provoked seizures (drug and alcohol, head injury, hypoglycaemia, febrile convulsions, other), 257 not seizures (stroke/TIA, convulsive syncope, pseudo-seizures), 46 unknown diagnosis, 36 out of recruitment area.
Conclusion: The pilot phase demonstrated the feasibility of recruitment methods for SEISMIC, an important study of direct relevance to health care and service planning and policy development in Australia.

036
Behavioural and electrophysiological evidence of (central) auditory processing disorder in patients with refractory mesial temporal lobe epilepsy and mesial temporal lobe sclerosis
ARAVIND KUMAR R, SHIVASHANKAR N, SATISHCHANDRA P, SINHA S, SUBBAKRISHNA DK
National Institute of Mental Health and Neurosciences, Bangalore, India

Purpose: To study the central auditory processing disorder (C) APD in patients with refractory mesial TLE secondary to mesial temporal sclerosis (MTS).
Method: In this prospective study, ten patients with right MTS (M:F=6:4) and 14 with left MTS (M:F=12:2), and 19 healthy controls (M:F=12:7) were administered auditory behavioral tests viz., gaps-in-noise test (gap detection threshold-GDT and total percentage score-TPS), duration pattern test (hum and verbal)), dichotic digits test (free recall-FR and directed recall-DR) and the electrophysiological test viz., P300 (tonal and syllabic).
Results: The age at onset and duration of seizures were 13.2±6.57 and 8.46±6.66 years and 15.6±5.94 and 15.03±7.19 years for right and left MTS groups respectively. Statistically significant difference was evident when RMTS and LMTS groups were compared with normal control group on right and left TPS, right and left hum, right and left FR and, tonal and syllabic latency. Significant difference was observed for right GDT and right DR in RMTS, right and left verbal and left DR in LMTS groups. A significant difference was observed for tonal and syllabic latency in the LMTS group. There were no differences on any other parameters between the RMTS and LMTS groups. There was also no correlation between the age at onset or duration of seizures with any of the tested parameters.
Conclusion: The presence of (C) APD in this cohort warrants further research for better understanding of the underlying mechanisms and its impact.

037
Causes of deaths among people with convulsive epilepsy in rural West China: a prospective study
MU J, LIU L, ZHOU D
Sichuan University, Chengdu, China

Background: Epilepsy is a serious health problem with an increased risk of premature mortality. Only a few studies describe causes of death. Clarifying the risks of each cause of death (COD) may be an effective assistance to develop preventative measures to reduce premature mortality.

PLATFORM SESSION ABSTRACTS

Method: A prospective assessment of the management of convulsive forms of epilepsy at primary health level amongst a large cohort was carried out in rural west China from May 2005. Demographic data and putative causes of death during the follow-up period were recorded. The case fatality rate (CFR) , the proportional mortality ratios (PMRs) and standardized mortality ratios (SMRs) were estimated on the basis of the 2007 Chinese rural population.
Results: There were 106 reported deaths (70 males) among 3,568 people in the cohort. Case fatality rate was 2.97% during a median of 28 months' follow-up. The PMRs for injury (drowning), sudden unexpected death in epilepsy (SUDEP), status epilepticus were high. The SMR was 5.11 (95% CI 4.68-5.59), while men was 5.25 higher than women 4.64. Patients aged 5-39 years had striking high mortality ratios with SMRs exceeding 24. The risk of drowning was 87 times in convulsive epilepsy patients than general population.
Conclusion: In rural west China, risk for premature death is five to six times higher in people with convulsive epilepsy than in the general Chinese population. Furthermore, the risk in young people is higher than previously reported. Injury, especially drowning is the leading putative COD in patients with convulsive epilepsy in west rural China.

Basic science
Saturday 23rd October
15:00-16:00
Room 110

038
The GAERS Cav3.2 T-type calcium channel mutation affects the expression ratio of the channel splice variants
NG C[1], POWELL KL[1], TYSON JR[2], REID CA[1], CAIN SM[2], SNUTCH TP[2], O'BRIEN TJ[1]
[1]The University of Melbourne, Melbourne, Australia, [2]The University of British Columbia, Vancouver, Canada

The GAERS absence epilepsy rat model harbors a $Ca_v3.2$ T-type calcium channel missense mutation that co-segregates with seizure expression. This mutation (R1584P) induces a gain-of-function on $Ca_v3.2$ channels that is splice variant-specific, requiring the presence of exon 25 to manifest its effects. A shift in the ratio of two $Ca_v3.2$ splice variants was previously observed in epileptic GAERS thalamus, with an increase in the proportion of the $Ca_v3.2$(+25) variant. However, it is unknown whether this change in splice variant ratio is a consequence of the mutation or a reflection of the disease-process. Here, we demonstrate that GAERS and F2 rats (double-crossed between GAERS and non-epileptic controls) homozygous for the $Ca_v3.2$(R1584P) mutation have a higher $Ca_v3.2$(+25) to $Ca_v3.2$(-25) splice variant mRNA ratio, which is present in all brain regions and developmental ages examined. Furthermore, $Ca_v3.2$(+25/-25) mRNA ratio in the somatosensory and motor cortices of F2 rats was positively correlated with the number of seizures expressed. Importantly, this ratio shift was absent in another polygenic rat model (WAG/Rij) that lacks the $Ca_v3.2$(R1584P) mutation and remained unaffected when the influence of T-type calcium channel on neuronal function in dissociated cultures was removed by nickel application. Our results collectively argue that the $Ca_v3.2$(R1584P) mutation alters splice variant ratio in favour of the splice variant in which it has its gain of biophysical function effects by a genomic mechanism. Given that $Ca_v3.2$ mutations have been found in absence epilepsy patients, these findings may have clinical relevance for the pathophysiology of the human disease.

PLATFORM SESSION ABSTRACTS

039

The activation of mammalian target of rapamycin signaling pathway is related to the gliosis in hippocampal sclerosis in a mouse model of mesio-temporal lobe epilepsy

NITTA N, NOZAKI K
Department of Neurosurgery, Shiga University of Medical Science, Otsu, Japan

Purpose: The mammalian target of rapamycin (mTOR) signaling pathway has been reported to relate to astrocyte cell size regulation. In hippocampal sclerosis, marked hypertrophy of astrocytes is observed. We show that the mTOR signaling pathway is activated in astrocytes in hippocampal sclerosis in a mouse model of mesio-temporal lobe epilepsy and that the inhibition of mTOR signaling pathway results in reduced hypertrophy of astrocytes in hippocampal sclerosis, suggesting that mTOR should contribute to gliosis in hippocampal sclerosis.
Method: Stereotactic kainic acid (KA) injection into mouse hippocampus, which induces progressive reactive gliosis in hippocampus, was performed in two mice groups; one was treated with vehicle and the other was treated with an mTOR inhibitor, rapamycin. We performed immunohistochemistry for P-S6, which is in the downstream of mTOR, at various time points after KA injection, and GFAP at 3 weeks after KA injection.
Results: P-S6 increased in astrocytes in hippocampus after KA injection, indicating that mTOR signaling pathway was activated. Rapamycin reduced the hypertrophy of astrocyte in hippocampal sclerosis.
Conclusion: Our data show that activation of mTOR signaling pathway induces hypertrophy of astrocytes in hippocampus sclerosis, suggesting that mTOR signaling pathway is one of the key factors causing hippocampus sclerosis of mesio-temporal lobe epilepsy.

040

The modulatory effects of VEGF-C on NMDA receptor function in dysplasia cortex from an animal model of malformations of cortical development

ZHANG C-Q[1], SHU H-F[1], ZHANG B[2], AN N[1], ZHAO B-Y[1], SONG Y-C[1], LIU S-Y[1], ZHOU Z[2], YANG H[1]
[1]Department of Neurosurgery, Xinqiao Hospital, Third Military Medical University, Chongqing, China, [2]Institute of Molecular Medicine and State Key Laboratory of Biomembrane Engineering, Peking University, Beijing, China

Purpose: Malformations of cortical development (MCD) represents a well-recognized cause of intractable epilepsy, the exact mechanism underlying epileptogenesis of MCD has not yet been elucidated. Recently, several studies reveal VEGF-C, the most important lymphangiogenic factor, mediates multiple effects in central nerve system. As NMDA receptor (NMDAR)- mediated excitatory neurotransmission is closely related to expression of epileptic activity, our aim was to investigate the effects of VEGF-C on NMDAR function in dysplasia cortex from an animal model of malformations of cortical development.
Method: We employed a MCD rat model by in utero irradiation, and observed the expression of VEGF-C system in dysplasia cortex by immunostaining. In vitro slices, the VEGF-C modulation of NMDAR was examined using whole-cell recordings from malformed neurons (MNs) in dysplastic cortex and pyramidal neurons in control neocortex.
Results: Immmunostaining results showed a significant increase of VEGF-C and VEGFR-2 expression in MNs from MCD rats, and the level of VEGFR-3 was also slightly increased. In electrophysiological experiments, we analyzed evoked-EPSCs and found the current ratio of NMDAR/AMPAR was increased in MNs compared with pyramidal neurons from control, this effect mainly induced by the increase of NMDAR-mediated currents. Furthermore, We found that bath perfusion of VEGF-C (100 ng/mL) significantly increased whole-cell NMDAR currents in MNs of dysplastic cortex. The effect of VEGF-C was mainly blocked by application of the VEGFR-2 inhibitor (Ki8751).
Conclusion: Our study suggests that a synergistic action between VEGF-C and NMDAR exists in dysplasia cortex of MCD rats, which may involve in epileptogenesis of MCD.

PLATFORM SESSION ABSTRACTS

041

The effects of strontium on outward currents in the postsynaptic membrane

GARCIA PG[1], FRENCH C[1,2]

[1]University of Melbourne, Department of Medicine, Melbourne, Australia, [2]Royal Melbourne Hospital, Melbourne, Australia

Purpose: Strontium is a divalent cation that is a commonly used as a substitute for extracellular calcium to probe neurotransmitter release. It is generally assumed Sr only affects only pre-synaptic vesicular release. As transmembrane Ca flux affects several other processes including calcium-activated potassium conductances (ICaK), we have investigated equimolar Sr substitution on neuronal outward currents.
Method: Hippocampal slices from 3-7 week old Wistar rats were enzymatically treated and individual CA1 neurons (n=16) obtained by mechanical dissociation. Outward currents were recorded using whole-cell voltage-clamp with a KF based pipette solution in electrodes with resistances typically 1.5 megΩ. Sodium currents were blocked with tetrodotoxin and solutions applied via a 100 um diameter tube placed about 100 um from the cell, allowing solution exchange times of 1-2 s. Responses to depolarizing voltages commandswere recorded in the same neurons before and after Sr treatment.
Results: Sr substitution resulted in reversible reduction in amplitude of outward currents with kinetics and reversal potentials consistent with potassium conductances. Addition of the calcium antagonist cadmium to standard solution reduced total outward current amplitude with abolition of the Sr effect. It is therefore likely that Sr action is through calcium-activated potassium conductances.
Conclusion: Sr substitution for Ca in extracellular solution reduces outward current amplitude most likely through inhibition of calcium-activated potassium conductances. Care should be taken in interpretation of post-synaptic responses in the presence of Sr. Any therapeutic use of Sr should be done with vigilance, as ICaK is present in many tissues.

042

The modulation of sodium conductance by phenytoin is mediated by slow rather than fast inactivation processes

ZENG Z[1], FRENCH CR[2]

[1]Department of Medicine, University of Melbourne, Melbourne, Australia, [2]Neurosciences, Royal Melbourne Hospital, Melbourne, Australia

Introduction: Phenytoin (PHT) is a modulator of the voltage gated sodium channels (Nav), reducing the peak amplitude of evoked currents by sequestering channels into inactivated states and slowing their return from these states. These effects are probably sufficient to explain the efficacy of PHT as an anti-epileptic drug (AED). It has generally been assumed that the inactivated states favoured by PHT exposure are those corresponding to the fast inactivation cytoplasmic gate, but there are other slower inactivation processes that may be involved. To clarify this we studied the effects of 50 uM PHT on both fast and slow inactivation processes.
Method: Hippocampal slices from 3-7 week old Wistar rats were enzymatically treated and isolated CA1 neurons (n=36) obtained by mechanical dissociation. Inward currents were recorded using whole-cell voltage-clamp with a CsF based pipette solution in electrodes with resistances typically of 1.5 megΩ. Steady-state inactivation (h_{inf}) was measured with different length conditioning pulses, and dynamic transitions between closed states measured with double pulse protocols with timescales over several orders of magnitude (1 to 10000 ms). Persistent (I_{Nap}) was also measured, and in some experiments pronase was applied cytoplasmically to remove fast inactivation.
Results: PHT hyperpolarized the midpoint of hinf curves, moreso with longer pre-pulses (500 ms). Macroscopic inactivation by depolarisations as well as INap were unaffected by PHT. Transitions between closed and closed inactivated states were slowed by PHT, but predominantly affected longer components rather than fast interconversions attributable to fast inactivation. Entry into slow inactivation was accelerated and recovery slowed by PHT. PHT had no effect of recovery of INa from fast inactivation, and exerted proportionally the same reduction in I_{Na} amplitude after fast inactivation removal.
Conclusion: PHT appears to modulate sodium channels mainly through slow or intermediate duration inactivation processes rather than fast inactivation as is commonly assumed.

PLATFORM SESSION ABSTRACTS

Translational research
Saturday 23rd October
17:30-18:30
Room 104

043
The anticonvulsive and antioxidant effects of curcumin on pilocarpine-induced seizure in rats
DU P[1], TANG H[1], LI X[1], LIN H[1], PENG W[1], MA Y[1], FAN W[1], WANG X[2]
[1]Department of Neurology, Zhongshan Hospital, Fudan University, Shanghai, China, [2]Department of Neurology, Zhongshan Hospital, The Institutes of Brain Science, State Key Laboratory of Medical Neurobiology, Fudan University, Shanghai, China

Purpose: To investigate the anticonvulsive and antioxidant effect of curcumin on pilocarpine-induced seizure in rats.
Method: Pilocarpine-induced seizure in rats was used as a model. Curcumin was intraperitoneally injected to rats 30 minutes before the administration of pilocarpine (300 mg/kg, i.p) at doses of 30, 100 and 300 mg/kg. Latency to the first episode of limbic seizures or status epilepticus, and the mean seizure severity score were quantatively analyzed. The activities of the hippocampal nitric oxide synthase (NOS), lactate dehydrogenase (LDH) and superoxide dismutase (SOD) were assayed 24 hours after the administration of pilocarpine. In addition, the contents of the hippocampal glutathione (GSH) and malonaldehyde (MDA) were measured in the same time.
Results: Curcumin at a dose of 100 or 300 mg/kg significantly increased the seizure threshold compared to vehicle-injected rats ($p < 0.01$). In pilocarpine-induced seizure models, the levels of MDA, NOS, and LDH were significantly increased ($p < 0.01$), while the levels of SOD and GSH were decreased ($p < 0.01$). Interestingly, curcumin also could reverse the effects of pilocarpine-induced seizure on NOS, LDH, GSH, and SOD ($p < 0.01$), but not MDA ($p > 0.05$).
Conclusion: Our findings suggest that curcumin can inhibit pilocarpine-induced seizure in rats, and such anticonvulsant properties may be possibly due to the antioxidant effects of curcumin.

044
Action potential dysfunction in pyramidal neurons of a Scn1b mutant mouse model of Dravet syndrome
REID C[1], HILL E[2], THOMAS EA[2], RICHARDS K[2], WIMMER V[2], LERCHE H[3], SCHEFFER I[4], BERKOVIC S[4], PETROU S[2]
[1]University of Melbourne, Parkville, Australia, [2]Florey Neuroscience Institute, Parkville, Australia, [3]Hertie Institute of Clinical Brain Research, University of Ulm, Germany, [4]University of Melbourne, Melbourne, Australia

Purpose: Dravet syndrome is a neurological condition that falls at the most severe end of the spectrum of Genetic Epilepsy with Febrile Seizure Plus (GEFS+). *SCN1A* mutations are the most common cause of GEFS+ and account for >70% of patients with Dravet syndrome. *SCN1B* is also an important cause of GEFS+ and recently a homozygous *SCN1B* mutation was reported in a patient with Dravet syndrome. To further elucidate the pathogenesis of *SCN1B* mutations, we investigated a mouse model homozygous for the most common GEFS+ *SCN1B* mutation, C121W.
Method: EEG recordings were made from epidural electrodes. Thermal seizures were induced by a hot stream of air. Acute slices were cut from P14 animals and whole-cell recordings made in current clamp mode in the CA1.
Results: The phenotype of the homozygous mice resembled Dravet syndrome. This included spontaneous seizures, thermally triggered tonic-clonic seizures, abnormal gait, EEG abnormalities and premature death. Whole-cell electrophysiological measurements in our *Scn1b* model revealed an action potential (AP) 'collapse' at large current injections only in pyramidal neurons and not in interneurons, in marked contrast to *Scn1a* based Dravet syndrome models. Broadening of APs occurred during this collapse in pyramidal neurons. Computer modelling predicted a 25% increase in pre-synaptic Ca^{2+} influx for the broadened APs providing a plausible mechanism of increased network excitability in homozygous animals.

PLATFORM SESSION ABSTRACTS

Conclusion: Comparative analysis with the Scn1a model suggests that distinct genetic mechanisms converge at a network level to produce the same epilepsy syndrome.

045

Pharmacogenomics of carbamazepine: from kinetics to dynamics

CHANG W-L[1], LIOU H-H[2], HUNG C-C[1]

[1]Department of Pharmacy, College of Pharmacy, China Medical University, Taichung, Taiwan, Republic of China, [2]Pharmacology and Neurology, National Taiwan University Hospital and College of Medicine, National Taiwan University, Taipei, Taiwan, Republic of China

Purpose: To identify the influence of genetic polymorphisms on the carbamazepine serum concentration and daily dosage for individualize the medication for seizure control.
Method: Epileptic patients treated with carbamazepine were recruited from the department of Neurology of National Taiwan University Hospital (NTUH), and the daily dosage and serum level of carbamazepine were recorded. After signed informed consent, 10 ml blood was drawn to extract genomic DNA. The polymerase chain reaction (PCR)-restriction fragment length polymorphism (RFLP) and Real-Time PCR were applied to analyze the 12 single nucleotide polymorphisms in the candidate genes: SCN1A, SCN2A, EPHX1, ABCB1and UGT2B7.
Results: There were 157 patients enrolled in the present study. The mean dose of carbamazepine was 836±24 mg and the mean serum concentration was 7.63±0.19 mg/ml. Among these patients, there were 51 patients (32.5%) under carbamazepine monotherapy. The results demonstrated that the SCN1A c.3184A>G, EPHX1 c.416A>G, UGT2B7 c.735A>G, -161C>T, c.802C>T, -842A>G polymorphisms were associated with lower effective doses of the carbamazepine. On the other hand, the effective doses of the carbamazepine were higher in patients with SCN1A IV5-91G>A, EPHX1 c.337T>C and ABCB1 c.2677C>A/T polymorphisms. Moreover, patients with CT or TA genotype in ABCB1 c.2677C>A/T, AA genotype in EPHX1 c.416A>G and TT genotype in c.337T>C were with higher carbamazepine serum concentrations.
Conclusion: The present study suggests that polymorphisms in genes encoding for pharmacodynamic targets, disposition proteins and metabolizing enzymes are associated with carbamazepine treatment outcome.

046

The enduring effects of early-life stress on limbic epileptogenesis in rodents are mediated by seizure-induced corticosterone release

KOE AS[1], JONES NC[1], SALZBERG MR[1,2], O'BRIEN TJ[1,3]

[1]Department of Medicine (RMH), University of Melbourne, Melbourne, Australia, [2]Department of Psychiatry, St Vincent's Hospital, Melbourne, Australia, [3]Department of Neurology, Royal Melbourne Hospital, Melbourne, Australia

Purpose: Evidence suggests that early-life stress has enduring effects on the brain that may contribute to the development of limbic epilepsy in later life, possibly involving stress hormones. Using the maternal separation model of early-life stress in rats, this study was designed to assess whether the effects of early-life stress on electrical amygdala kindling are mediated by corticosterone.
Method: On P2-14, rats were exposed to either maternal separation for 180 min/day (MS180), or a handling control for 15 min/day (MS15). At P49, bipolar electrodes were implanted into the left amygdala, and one week later rats were tested for seizure threshold and subjected to electrical amygdala kindling until fully kindled (5 class V seizures, Racine scale). Rats were injected with either metyrapone, a corticosterone synthesis inhibitor (50mg/kg, s.c), or vehicle 60 minutes prior to each stimulation throughout the kindling period.
Results: MS180 rats displayed a reduced seizure threshold (p=0.03), and longer seizure duration (p=0.02) compared to MS15 rats. These effects of MS180 stress were significantly attenuated by treatment with metyrapone (seizure threshold: p=0.0001; seizure duration: p=0.018). In addition, metyrapone-treated rats required more stimulations to reach the fully kindled state compared with vehicle-treated rats, an effect that was seen exclusively in the MS180 group (p=0.03). Groups: MS180-MET: n=18, MS180-VEH: n=19, MS15-MET: n=17, MS15-VEH: n=18.

PLATFORM SESSION ABSTRACTS

Conclusion: Our data demonstrates that inhibition of seizure-induced corticosterone synthesis alleviates the enduring effects of maternal separation stress on seizure threshold, seizure duration and kindling rate in rats, suggesting that corticosterone may be involved in the epileptogenic process.

047
Bilateral focal neuropeptide Y injections in the somatosensory cortex maximally suppresses absences seizures in a genetic rat model
VAN RAAY L[1], JOVANOVSKA V[1], MORRIS MJ[2], O'BRIEN TJ[1]
[1]The University of Melbourne, Melbourne, Australia, [2]The University of New South Wales, Sydney, Australia

Purpose: Neuropeptide Y (NPY) is an inhibitory neurotransmitter which can ameliorate focal seizures, and has more recently been discovered to control generalised absence seizures in the GAERS animal model. In this study we investigated the intra-cerebral location of this effect by performing bilateral focal injections of NPY in different regions of the thalamocortical circuit.
Method: After bilateral implantation of injection cannulae rats were allowed one week recovery. Neuropeptide Y was administration in the primary motor cortex, primary somatosensory cortex (S1), secondary somatosensory cortex (S2), nucleus reticular thalamus and the ventrobasal thalamus were determined. Animals received three doses of NPY and vehicle in randomised order. EEG recordings were carried out for 30 min prior to injection and 90 min after the injection of NPY or vehicle.
Results: Our results showed that total time in seizure post NPY injection decreased with increasing NPY dose in both somatosensory cortex regions; with the S2 being significantly different at the highest dose compared to vehicle (6.71 ±2.1 min post vehicle to 0.24 ±0.1 min post 1.5nmol NPY, $p< 0.05$). No significant effect on seizures was seen in any other region. The numbers of seizures per minute expressed as percentage of control show a similar pattern.
Conclusion: These findings provide evidence that NPY is most effective in suppressing seizures when delivered focally to the S2 somotosensory cortex, suggesting this region is primarily responsible for the anti-absence effect of NPY.

Genetics
Saturday 23rd October
17:30-18:30
Room 106

048
Is HLA-B*1502 associated with carbamazepine / phenytoin-induced severe cutaneous adverse reactions in a Thai population?
TIAMKAO S, SRINAGARIND EPILEPSY RESEARCH GROUP
Khon Kaen University, Khon Kaen, Thailand

Purpose: Carbamazepine (CBZ) and phenytoin (PHT) has been reported as the most common culprit drug for Stevens-Johnson syndrome (SJS) and toxic epidermal necrolysis (TEN). Since the number of spontaneous reports of CBZ/PHT - related SJS/TEN in Thailand is among the highest in the World, the use of *HLA-B*1502* as a pharmacogenetic test for CBZ/PHT - induced SJS/TEN may help to prevent these severe cutaneous adverse drug reactions. Therefore, a case-control study was conducted to assess the degree of association between the *HLA-B*1502* allele and CBZ/PHT - induced SJS/TEN in a Thai population.
Method: 48 CBZ/18 PHT - induced SJS/TEN and 42 CBZ/36 PHT - tolerant patients were enrolled in the study. The presence of *HLA-B*1502* allele were analyzed by allele specific polymerase chain reaction using a PG1502 DNA detection kit.

PLATFORM SESSION ABSTRACTS

Results: Among 48 CBZ/ 36 PHT - induced SJS/TEN patients, 43 (89.58%)/ 5 (27.78%) patients carried the *HLA-B*1502*, respectively, while 5 (11.90%)/ 7 (19.44%) of the CBZ/ PHT - tolerant controls had this allele, respectively. The risk of CBZ-induced SJS/TEN was significantly higher in the patients with *HLA-B*1502* with an odds ratio of 63.64 (95% CI 14.91-295.23, p< 10-4). But, the risk of PHT - induced SJS/TEN was not significant, with odds ratio of 1.59 (95% CI 0.32-7.13, *p*= 0.5) The sensitivity and specificity of *HLA-B*1502* for prediction of CBZ-induced SJS/TEN were 89.58% and 88.10%.
Conclusion: The results suggest that HLA-B*1502 is a valid genetic marker for screening Thai individuals who may be at risk for CBZ-induced life-threatening SJS and TEN.

049

Autosomal dominant vasovagal syncope: clinical and linkage results

KLEIN KM[1,2], BROMHEAD C[3], SMITH K[3], O'CALLAGHAN CJ[2], SCHEFFER IE[1,2], MCMAHON JM[1,2], LAWRENCE KM[1,2], DIBBENS LM[4,5], BAHLO M[3], BERKOVIC SF[1,2]
[1]Epilepsy Research Centre, The University of Melbourne, West Heidelberg, VIC, Australia, [2]Department of Medicine, Austin Health and Northern Health, Heidelberg, VIC, Australia, [3]The Walter and Eliza Hall Institute of Medical Research, Parkville, VIC, Australia, [4]Department of Genetic Medicine, Women's and Children's Hospital, North Adelaide, SA, Australia, [5]School of Paediatrics and Reproductive Health, University of Adelaide, Adelaide, SA, Australia

Purpose: Syncope is the most common differential diagnosis of epilepsy. The most frequent type is vasovagal syncope (VVS) for which the aetiology remains elusive. Here we provide evidence for a genetic aetiology by describing six families with autosomal dominant VVS and the linkage results in the largest family.
Method: Patients with VVS and a family history of syncope were recruited. A questionnaire addressing features differentiating syncope from epilepsy was administered to all available family members. Medical records were obtained and additional diagnostic tests performed in selected individuals. Linkage analysis was performed in the largest family, containing 23 affected and 12 unaffected (including 9 married-in) family members.
Results: Six families with VVS, consistent with autosomal dominant inheritance, were recruited in addition to several smaller families. The largest comprised 30 affected individuals over three generations with a median onset at 8-9 years. Typical triggers for VVS were heat, sight of blood, injury, medical procedures, prolonged standing, pain and frightening thoughts. The triggers varied considerably within the family. The second largest family included 10 affected subjects over three generations with a median onset at 10-11 years and varying triggers. The other four families were smaller with 4-5 affecteds over 2-3 generations.
Linkage analysis in the largest family revealed a significant maximum LOD score of 3.28 on chromosome 15q.
Conclusion: Autosomal dominant VVS is not a rare phenomenon. Linkage to chromosome 15q was demonstrated in a large family. Identification of the genetic cause will help to further understanding of pathophysiology.

050

Haploinsufficiency of STXBP1 is an important cause for Ohtahara syndrome, but not for cryptogenic West syndrome

KATO M[1], SAITSU H[2], OKADA I[2], ORII KE[3], HIGUCHI T[4], HOSHINO H[5], KUBOTA M[5], ARAI H[6], TAGAWA T[7], KIMURA S[8], SUDO A[9], MIYAMA S[10], TAKAMI Y[11], WATANABE T[12], NISHIMURA A[2], NISHIYAMA K[2], MIYAKE N[2], WADA T[13], OSAKA H[13], KONDO N[3], HAYASAKA K[1], MATSUMOTO N[2]
[1]Yamagata University Faculty of Medicine, Yamagata, Japan, [2]Yokohama City University Graduate School of Medicine, Yokohama, Japan, [3]Gifu University, Gifu, Japan, [4]Shinshu University School of Medicine, Matsumoto, Japan, [5]National Center for Child Health and Development, Tokyo, Japan, [6]Morinomiya Hospital, Osaka, Japan, [7]Osaka Koseinenkin Hospital, Osaka, Japan, [8]Akita Red Cross Hospital, Akita, Japan, [9]Sapporo City General Hospital, Sapporo, Japan, [10]Tokyo Metropolitan Children's Medical Center, Fuchu, Japan, [11]Himeji Red Cross Hospital, Himeji, Japan, [12]Hokkaido Medical Center for Child Health and Rehabilitation, Sapporo, Japan, [13]Kanagawa Children's Medical Center, Yokohama, Japan

PLATFORM SESSION ABSTRACTS

Purpose: *De novo STXBP1* mutations have been found in individuals with Ohtahara syndrome or early infantile epileptic encephalopathy with suppression-burst. Our aim was to delineate the clinical spectrum of subjects with *STXBP1* mutations.
Method: *STXBP1* was analyzed in 29 and 54 cases of Ohtahara syndrome and West syndrome, respectively, as a second cohort. Brain malformations were not found in all cases. Mutation screening of *STXBP1* was performed by high resolution melt analysis (HRM), and then direct sequencing was done for the samples showing an aberrant HRM pattern.
Results: A total of seven novel *STXBP1* mutations were found in nine Ohtahara syndrome cases, but not in West syndrome. The mutations include two frameshift mutations, three nonsense mutations, a splicing mutation, and a recurrent missense mutation in three unrelated cases. Including our previous data, 10 out of 14 individuals (71 %) with *STXBP1* aberrations had the onset of spasms after one month, suggesting relatively later onset of epileptic spasms.
Conclusion: Collectively, *STXBP1* aberrations can account for about one third individuals with Ohtahara syndrome (14 out of 43). These genetic and biological data clearly showed that haploinsufficiency of *STXBP1* is the important cause for cryptogenic Ohtahara syndrome, but not or rarely for cryptogenic West syndrome.

051
Benign familial neonatal seizures: the mutational spectrum, clinical overlap with benign familial neonatal-infantile seizures and evidence for an additional locus
HERON SE[1,2], GRINTON BE[3], PELEKANOS J[3], CARRANZA D[3], ZUBERI SM[4], LUNAN R[4], KIVITY S[5], AFAWI Z[6], SCHEFFER IE[3,7], BERKOVIC SF[3], MULLEY JC[1,2]
[1]SA Pathology at Women's and Children's Hospital, North Adelaide, SA, Australia, [2]University of Adelaide, Adelaide, SA, Australia, [3]Epilepsy Research Centre, The University of Melbourne, Heidelberg, VIC, Australia, [4]Royal Hospital for Sick Children, Glasgow, United Kingdom, [5]Schneider Children's Medical Center of Israel, Petach Tikva, Israel, [6]Tel Aviv Sourasky Medical Center, Tel Aviv, Israel, [7]Royal Children's Hospital, Melbourne, VIC, Australia

Purpose: Benign familial neonatal seizures (BFNS) can be caused by mutations in the potassium channel subunit genes *KCNQ2* and *KCNQ3*. This study aimed to determine the frequency of potassium channel mutations in BFNS and to analyze families without detectable potassium channel mutations to determine whether additional genes are implicated.
Method: Probands from 33 BFNS families were tested for potassium channel mutations by sequencing and multiplex ligation-dependent probe amplification (MLPA) to detect deletions and duplications where no mutation was found by sequencing. Microsatellite markers linked to five loci (*KCNQ2, KCNQ3, SCN2A*, two chromosome 5 inversion breakpoints described by Concolino et al, *J Med Genet* 2002;39:214-216) associated with neonatal seizures were genotyped in families without mutations. Probands from families consistent with linkage to *SCN2A* were tested for mutations by sequencing.
Results: Twenty-eight families had mutations in *KCNQ2* or *KCNQ3* (22 detected by sequencing and six by MLPA). Mutations in *SCN2A* were found in another two families. Linkage to the known BFNS loci was excluded for one of the three unsolved families and linkage to *KCNQ2, KCNQ3* and *SCN2A* was excluded for another.
Conclusion: The great majority (85%) of BFNS families have potassium channel mutations. Two families had mutations in *SCN2A*, showing that there is overlap in the distribution of phenotypes between BFNS and benign familial neonatal-infantile seizures that can only be resolved at the molecular genetic level. Linkage to known neonatal seizures genes was excluded for two families, indicating that there is an additional unknown gene or genes for BFNS.

052
Genetic analysis in the patients of epilepsy with mental retardation
NAKAGAWA E[1,2], TAKESHITA E[1,2], GOTO Y-I[1,2]
[1]National Center Hospital of Neurology and Psychiatry, National Center of Neurology and Psychiatry (NCNP), Kodaira, Japan, [2]Department of Mental Retardation and Birth Defect Research, National Institute of Neuroscience, NCNP, Kodaira, Japan

PLATFORM SESSION ABSTRACTS

Purpose: Mental retardation (MR) affects 1-3% of the population. One of the major causes of MR is based on mutations in the related genes which are timely and locally expressed in concert with one another in CNS. Our research group intended to organize a depository at NCNP for genetic analysis of MR. In this study, we evaluated the genetic and clinical features on epilepsy associated with MR.
Method: We have already established the research resource facility and collected samples under informed consent by providing the diagnostic service for known genetic defects or chromosomal abnormalities, such as genes for fragile X, *UBE3, ATRX, MECP2, FMR1, ARX, PQBP1, RPS6KA3, IL1RAPL1, TM4SF2, OPHN1, PAK3, FACL4, AGTR2, ARHGEF6, GDI1, SLC6A8, FTSJ1, ZNF41, DLG3*, and copy number aberrations in X-tiling CGH array analysis.
Results: As of the end of June in 2010, 343 MR families consisted of 146 familial and 197 sporadic cases have been registered. In these cases, 52% cases accompanied with epilepsy and 25% cases with developmental disorders, respectively. 50 positive genetic results for 300 examined cases by analyses of 19 X-linked genes and array CGH, which accounts for approximately 17% of unexplained MR. 26 cases with epilepsy, including *ARX*, *ZNF41* gene mutation, chromosomal micro-deletions and Xq28 duplications including *MECP2*.
Conclusion: By analyses of 19 X-linked genes and chromosomes including array CGH, approximately 17% of cases had positive results. Approximately 52% accompanied with epilepsy and 25% with developmental disorders. The cases of Xq28 duplications including *MECP2* presented with mental retardation and intractable epilepsy.

AED issues
Saturday 23rd October
17:30-18:30
Room 109

053
Antiepileptic drug use in pregnancy: impact on brain function of exposed Australian children
NADEBAUM C[1,2], ANDERSON VA[2,3,4], VAJDA FJE5, REUTENS D[6], BARTON S[1,2], WOOD AG[1,2]
[1]Monash University, Melbourne, Australia, [2]Murdoch Childrens Research Institute, Melbourne, Australia, [3]Royal Children's Hospital, Melbourne, Australia, [4]University of Melbourne, Melbourne, Australia, [5]Australian Pregnancy Register for Women with Epilepsy and Allied Disorders, Melbourne, Australia, [6]University of Queensland, Brisbane, Australia

Purpose: Despite the risk of major malformation or intellectual impairment due to fetal antiepileptic drug exposure, pharmacotherapy is typically continued throughout pregnancy because of the increased risk of complications due to recurrent seizures. This research aims to characterise the long-term impact of exposure to antiepileptic medications *in utero* by studying cognitive outcomes in children born to mothers with epilepsy.
Method: One hundred and two school-aged children (six to eight years) exposed to antiepileptic medications during pregnancy participated in neuropsychological examination. Details of drug type and dose during each trimester of pregnancy was obtained from prospectively collected records. Children without reported major malformations were eligible for the study. All assessors were blinded to drug status. Preliminary results are presented.
Results: T-test comparisons indicated that children exposed to valproate or polytherapy performed significantly below the population mean on standardised tests of intelligence and language ($p \leq .05$). Outcomes of children exposed to carbamazepine did not significantly differ from the mean. Regression analyses showed that valproate and polytherapy exposure significantly predicted outcomes, even when controlling for maternal IQ.
Conclusion: Preliminary findings indicate that fetal exposure to valproate or polytherapy impacts negatively on long-term intellectual and language outcomes. Further investigation of our data is required to determine whether specific doses or combinations of drugs are associated with poorer outcomes, and to better understand the underlying mechanisms. These findings will have major implications for clinical management of affected women. Ongoing longitudinal research on newer agents and their cognitive consequences is required.

PLATFORM SESSION ABSTRACTS

054

Effect of seizures on cognition, behavior, and quality of life during carbamazepine or lamotrigine monotherapy in patients with newly diagnosed or untreated partial epilepsy

LEE S-A[1], KIM M-J[1], LEE H-W[2], HEO K[3], SHIN D-J[4], SONG H-K[5], KIM O-J[6], LEE S-M[1], KIM S-O[1], LEE B-I[3]

[1]Asan Medical Center, University of Ulsan College of Medicine, Seoul, Korea, Republic of, [2]Department of Neurology, Ewha University College of Medicine, Seoul, Korea, Republic of, [3]Department of Neurology, Yonsei University College of Medicine, Seoul, Korea, Republic of, [4]Department of Neurology, Gacheon University College of Medicine, Incheon, Korea, Republic of, [5]Department of Neurology, Hallym University Institute of Epilepsy Research, Seoul, Korea, Republic of, [6]Department of Neurology, CHA University College of Medicine, Bundang, Korea, Republic of

Purpose: We investigated effects of recurrent seizures on cognition, behavior, and quality of life during the first year of monotherapy in patients with newly diagnosed epilepsy.
Method: Patients with newly diagnosed or untreated partial epilepsy were randomized to lamotrigine or carbamazepine monotherapy. Study consisted of 8-week dose-titration and 40-week maintenance period. Patients were categorized as seizure-free (SF) or not-seizure-free (NSF) groups depending on seizures recurrence during the maintenance period. Neuropsychological tests, symptom check list-90 (SCL-90), and QOLIE-31 were assessed at baseline and final 48-week. Primary and secondary outcome measures were a group-by-time interaction and seizure group effects, respectively. They were analyzed using a linear mixed model.
Results: Seventy three completed the 48-week study (lamotrigine, n=39; carbamazepine, n=34). The percentage of SF patients was 68.5%. Significant group-by-time interaction was identified in Serial Clustering and Recognition Hits of California Verbal Learning Test (CVLT), Positive Symptom Distress Index of SCL-90, and subscales such as Seizure Worry and Social Function and total scores of QOLIE-31, with the SF group being significantly better. Seizure effects were observed on Copy and Recognition of Rey Complex Figure Test, Color correct response and Color-Word Interference of Stroop Test, semantic and phonemic fluency of Controlled Oral Word Association Task, several variables of CVLT, and Energy of QOLIE-31. All variables except for Energy were worse in NSF group.
Conclusion: Our results showed that recurrent seizures have significant influences on some aspects of cognition, behavior, and quality of life in patients with newly diagnosed epilepsy.

055

KONQUEST: Keppra versus older AEDS and neuropsychiatric, neurocognitive and quality of life outcomes in treatment of epilepsy as substitution

HAKAMI T[1], TODARO M[1,2], ROTEN A[2], BRIGHT T[1], GERMAINE D[2], PETROVSKI S[1,3], MACGREGOR L[1], GASKO H[3], MATKOVIC Z[2], YERRA R[1,2], O'BRIEN TJ[1,2]

[1]The University of Melbourne, Melbourne, Australia, [2]Royal Melbourne Hospital, Melbourne, Australia, 3BioGrid Australia, Melbourne, Australia

Purpose: To compare a broad range of epileptic and psychological outcome measures in patients who had substitution monotherapy with levetiracetam (LEV) versus two older anti-epileptic drugs (AEDs), carbamazepine (CBZ) or valproate (VPA).
Method: KONQUEST was a single-centre, randomised, open-label study. Participants had partial epilepsy on monotherapy which had "failed" either due to lack of efficacy or adverse effects. Participants taking phenytoin (PHT) or CBZ were randomised to either LEV or VPA, and participants taking VPA were randomised to LEV or CBZ. Assessments were performed at baseline, 3 months, and 12 months using questionnaires measuring seizure control, anxiety and depression (HADS), psychiatric distress (Symptoms Checklist 90 - SCL 90), Quality of Life in Epilepsy (QOLIE 89), adverse effects (Liverpool Adverse Effect Profile - LEAP) and, neurocognitive performance (IntegNeuro™). Outcomes analysis was performed on the basis of intention to treat.
Results: 96/106 (90.6%) of enrolled patients completed the study: 51 in the LEV and 45 in the older AED groups (VPA, n=26, CBZ, n=19). All assessments improved from baseline at both 3 and 12 months (P< 0.05); however, we found no differences between the LEV and older AED treatment groups (P>0.05). A similar proportion of patients in both groups remained seizures free (49% vs. 53%, P>0.05)

PLATFORM SESSION ABSTRACTS

Conclusion: Switching to a different AED in patients who are experiencing ongoing seizures or adverse effects to their first AED is associated with improvement on a wide variety of epilepsy and psychosocial measures. This effect is similar for both LEV and the older AEDs.

056
Cognitive effects of antiepileptic drugs: a meta-analysis
SANTOS RP
University of the Philippines - Philippine General Hospital, Manila, Philippines

Purpose: Patients with epilepsy frequently experience cognitive difficulties and mood alterations with use of antiepileptic drugs (AEDs). This study aims to determine the overall effect of AEDs on mood and cognition, specifically on depression, tension, anger, vigor, fatigue, choice reaction time, confusion and memory.
Method: Using Medline, Cochrane and Ovid data bases, all randomized controlled trials published through August 2007 that studied the effects of AEDs on mood and cognition in healthy adults. A manual search was also performed using a reference list from the retrieved trial and included only in English and with human subjects. The criteria for inclusion were: 1. Randomized allocation, 2. any AEDs use, and 3. subsequent evaluation of its cognitive effects. Details of neurobehavioral measures common in all the studies were extracted and analyzed using the RevMan 4.2 software. Treatment effects were measured using standardized mean deviations and pooled using random effects. All studies included were homogenous (I^2 = 92.8% · 98.4%).
Results: Four studies involving 146 patients (97 males, 49 females) aged 19 · 76 (mean age: 47.5 years) and 5 AEDs (Phenytoin, Valproate, Topiramate, Carbamazepine, Gabapentin, and Lamotrigine) were included in this meta-analysis. Phenobarbital consistently had the most significant effect on mood and cognition, followed by Phenytoin and Valproic Acid with Gabapentin causing the least mood and cognitive dysfunction.
Conclusion: The use of older AEDs demonstrated greater effects on cognition and mood than newer ones. These effects should be taken into consideration when prescribing AEDs.

057
Biodegradable polymers as drug delivery devices for the treatment of epilepsy
SUHR C[1], MOULTON S[2], HALLIDAY A[1], MCLEAN K[1], WALLACE G[2], COOK M[1]
[1]St Vincent's Hospital, Melbourne, Australia, [2]University of Wollongong, Wollongong, Australia

Purpose: Epilepsy remains an important target for the development of new treatment strategies, with approximately one third of patients receiving minimal or no benefit from antiepileptic drugs. Evidence suggests that an insufficient amount of drug is able to cross the blood brain barrier to reach the intended area. To address these limitations we aim to develop a long-term drug delivery device using biodegradable polymers, which can introduce drugs directly to the brain at the seizure focus. Biodegradable polymers have been widely explored as drug delivery devices, and have been shown to be well tolerated by the brain, can achieve slow degradation rates, and eliminate the need for surgical removal.
Method: Biodegradable polymer sheets (PLA:PLGA, 85:15) loaded with Valproate were bilaterally implanted subdurally onto the somatosensory cortex in GAERS (Genetic Absence Epilepsy Rats from Strasbourg) to investigate effects of passive drug release on seizure frequency, percentage of time spent in seizure and average seizure length over a period of eight weeks.
Results: Preliminary results reveal a sustained reduction of both average seizure length, and the percentage of time spent in seizure compared to controls over the recording period.
Conclusion: These results lend weight to further research, suggesting that polymer drug delivery devices have the capacity for long-term neurological benefit in epileptic patients.

PLATFORM SESSION ABSTRACTS

Surgery
Saturday 23rd October
17:30-18:30
Room 110

058
Vagus nerve stimulation: outcome and predictors of seizure freedom in long-term follow-up
GHAEMI K, SURGERY
Birjand University of Medical Science, Birjand, Iran, Islamic Republic of

Objectives: To present long-term outcome and to identify predictors of seizure freedom after vagus nerve stimulation (VNS).
Method: All the patients who had undergone VNS implantation were retrospectively reviewed in the Bethel epilepsy centre. These patients had undergone complete presurgical evaluation including detailed clinical history, magnetic resonance imaging ,and long term video-EEG by means of ictal and interictal recordings. All patients had a minimum of 2 year follow-up. After implantation, adjustment of stimulation parameters and concomitant antiepileptic drugs were at the treating physician's discretion.
Results: Ten patients remained free from seizure for more than one year after VNS implantation(6.9%). Seizure improved in 89 patients (61.8%) but no changes were observed in 45 patients (31.3%). In univariate analysis the following factors were significant: age at implantation, multifocal interictal epileptiform discharge, unilateral interictal epileptiform discharge, cortical dysgenesis, and psychomotor seizure. Stepwise multivariate analysis showed that for unilateral IED, P=0.014, HR=0.112 (95%CIs, 0.019 · 0.642),for cortical dysgenesis P=0.007, HR=0.065 (95% CIs, 0.009 · 0.481) and for younger age at implantation P=0.026, HR=7.533 (95% CIs 1.28·44.50) were independent predictors of seizure freedom in long-term follow-up.
Conclusion: VNS implantation may render patients with some forms of cortical dysgenesis (parietooccipital polymicrogyria, macrogyria) seizure free. Patients with unilateral IEDs and earlier implantation achieved the most benefit from VNS.

059
Avoidance of complications in epilepsy surgery
MALLA BR
Kokilaben Dhirubhai Ambani Hospital, Mumbai, Maharashtra, India

Purpose: Surgery for epilepsy ideally would cure or control seizures without causing any disabling neurological deficits. However invasive monitoring as well as various resective and disconnection procedures carry a unique set of potential adverse outcomes or complications. This presentation will highlight the occurrence of surgical complications and emphasize upon their avoidance.
Method: Between 1995 and 2010, the author has performed five hundred epilepsy surgery procedures. These procedures consisted predominantly anteromesial temporal resections for mesial temporal sclerosis, lesions with the rest being extra temporal resections and disconnections for focal cortical dysplasia and various other pathologies.
Results: The complications ranged from meningitis (5), permanent hemiplegia (3), intracranial hemorrhage (3), and CSF otorrhea (2), and subdural emphyema (1), expanding cyst at the temporal lobectomy site (1) to death (1). Though the incidence of these surgical complications is low, it has an adverse effect on developing epilepsy surgery programmes especially in countries with limited resources.
Conclusion: Neurosurgeons must strive to understand the intricacies of the surgical procedure and be aware of potential pitfalls, in order to maximize surgical outcome and reduce neurological complications. Quality control in epilepsy surgery is very important and we must endeavor to attain high and acceptable standards.

PLATFORM SESSION ABSTRACTS

060

Long-term outcome of corpus callosotomy for West syndrome: second report

BABA H, TODA K, ONO K
National Nagasaki Medical Center, Omura, Japan

Purpose: In previous AOEC meeting, we report surgical results of corpus callostomy (CC) for young children with West syndrome (WS). In this study, we analyzed surgical results, change of psychomotor developments and prognostic factors of CC in 49 patients with WS.
Method: CC was performed in 49 patients. Age at the seizure onset ranged from 0 to 23 months (mean 4.2). Severe developmental delay before the onset of epilepsy was observed in 26 patients (53.1%). Patients with resectable lesion on MRI were excluded. Mean age at CC was 23.6m. Anterior CC was performed in 2 patients and total CC in 47 patients. For a psychological test, Kinder Infant Developmental Scale (KIDS) was used. Surgical outcome was categorized as F (seizure free), E (greater than 80% reduction), G (greater than 50% reduction) and P (no significant change).
Results: Tonic spasm (SP) was recorded in all patients except for 2, whose seizure was tonic seizure(TS). Preoperative DQ and DA is mean 18.5 and 3.9 months, respectively. Surgical outcome was F in 30.6%, E in 18.4%, G in 30.6% and P in 20.4% of patients. 79% had significant improvements after CC. SP abolished in 40% of patients. Pre-operative prognostic factors were analyzed between F+E group and G+P group. Higher preoperative DA (p=0.04) and normal developments before the onset of epilepsy (p=0.001) are significant factors for seizure control. In F + E group, Developmental velocity was significantly greater than in G + P group at the final follow-up point.
Conclusion: This study reconfirmed our preliminary previous results.

061

Surgical treatment of the patients with Rasmussen's encephalitis (20 cases)

LUAN G, GUAN Y
Capital Medical University Sanbo Brain Institute, Beijing, China

Purpose: Presenting of 20 cases with Rasmussen´s encephalitis (RE) being surgically treated, to observe the clinical effects after 3 to 54months follow-up.
Method: Retrospectively analyze the clinical data of 20 cases with RE in our epilepsy center from April 2004 to June 2009. Of the 20 cases, 9 were males and 11 were females. The age of onset was from 1.7 to 17 years (average 5.7) and disease duration of 5 months to 12 years (average 3.6). Different operations had been made based on the pre-operation evaluations (Adame's Modified hemispherectomy, functional hemispherectomy, hemispherotomy and selectively epileptogenic zone resection. Regular follow-up of all cases after operation was made.
Results: 4 cases were operated with Adame's Modified hemispherectomy (AH), 10 case were operated with functional hemispherectomy(FH) and 5 cases with hemispherotomy, 1 cases with selectively epileptogenic zone resection. During 3 to 54 months follow-up, 16cases had seizure free (Engel I, 80%) , 3 cases with alleviation of seizures (1 cases with Engel class II and 1 with Engel class III), 1 case had frequent seizures after operation(probably bilateral RE). After operation, most patient are able to walk without the use of assistive devices except one case, but the fine motor hand movements were lost.
Conclusion: RE is a drug-resistant epilepsy syndromes accompanied by progressive neurological deterioration. Hemispherectomy is a viable alternative, which should be considered in the treatment of RE based on the pre-evaluations, various operation methods (AH, FH, hemispherotomy) don't influence seizure outcome, but the long-term effect remains to be observe.

062

Feasibility and safety of antiepileptic drug withdrawal following resective extratemporal surgery

MENON RN, RATHORE C, RADHAKRISHNAN K
Sree Chitra Tirunal Institute for Medical Sciences and Technology, Trivandrum, India

Purpose: To investigate the feasibility of antiepileptic drug (AED) withdrawal following extratemporal resective surgeries and to identify the predictors of seizure recurrence on attempted AED reduction.

PLATFORM SESSION ABSTRACTS

Method: We studied the postoperative AED profile of 105 patients who underwent resective surgery for medically refractory extratemporal epilepsy and were followed for a minimum of two years. AED withdrawal was attempted in all patients who were seizure free for initial three months or for at-least one year, in case of early recurrence. Recurred and non-recurred groups, on attempted AED withdrawal, were compared by logistic-regression analyses.
Results: The mean age at surgery was 19.6±8.7 years and pre-surgery epilepsy duration was 12.4±8 years. At mean follow-up duration of 4.3±2.2 years, 61(58.1%) patients were seizure-free. Following surgery, 66(62.9%) patients were on duotherapy and 39(37.2%) were on polytherapy. AED withdrawal was attempted in 93(88.6%) patients. At last follow-up, AEDs were stopped in 9(8.6%) patients, reduced in 56(53.3%) patients and unchanged in 40(38.1%) patients. Forty-three (41%) patients had seizure recurrence while reducing AEDs, of which only eight became seizure-free after restarting AED. On multivariate analysis, older age at surgery (p=0.001), longer preoperative epilepsy duration (p=0.001), abnormal postoperative EEG (p=0.005) and early postoperative seizures (p=0.024) were predictive of seizure recurrence.
Conclusion: Following resective extratemporal surgery, AEDs can be reduced in ~60% of patients. As ~40% of patients develop seizure recurrence on attempted taper, with lesser likelihood of seizure freedom subsequently, AED withdrawal should be attempted very cautiously in presence of certain risk factors identified in this study.

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POSTER ABSTRACTS

Basic science

p063

Levetiracetam-releasing biodegradable polymer sheets effective at reducing seizures in the tetanus toxin model of chronic mesial temporal lobe epilepsy in rats

HALLIDAY AJ[1,2], CAMPBELL TE[2], NELSON TS[3], MCLEAN KJ[1], SUHR CL[2], COOK MJ[1,3], WALLACE GG[2]
[1]Centre for Clinical Neurosciences and Neurological Research, Fitzroy, Australia, [2]Intelligent Polymer Research Institute and ARC Centre of Excellence for Electromaterials Science, University of Wollongong, Wollongong, Australia, [3]The Bionic Ear Institute, East Melbourne, Australia

Purpose: Approximately one third of people with epilepsy receive insufficient benefit from currently available anticonvulsant medication. Recent evidence suggests that this may be due to a lack of effective drug penetration through the blood brain barrier. We sought to investigate the efficacy of a polymer-based drug delivery device implanted within the CNS in order to administer anticonvulsant drugs directly to the brain in an animal model of epilepsy.
Method: Biodegradable PLGA-based implants loaded with Levetiracetam were produced and implanted above the motor cortex of Sprague Dawley rats that had received 50ng injections of tetanus toxin into the right hippocampus. Spontaneous seizures were observed and recorded for three weeks via a multichannel electrode array implanted in the left motor cortex. The seizure frequency, duration and severity of implanted rats was compared to control rats that received no implant.
Results: Implantation of Levetiracetam-loaded sheets led to a significant decrease in seizure duration for one week. There was also a non-significant trend towards fewer seizures in the first week and less severe seizures in the second week following implantation.
Conclusion: The results of this study indicate that drug-eluting polymer implants represent a promising evolving treatment option for intractable epilepsy. Future experiments will examine the effects of sham and blank polymer implantations. Future research is warranted to investigate issues of device longevity.

p064

Novel oxazolidinones as potential anticonvulsant agents: in vitro testing

KOMBIAN SB, ANANTHALAKSHMI KVV, PHILLIPS OA
Faculty fo Pharmacy, Kuwait University, Kuwait City, Safat, Kuwait

Purpose: Currently, about 30-40% of seizures are either not adequately controlled or are resistant to available antiepileptic drugs(AEDs). Most of these AEDs also have side effects that make patients non-adherent. Hence the need for newer, more effective anticonvulsants that may cover these resistant cases with few side effects is paramount. Here we tested a novel oxazolidinone, PH084 for anticonvulsant activity *in vitro*.
Methods: Whole-cell and extracellular field recordings were done in coronal slices of rat hippocampus and the effect of PH084, a prototype oxazolidinone, was tested on excitatory postsynaptic currents (EPSCs), action potentials and chemically-induced seizures.
Results: Bath application of PH084 caused suppression in EPSC amplitude in a concentration-dependent manner, with 10uM suppressing EPSC by -25.4 ± 10.4% (n=6). PH084(10 uM) also suppressed action potential firing frequency by -42.2± 13.5% (n=6) and population spike (PS) by -38.2 ± 8.9% (n=4). Perfusion with buffer containing zero Mg^{2+} converted an evoked PS to multiple PS (mPS: 5.0 ± 0.3 spikes; n=7) accompanied by spontaneous bursting (SB) activity (9.0 ±1.6/min; n=5). PH084 (10 uM) suppressed the number of spikes mPS by -38.7 ± 8.3% (n=7) and the frequency of SBs by -47.5 ± 8.9% (n=5). However, pretreatment of slices with PH084 (10 uM) did not block the development or attenuate the zero Mg^{2+}-induced mPS (3.9 ± 0.6 spikes, n=5) and SBs (11.0 ±1.8/min, n=4)

POSTER ABSTRACTS

Conclusion: Our data suggest that, while PH084 has potential for use as an anticonvulsant, it may not have potential for use as an antiepileptogenic agent.

p065

MicroRNA profiles involved in the hippocampal epileptogenesis

LEE J-J[1], PARK K-I[2], CHU K[3], LEE S-T[3], JUNG K-H[3], LEE SK[4], ROH J-K[3]

[1]Department of Neurology Eulji General Hospital, Eulji University College of Medicine, Seoul, Korea, Republic of, [2]Department of Neurology, Seoul Paik Hospital, Inje University College of Medicine, Seoul, Korea, Republic of, [3]Stroke & Neural Stem Cell Laboratory in Clinical Research Institute, Department of Neurology, Seoul National University Hospital, Seoul, Korea, Republic of, [4]Department of Neurology, Comprehensive Epilepsy Center, Clinical Research Institute, Seoul National University College of Medicine, Seoul, Korea, Republic of

Background and purpose: Epilepsies are very common disorders. However, their pathomechanisms and genetic control mechanisms are not elucidated yet. We investigated alterations of microRNA (miRNA) profiles to explain the genetic control mechanisms of hippocampal sclerosis.
Material and methods: We induced status epilepticus (SE) using C57BL mouse by pilocarpine injection. Bilateral hippocampi were obtained from each control group (N=3), 6h after pilocarpine induced SE group (PISE-6h, N=3), 7d after pilocarpine induced SE group (PISE-7d, N=3), and 28d after pilocarpine induced SE group (PISE-24d, N=3) and miRNA preparation was performed. Using microarray and real time PCR method, differences among each group were analyzed. Gene expression of PISE-28d group were also analyzed.
Results: Compared with normal control group, expressions of some miR decreased and those of other miR increased in PISE-6h, PISE-7d and PISE-28d compared with normal control. Results of RT-PCR analyses were compatible with the findings of microarray analyses. Each meaning of profile change and gene expression is discussed.
Conclusions: This study is the first attempt to reveal the alterations of miRNA profile in each epileptogenic step. Researches to elucidate the role of miRNAs in epileptogenesis and therapy should be followed.
Key words: Epilepsy, Hippocampal sclerosis, MicroRNA

p066

Reorganization of neural circuits of reuniens thalamic nucleus in the mouse brain after pilocarpine induced temporal lobe epilepsy

MA DL[1], TANG FR[2], GOH E[1]

[1]Duke-NUS Graduate Medical School, Singapore, Singapore, [2]National University of Singapore, Singapore, Singapore

Purpose: This study investigated the reorganization of neural circuit of the reuniens thalamic nucleus (RE) and its related brain regions at 2 months after pilocarpine induced temporal lobe epilepsy.

POSTER ABSTRACTS

Method: We examined the efferent and afferent pathway of RE in the control and epileptic mice, by iontophoretical injection of an anterograde tracer phaseolus vulgaris leucoagglutinin (PHA-L) or a retrograde tracer cholera toxin B subunit (CTB) into the RE.
Results: Our results showed more PHA-L labelled en passant in the efferent pathway of RE of epileptic mice as compared to control mice. The terminal boutons with larger dimensions in these epileptic mice were found not only in the CA1 area of hippocampus and subiculum, but also in the basal lateral amygdala and perirhinal cortex. Furthermore, the numbers of calbindin (CB), calretinin (CR) or parvalbumin (PV) immunopositive neurons contacted by PHA-L labelled en passant and terminal boutons were much lower in most of the above areas in epileptic mice. The number of CTB retrogradely-labelled neurons was also significantly reduced in the basal lateral amygdala, perirhinal cortex and subiculum in epileptic mice. However, there was no co-localization in the above areas shown by CTB and PV, CB or CR double labelling in the control and epileptic mice.
Conclusion: These observations indicate the existence of abnormal connections between the surviving neurons in the hippocampus, perirhinal cortex or amygdala and RE in the mouse model of temporal lobe epilepsy. Therefore, our results suggest that the RE-related pathways are involved in seizure generation and propagation.

p067
Susceptibility of adult rats to pentylentetrazol induced epileptiform convulsions after fetal and infantile exposure to noise stress and hyperthermic-induced seizures
MOGHIMI A, ZOBEIRI M, MORADPOOR M, GHASSEMZADEH F, BEHNAM RASOULI M
Ferdowsi University Of Mashhad, Mashhad, Khorassan Razavi, Iran, Islamic Republic of

Purpose: Environmental factors such as noise pollution in the fetal and infantile periods and hyperthermic-induced seizures may have serious effects on epilepsy or seizures susceptibility of mature organisms. The purpose of this study was to examine the effects of hyperthermic-induced seizures and fetal/infantile exposure to noise in the onset of Pentylentetrazol induced convulsions in adulthood period.
Method: Wistar rats were used in three groups: 1st group exposed to noise during fetal and infantile lifespan until 8 days old (2 hours/day) and experienced hyperthermic-induced seizures on the 8th day of life. 2nd group only experienced hyperthermic seizures in infancy (8 days old) without noise exposure and the 3rd (control) group that kept in laboratory normal conditions. The epileptiform behaviors were recorded after IP injections of 25mg/Kg Pentylentetrazol (three times with 15 min intervals, totally 75 mg/kg). After every injections the latent periods of generalized convulsions was recorded
Results: The results revealed a significant difference in the mean latent period of epileptiform seizures onset between 1st and control ($P< 0.05$) groups. However, between 2nd and control group and also between 1st and 2nd groups was not any significant difference.
Conclusion: According to the results, hyperthermic-induced seizures along with prenatal and infantile noise exposure of rats may increase the predisposition to generalized convulsions in the adulthood period.

p068
FFT analysis on kindling-induced afterdischarge in the rabbit hippocampus
ANG YF, TSUCHIYA K, KOGURE S
Soka University, Tokyo, Japan

Purpose: Kindling is a widely used animal model of intractable temporal lobe epilepsy. To reveal the underlying mechanism of kindling-induced epileptogenesis, the duration of afterdischarge (AD), the frequency of interictal discharge (IID), and the behavioral response have been examined. In the present study, we examined FFT analysis on kindling-induced AD in the rabbit hippocampus.

POSTER ABSTRACTS

Method: Ten adult rabbits were used. Under pentobarbital anesthesia, two pairs of bipolar stainless steel electrodes were implanted in the right dorsal hippopcampus. Kindling stimulation was a train of biphasic pulses (1 ms duration each) of 50 Hz for 1 s, with a suprathreshold intensity for AD. FFT analysis on each AD was performed with sampling frequency of 1 kHz by Power Lab (Chart, ADInstrument).
Results: Out of 10 animals, 5 developed stage 5 convulsions with a mean of 18 stimulations (Kindled (K) group), whereas the remaining 5 animals did not (Incomplete kindling (IK) group). FFT analysis showed a different property between K and IK group. In IK group, the peak frequency at 3·9 Hz was characterized during early as well as late phases of kindling. In K group, the peak frequency at 3·9 Hz also appeared during early phase of kindling, but two peaks at 3·9 and 15·30 Hz appeared during late phase. The shift from one- to two-peak FFT pattern was closely related to the enhancement of behavioral responses during kindling.
Conclusion: We conclude that a relatively higher frequency discharge induced by daily kindling stimulation plays an important role to develop kindling-induced epileptogenesis.

p069
Frequency analysis on the afterdischarge induced by bilateral or alternate-site kindling of the rabbit hippocampi
TSUCHIYA K, KAKUNO M, MURAYAMA N, KONDOU T, KOGURE S
Soka University, Hachioji, Tokyo, Japan

Purpose: Kindling is a widely used animal model of intractable temporal lobe epilepsy. We have performed frequency (FFT) analysis on the afterdischarge (AD) induced by unilateral hippocampal kindling of the rabbit. In the present study, we examined FFT analysis on ADs induced by bilateral kindling (BK) or alternate-site kindling (AK) of the rabbit hippocampi to reveal the underlying mechanism of kindling-induced epileptogenesis.
Method: Twenty-six adult rabbits were used. Conventional BK and AK stimulation to both hippocampi and behavioral criteria were used. FFT analysis on each AD was performed with sampling frequency of 1 kHz by Power Lab (Chart, ADInstrument).
Results: All animals developed stage 5 convulsions. FFT analysis showed the peak frequency at 0·9 Hz during early phase of both types of kindling, but two peaks at 0·9 and 12·30 Hz during late phase. Ratio of power spectral density (PSD) of 0·9 Hz or 12·30 Hz for total PSD was calculated. Standardizing each ratio in the initial stage as 1.00, the ratio of 0·9 Hz significantly decreased to 0.39 (BK) and 0.62 (AK) while the ratio of 12·30 Hz increased to 13.08 (BK) and 3.93 (AK) ($p < 0.01$). Correlation analyses showed positive relations between alteration of higher PSD component and AD duration, interictal discharge frequency, and behavioral stage.
Conclusion: FFT analyses on BK- and AK-induced AD provided a conclusion that enhancement of higher PSD component leads to kindled stage. It is suggested that FFT analysis on stimulus-induced or spontaneous seizure discharge is useful for progression of epileptic disorders.

p070
Decreased antioxidant levels and oxidative stress in patients with epilepsy
BINDU M, RAMALINGAM K, VINOTHKUMAR R, LOKESH B
Narayana Medical College and Superspeciality Hospital, Nellore, Andhra Pradesh, India

Purpose: There has been increasing evidence supporting the association between MOS and epilepsy. This study aimed to evaluate the status of oxidants and antioxidants in patients with epilepsy (PWE).

POSTER ABSTRACTS

Methods: 100 PWE and 99 controls were included in the study. Demographic,seizure frequency, classification, duration of illness, antiepileptic (AED) medication used were evaluated in patients. Fasting blood samples were collected for Oxidants; Malondialdehyde (MDA), Nitric Oxide (NO) and Protein Carbonylsation (PC); Antioxidants (non enzymatic); reduced glutathione (RG), vitamin E (VitE), uric acid (UA); antioxidant (enzymatic); glutathione peroxidase (GPX), catalase, total antioxidant status. The group was further subdivided according to the AED type and seizure control. Data analyzed with SPSS 11.5 with t test, correlation analysis; Annova on the subgroups.p< 0.05 was taken as significant.
Results: Mean age of patient and control was 26+10and26+8; Male: female::1:1; 58% had localization related epilepsy;36% had idiopathic generalized epilepsy. Mean duration of illness was 7+6 yrs. 27%,27%,22%and 24% were on Carbamazepine, Phenytoin,Valproate and polytherapy. MDA(6.80±2.84µmol/ml), PC(0.71±0.19mg of protein/ml) in patients was higher than control MDA1.64±0.82µmol/ml)(P< 0.0001),PC(0.46±0.16mg of protein/ml)(P< 0.0001). Catalase(7.52±2.14k/ml),VitE(0.73±0.19mg/dl),GPX(74.54±12.20U/g Hb), Total Sulfhydryl groups (SH) (14.06±6.37mmol/lit), Ferric reducing antioxidant power (FRAP) (5.93±2.1437mmol/lit) was lower in patients than controls; Catalase (9.83±2.52k/ml) (P< 0.0001),VitE (1.45±0.31mg/dl)(P< 0.0001),GPX (47.78±9.49U/gHb) (P< 0.0001), (SH) (18.68±5.4837mmol/lit) (P< 0.0001), FRAP (8.51±2.4137mmol/lit) (P< 0.0001).
Catalase positively correlated with UA(r=0.22;p< 0.03); negatively cholesterol (r=-0.28;p< 0.01); LDL(r=-0.27;p< 0.01); RG negatively correlated LDL (r=-0.21;p< 0.04). GPX positively correlated with FRAP (r=0.24;p< 0.023). Annova showed NO higher in phenytoin group than carbamazepine Valproate(p< 0.03).
Conclusion: Our study showed an increased oxidant level (MDA and PC) and decreased antioxidant levels (Catalase,VitE,GPX,SH, FRAP) in patients as compared to controls indicating enhanced oxidative stress and decreased antioxidant activity. AED and seizure frequency had no effect. We conclude that PWE are under oxidative stress and free radicals may have a possible role in epilepsy.

p071
Differential DNA methylation correlates with differential expression of neuronal cell adhesion molecule in human epilepsy
WANG L, XI Z
Department of Neurology, The First Affiliated Hospital of Chongqing Medical University, Chongqing, China

Purpose: Methylation modification of DNA is an epigenetic mechanism that playsimportant roles in regulating gene expression, which is one of the key prosses underlying the funcations of neurons. But few studies focued on the abnormal DNA methylation to elucidate the differential genes expression in epilepsy. This study aimed to define the genome differential DNA methylation in the anterior temporal neocortex of drug-refractory epilepsy patients relative to control patients and to investigate the roles of abnormal DNA methylation in regulating gene expression in epilepsy.
Method: The profile of differential DNA methylation patterns were identified using six methylated-DNA immunoprecipitation-chips (3 epileptic patients vs. 3 controls); differential methylation loci were validated by bisulfite-(BS) PCR; mRNA level of corresponsing gene was evluated by quantitative RT-PCR.
Results: Through the analysis of 385k CpG island and promoter microarray, 28 differentially methylated DNA regions (18 hypermethylated CpG islands and 10 hypomethylated CpG islands) were identifed in temporal neocortex of epileptic patients. Hypermethylated CpG island of neuronal cell adhesion molecule isoform A (NRCAM) was comfirmed. Analysis of 25 samples of tempral cortex of epileptic patients and 20 controls using BSP validated NRCAM as methylated in 96% and 10%, respectively. Gene expression of NRCAM in epileptic patients was significant lower than in controls ($p < 0.05$).

POSTER ABSTRACTS

Conclusion: Our data suggest that DNA methylation changes play a causative role in the progression of epileptogenesis and NRCAM gene expression is regultated by DNA methylation in epilepsy.

p072

Can structural or functional changes following traumatic brain injury in the rat predict the epileptic outcome?

CARDAMONE L[1], BOUILLERET V[1,2], LIU YR[1], GREGOIRE MC[3], HICKS RJ[4], WILLIAMS JP[5], JONES NC[1], MYERS DE[1], O'BRIEN TJ[1]

[1]Department of Medicine (RMH), University of Melbourne, Parkville, Australia, [2]Department of Neurophysiology and Epilepsy, APHP, CHU Bicêtre, Paris, France, [3]Australian Nuclear Science and Technology Organisation, Lucas Heights, Australia, [4]Centre for Molecular Imaging, Peter MacCallum Cancer Centre, Melbourne, Australia, [5]Small Animal MRI Facility, Florey Neurosciences Institute, Parkville, Australia

Lateral fluid percussion injury (FPI), an animal model of traumatic brain injury (TBI), has been associated with the development of spontaneous recurrent seizures (SRS) and epilepsy. While structural and functional changes using MRI and PET have been shown post-FPI, the association of these changes to the development of epilepsy is unknown. Therefore, this study aimed to investigate if structural, functional and behavioural changes post-trauma relate to the development of post-traumatic epilepsy.

Rats underwent a 3.5atm lateral fluid percussion (FPI; n=31) or sham injury (n=10). Imaging and behavioural analyses were performed at 1, 3 and 6 month post-FPI followed by implantation of six sub-cortical electrodes and continuous two week video-EEG monitoring.

Of the FPI rats, 8 developed SRS, with a mean of 5.1 seizures (range 1-17) in the two week recording period. Additionally, 4 rats developed epileptic discharges, with 2-20 epileptic discharges detected. These rats were combined to form an epileptic group (n=12) and compared to the non-epileptic group (n=19). Between the epileptic and non-epileptic rats, there were no differences in structural volume (cortex, hippocampus, amygdala, ventricles, whole brain), brain metabolism or anxiety levels ($p>0.05$ for all analyses) despite differences being observed between FPI and sham rats.

This study confirms previous studies in which rats develop seizures post-FPI, however it also shows that the development of epilepsy is independent of the structural, functional and behavioural changes that result from FPI. This suggests that other mechanisms are involved in the development of post-traumatic epilepsy, with further investigations required to determine this.

p073

Experimental study on the effects of EPO、C-EPO on the protein expression of Jak2/ STAT5 and PI3K/akt in hippocampus of epilepsy rats induced by kainic acid

WANG Z, LIN WH, JIANG HY, WU SS, EPILEPSY GROUP

Jilin University, Changchun, China

Carbamylated EPO (C-EPO) is a derivative of erythropoietin (EPO), it can protect brain from injury as EPO except for stimulating erythropoiesis. The aim of this study is to observe the effects of EPO、C-EPO on the expression of Jak2/ STAT5 and PI3K/akt in hippocampus of epilepsy rats and explore the neuroprotection mechanism of both.

Methods: 2h to 24h after kindling in epilepsy and EPO group, whic120 male rats were divided into 4 groups: the control group; epilepsy group; EPO group; C-EPO Group. Epilepsy models were made by injecting kainic acid into amygdale under stereotactic instrument. Rats were decapted at 0h、2h、6h、12h、24h after epilepsy ; The expressions of Jak2/ STAT5 and PI3K/akt in the rat hippocampus were tested by the methods of western blot , and the grey values were calculated.

POSTER ABSTRACTS

Results: The protein expression of Jak2/ STAT5 increased from h in EPO group are much higher than that in epilepsy group at 24h. There are no difference of Jak2/ STAT5 expression between epilepsy and C-EPO group. The protein expression of PI3K/akt increased from 2h to 24h after kindling, which increased significantly in C-EPO group, compared with other groups.
Conclusion: EPO increase the protein expression of Jak2/ STAT5,C-EPO have no effect on the pathway of Jak2/ STAT5; C-EPO increase the protein expression of PI3K/akt, EPO have no effect on the pathway of PI3K/akt.

p074
The role of GABA neurons in the pathogenesis of epileptic FMR1 KO mice
ZENG Y, XU M, YU M, SHI Y, LONG Y, YI Y, LIAO W
Institute of Neuroscience & the Second Affiliated Hospital of Guangzhou Medical University, Guangzhou, China

Purpose: Investigate whether the number and the structure changes of GABA neurons in the *FMR-1* KO mice are susceptible to epilepsy, by comparing with the changes of GAD, GABAAR *α5* expression between *FMR-1* KO mice and WT mice hippocampus. Find whether changes of neuronal excitability in the *FMR-1* KO mice plays a critical role in epilepsy, by comparing with co-expression $Na_v1.1$ and GAD in *FMR-1* KO mice and WT mice and by detecting the electrophysiological changes of GAD neurons and pyramidal neurons in *FMR-1* KO and WT mice hippocampus.
Method: Detect the target gene and protein expression levels and do the whole-cell patch clamp recording to acutely isolated hippocampal neurons.
Results: The number of GABAergic inhibitory interneurons is reduced and the expression level of GABAAR *α5* is also down-regulated in hippocampus in *FMR-1* KO mice. The expressions of GAD and $Na_v1.1$ in *FMR-1* KO mice hippocampus are increased. Compared with wildtype mice, sodium channels excitability in pyramidal neurons in *FMR-1* KO mouse increased, sodium channels excitability in interneurons in *FMR-1* KO mouse reduced.
Conclusion: The results suggest that the reduced inhibitory of the interneurons might be one of the causes of the increased epilepsy susceptibility of *FMR-1* KO mouse, provide more evidence for that FMRP suppresses translation of target mRNAs. The results not only show higher expression of $Na_v1.1$ in pyramidal neurons might be the reason of enhanced excitability of sodium channel, but also provide electrophysiological evidence of reduced inhibitory of interneurons in *FMR-1* KO mice increase susceptibility to epilepsy.

p075
Linking the brain and heart: alterations in cardiac function and HCN channel expression in genetically epileptic rats
NG C[1], URMALIYA V[2], POWELL KL[1], KENNARD JT[1], JONES NC[1], MEGATIA I[1], REID CA[3], PINAULT D[4], WHITE P[2], O'BRIEN TJ[1]
[1]University of Melbourne, Melbourne, Australia, [2]Department of Pharmaceutical Biology, Monash University, Melbourne, Australia, [3]Howard Florey Institute, University of Melbourne, Melbourne, Australia, [4]Faculté de Médecine, Université de Strasbourg, Strasbourg, France

Purpose: Epilepsy is associated with an increased risk of sudden unexplained death (SUDEP), possibly due to cardiac arrhythmias, although the precise mechanism remains unknown. Hyperpolarization-activated cyclic nucleotide-gated (HCN) channels play an important role in the generation of pacemaker activity in the brain and heart. Studies on human epilepsy patients, and in animal models, have reported alterations in HCN expression and function in the brain. Here we investigated cardiac electrophysiological properties and HCN expression in hearts of Genetic Absence Epilepsy Rats from Strasbourg (GAERS), a widely used animal model of idiopathic generalised epilepsy.

POSTER ABSTRACTS

Methods: HCN mRNA and protein expression in cardiac chambers of epileptic GAERS and Non-Epileptic Control (NEC) rats was assessed using quantitative-PCR and western blotting. Electrocardiograms were recorded in anaesthetized rats and in isolated heart preparations.
Results: Cardiac electrophysiology was significantly altered ($p < 0.05$) in-vivo in GAERS with shorter QRS duration, slower heart rate and greater standard deviation of RR intervals (indicative of cardiac dysrhythmia) compared to NEC. These findings were replicated in isolated heart preparations. HCN2 mRNA expression was significantly decreased in the right and left atria ($p < 0.01$) and left ventricle ($p < 0.05$), whereas HCN4 mRNA expression was reduced in the left ($p < 0.001$) and right ventricle ($p < 0.05$) in epileptic GAERS compared to NEC rats. HCN1 protein expression was significantly decreased in the left ventricle ($p < 0.001$).
Conclusion: These results are suggestive of a mechanistic link between alterations in ion channel expression in the heart and brain, which may contribute to the increased risk of SUDEP.

p076
A Cav3.2 T-type calcium channel mutation (R1584P) associates with altered neuronal firing pattern in the thalamic reticular nucleus (TRN) of genetic absence epilepsy rats from Strasbourg
VAN RAAY L[1], ABDULLAH ANN[1], JONES NC1, ÇARÇAK N[2], ONAT F[3], FRENCH C[1], PINAULT D4, O'BRIEN TJ[1], POWELL KL[1]
[1]The University of Melbourne, Melbourne, Australia, [2]Istanbul University, Istanbul, Turkey, [3]Marmara University, Instanbul, Turkey, [4]Université Louis Pasteur, Stransbourg, France

Purpose: Genetic Absence Epilepsy Rats from Strasbourg (GAERS), a well validated model of idiopathic generalized epilepsy, carry a gain-of-function mutation (R1584P) in the T-type calcium channel, $Ca_v3.2$, which segregates co-dominantly with seizure expression in F2 progenies derived from GAERS and non-epileptic control (NEC) rats. GAERS are resistant to the development of secondarily generalised seizures with amgydala kindling. We investigated the hypothesis that the $Ca_v3.2$(R1584P) mutation is linked with the kindling resistance and the associated changes in neuronal firing patterns in the reticular nucleus of the thalamus (TRN).
Method: F2 animals homozygous for the $Ca_v3.2$(R1584P) mutation (PP) and F2 animals null for the $Ca_v3.2$(R1584P) mutation (RR) were kindled twice daily to a maximum of 30 stimulations. Thereafter extracellular single neuronal recordings were performed in vivo under neuralept anaesthesia. The location of the recorded cells was confirmed by iontophoretic juxtacellular labelling with neurobiotin at the end of each experiment.
Results: TRN firing frequency in rats null for the $Ca_v3.2$(R1584P) mutation was significantly lower (RR: 5.1±1) than rats homozygous for the $Ca_v3.2$(R1584P) mutation (PP: 11.7±5)($p=0.03$). The kindling rate did not significantly differ between animals with the RR genotype and PP genotype ($p>0.05$).
Conclusion: These data indicate that the $Ca_v3.2$(R1584P) mutation is associated with resistance to the changes in TRN cell firing pattern that occur with amygdala kindling. This supports a role for the altered T-type calcium channel function in the TRN of GAERS in the resistance to kindling and may have implications for pathophysiology of secondary generalisation of mesial temporal lobe seizures.

p077
Ethosuximide treatment restricts epileptogenesis and alleviates behavioural co-morbidities in the GAERS model of absence epilepsy
DEZSI G[1], BLUMENFELD H[2], SALZBERG MR[1,3], O'BRIEN TJ[1,4], JONES NC[1]
[1]University of Melbourne, Parkville, Australia, [2]Yale University School of Medicine, New Haven, USA, [3]St Vincent's Hospital, Melbourne, Australia, [4]Royal Melbourne Hospital, Parkville, Australia

POSTER ABSTRACTS

Purpose: Ethosuximide (ETX) has been shown to restrict epileptogenesis in the WAG/Rij model of epilepsy. Here, we investigated whether ETX provided anti-epileptogenic activity in the GAERS model of absence epilepsy and assessed whether drug treatment alleviated the behavioral abnormalities present in this strain.
Method: GAERS (n=5) and non epileptic control (NEC, n=7) rats were treated with 300 mg/kg/day of ETX in drinking water from 3 to 22 weeks of age, when all rats were then switched to tap water until week 34. Control GAERS (n=6) and NEC rats (n=4) received tap water throughout this period. All rats underwent serial 24 hour EEG recordings for seizure expression and behavioral testing for anxiety levels.
Results: Prior to week 22, ETX-treated GAERS spent significantly less time in seizure than control (1.6 ± 0.5% vs 7.1 ± 1.1%; p=0.002). At week 34, GAERS previously receiving ETX treatment still exhibited significantly less seizure activity than controls (4.2 ± 0.4% vs 7.0 ± 0.7%; p=0.01). Anxiety levels were increased in control GAERS compared with NEC rats, but this behavioural deficit was significantly improved in ETX-treated GAERS (p=0.04).
Conclusion: ETX induces an anti-epileptogenic effect in GAERS, reducing seizure expression long after treatment has ceased. Behavioural deficits were also reduced following cessation of treatment, intimating a common causation between seizures and anxiety in this model. The ability of this drug to limit disease progression in another rat model validates the hypothesis that anti-epileptogenic therapy is achievable, and prevention of epilepsy in humans with early treatment may be possible.

p078
Supression of absence-like seizures in the rat by neuropeptide y is assosiated with increased neuronal firing in the reticular thalamus
GANDRATHI AK[1], O' BRIEN T[1], FRENCH C[1], MORRIS M[2]
[1]University of Melbourne, Department of Medicine, Melbourne, Australia, [2]Department of Pharmacology, University of New South Wales, School of Medical Sciences, Sydney, Australia

Background: Neuropeptide Y (NPY) is a 36 amino acid neurotransmitter which is associated with the number of physiological process in the brain. NPY suppress seizures in genetic rat model of absence epilepsy (GAERS), but the neuronal mechanisms for this are unknown. Thalamo-cortical circuitry plays a critical role in the generation of absence-like seizures in GAERS.
Aim: To characterize the effect of intracerebroventricular (ICV) administered NPY on neuronal firing patterns in thalamo-cortical structures in-vivo under neurolept anaesthesia.
Methods: Male GAERS aged 8-12 weeks (n=22) were studied. Under anaesthesia extradural EEG electrodes and ICV cannula were implanted. Two glass electrodes filled with neurobiotin (extracellular neuron tracer) were lowered into the brain using a micro manipulator to record action potentials from single neurons in the thalamo-cortical circuitry. While recording cells, saline (2µl) and NPY (2µl of 1.5nmol/µl) were injected ICV at different times, and at the end of the recording period cells were labelled with neurobiotin by iontophoretic process followed by histology.
Analysis: EEG and 12 reticular thalamic (TRN) cells were recorded and analysed at ictal and interictal regions by using clampfit software.
Results: A decrease was observed on EEG in % time seizures (P=0.0001) after NPY infusion. There was an increase in interictal firing of the reticular thalamic cells after the application of NPY (P=0.008) but not after saline.
Conclusion: NPY increases the firing frequency of the TRN cell and concurrently suppresses seizures. This implicates the reticular thalamus as a key structure by which NPY suppresses the seizures.

POSTER ABSTRACTS

p079

Viral therapy for idiopathic generalised epilepsy: neuropeptide Y overexpression in ventrolateral thalamus suppresses absence-like seizures in a genetic rat model

JOVANOVSKA V[1], KRISHNASAMY N[1], VAN RAAY L[1], BLAND R[2], DURING MJ[3], O'BRIEN TJ[1,4], MORRIS MJ[5]

[1]University of Melbourne, Parkville, Australia, [2]Neurologix Research Inc, New York, USA, [3]Weill Medical College, New York, USA, [4]Royal Melbourne Hospital, Parkville, Australia, [5]University of New South Wales, Sydney, Australia

Purpose: Studies have shown that overexpression of neuropeptide Y (NPY) in the hippocampus can inhibit seizures and epileptogenesis in limbic epilepsy models. This study investigated whether chronic delivery of NPY, using recombinant adeno-associated viral vector (rAAV) could produce sustained seizure suppression in GAERS (Genetic Absence Epilepsy Rats from Strasbourg) rat model of idiopathic generalised epilepsy, and on the associated anxiety related behaviours.
Method: Male GAERS, aged 8-9 (short term cohort, n=14) and 7 (long term cohort, n=8) weeks of age were injected bilaterally with rAAV-NPY or rAAV-empty/saline in ventrolateral thalamus. A 90min EEG recording was performed weekly post-treatment and seizure expression quantified (4 and 11 weeks in short and long term cohorts, respectively). Anxiety level was tested using elevated plus maze. The pattern and intensity of NPY staining was examined postmortem by immunohistochemistry and immunofluorescence.
Results: Markedly reduced percentage time spent in seizure, duration and number of seizures were observed in both rAAV-NPY treated cohorts compared to controls. These were sustained throughout the observation period (repeated measures ANOVA, $p < 0.0001$). Intense NPY overexpression was observed in rAAV-NPY injected rats compared to controls in both, short and long term (t-test, $p < 0.05$). There was no effect observed on anxiety behaviour.
Conclusion: Focal neuronal over-expression of NPY in the ventrolateral thalamus resulted in sustained reduction in seizure expression in GAERS demonstrating that NPY has a sustained anti-epileptic effect in generalised, thalamocortical-based epilepsies. Targeted viral vector-mediated thalamic NPY overexpression may represent a novel approach for the treatment of patients with absence epilepsies.

p080

Seizure control with very high frequency electrical stimulation in the GAERS model of epilepsy

NELSON T[1], SUHR C[1], COOK M[2]

[1]The Bionic Ear Institute, East Melbourne, Australia, [2]St Vincent's Hospital Melbourne, Fitzroy, Australia

Purpose: Research was conducted to evaluate the therapeutic effect of a range of novel electrical stimulation waveforms when applied to spontaneous seizures in the GAERS model of epilepsy.
Method: Female GAERS rats were implanted with two parallel, contralateral pairs of stainless steel screw electrodes that ran in the antero-posterior direction along the length of the sensorimotor areas. A set of four EEG features were used to detect seizures and varying electrical stimuli were delivered based on these detections. The therapeutic stimuli were varying combinations of standard (130Hz) and high frequency (500Hz), short (300us) and long (1000us) pulse width and temporality (aperiodic and asynchronous, Ap/As or periodic and synchronous, P/S). Seizure lengths as determined by electrographic measures were used to determine the therepeutic benefit of each stimulation type.
Results: Using this model, seizures were reliably detected with very short detection latencies, which allowed for rapid delivery of therapeutic stimuli. The seizure terminating efficacy of stimuli was affected by frequency, pulse width and temporality. The combination of long pulse width, high frequency and Ap/As stimulation type led to the most significant reduction in seizure duration (median = 2.7s) when compared to both low frequency, short pulse width, P/S events (median = 8.8s) and non-stimulated events (median = 9.5s).

POSTER ABSTRACTS

Conclusion: Further studies are warranted to understand the mechanisms by which this therapeutic effect is achieved, and which of the novel aspects of the stimulation strategies may have contributed to the improvement in seizure abatement performance when compared to standard electrical stimulation approaches.

p081
Environmental enrichment leads to delayed onset of limbic epilepsy, improved neuropsychiatric behavioural profile and neuroanatomical alterations
YANG M[1], SALZBERG MR[2], REES SM[3], O'BRIEN TJ[1], JONES NC[1]
[1]Department of Medicine (Royal Melbourne Hospital), University of Melbourne, Melbourne, Australia, [2]Department of Psychiatry, St Vincent's Hospital, Melbourne, Australia, [3]Department of Anatomy and Cell Biology, University of Melbourne, Melbourne, Australia

Purpose: Temporal lobe epilepsy (TLE) is commonly accompanied by neuropsychiatric and neurocognitive comorbidities. Recent animal studies have highlighted enhanced seizure susceptibility, mood disturbances and neurocognitive impairments following stress. We therefore hypothesized that positive experiences, in the form of environmental enrichment (EE), may have neuroprotective, anti-epileptic and psychoprotective effects.
Method: At weaning, male Wistar rats were randomly allocated into either EE (large plastic cages containing running wheels and toys) or Impoverished Housing (IH; standard laboratory cages). At P63, a bipolar electrode was implanted into the left amygdala, followed by rapid amygdala kindling until rats experienced five class V seizures (fully-kindled). The Elevated Plus Maze (EPM) and Morris Water Maze (MWM) behavioural tests were conducted to assess anxiety and spatial learning. Animals were then transcardially perfused,brains removed and processed for histological analysis.
Results: EE delayed the time-course of seizure progression, with enriched rats (n=18) requiring a significantly greater number of kindling stimulations to reach a fully-kindled state compared to IH rats (n=19; $p< 0.05$). EE also reduced anxiety-like behaviour in the EPM (EE: n=43, IH: n=39; $p< 0.05$) and facilitated superior performance in the MWM (EE: n=32, IH: n=33; $p< 0.05$). Preliminary histological analysis revealed a trend for EE to increase the number of hippocampal dentate granule cells (EE: n=10, IH: n=10; $p< 0.10$).
Conclusion: Our data demonstrates a beneficial effect of prolonged EE on vulnerability to limbic epilepsy, co-morbid anxiety and neurocognitive function, all of which may be mediated by a neuroprotective effect of EE at the structural level.

p082
Experimental study of lamotrigine intervention for epileptogenesis in the model of post-traumatic epilepsy
SUN MZ[1] , GUO ZZ[1] , WANG M[1] , VAN LUIJTELAAR ELJM[2] ([1]China, [2]Netherlands)
[1]Department of Neurology, The First Hospital of Shanxi Medical University, Taiyuan, China, [2]DCC-Biological Psychology, Radboud Nijmegen University, Nijmegen, Netherlands

Objetives: We observed characteristics of seizures and pathology changes in rat brain after administration of Lamotrigine(LTG) at different time and dosage.
Materials and methods: An animal model of Post-traumatic Epilepsy (PTE) was established with FeCl3 aqueous solution injection into the sensorimotor cortex of rat. 56 adult male Sprague-Dawley rats were randomly divided into 7 groups: normal control group; model control group; LTG low (20mg/kg,po), middle (40mg/kg,po) and high-dose (60mg/kg)prevention group; LTG low(20mg/kg), medium-dose(40mg/kg) treatment group. In each group, acute-onset latency, degree and frequency of seizures were observed. Acute and chronic EEG by cortical electrodes was recorded. Neuron loss, gliosis and dentate gyrus mossy fibre sprouting(MFS) in ipsilateral hippocampi were studied by Nissl staining, GFAP immunohistochemical analysis and Timm staining,respectively.

POSTER ABSTRACTS

Results: (1)Injected $FeCl_3$, EEG showed paroxysmal or sustained release of epileptic waves after a latency of 376.14 ± 18.78 seconds.The most highest amplitude was up to 600mv. At the same time behavioral changes of epilepsy were observed.Latencies in prophylactic LTG groups were all prolonged compared with model control group and therapeutic LTG groups in same dose($p< 0.05$),while latencies in mid-dose prevention group and high-dose prevention group were of no significant difference($P>0.05$).(2)The total number of Spontaneous Recurrent Seizures (SRS) in 7-14d after modeling in LTG groups was lower than in model group($p< 0.05$). The number of SRS in low-dose LTG prophylactic group was fewer than in low-dose LTG therapeutic group ($p< 0.05$). But, there was no significant difference between LTG prophylactic and therapeutic group ($p>0.05$) in middle dose level. 3)Pathology study of ipsilateral hippocampi 2 weeks after modeling indicated that neuron loss and gliosis in model group was remarkable decreased than normal group($p< 0.05$). The Neuron loss and Gliosis in LTG prophylactic groups were lower significantly than in same dose LTG therapeutic groups ($p< 0.05$). MFS in dentate gyrus was significantly reduced in LTG prophylactic and therapeutic group than in model control group ($p< 0.05$). But there was no difference between prophylactic LTG groups and therapeutic groups in same dose ($p>0.05$).
Conclusions: (1) LTG prolonged incubation period and reduced SRS. Prophylactic administration LTG had more obvious effects than therapeutic administration LTG in reducing SRS. (2) Both prophylactic administration and therapeutic administration of LTG decreased nerve cell death and gliosis obviously, and prophylactic administration had more obvious effects than therapeutic administration. (3) Both prophylactic administration and therapeutic administration of LTG reduced MFS in dentate gyrus, but there was no difference between prophylactic groups and therapeutic group.(4) Mid-dose and high-dose LTG prophylactic administration had similar effects in latencies and the number of SRS. But prevention high-dose LTG administration exhibited side effects.
Keywords: Post-traumatic Epilepsy FeCl3 Animal model, Lamotrigine, Prophylaxis, Nissl staining, GFAP immunohistochemical, Timm staining

Neurobiology

p083
Inhibition mechanism of focal epileptic seizure by thumb pressing method
KUSUMASTUTI K
Airlangga University, Surabaya, Indonesia

Purpose: to investigate the resposible mechanism of pinching of the thumb on reducing partial epileptic seizure
Method: randomized pre and post test control group design for human models and completely randomized design for animal models
Results: based on t-test, burst duration and amount of epileptiform activity in human decreased with thumb pinching procedure ($p< 0.05$). In the pinching group of mice, seizure severity and duration were less compared to those in the non pinching group ($p< 0.005$). The serotonin, noradrenaline and metenkephalin containing thalamus in the pinched seizing group of mice were greater compared to those in non pinched seizing group ($p< 0.001$)
Conclusion: pinching of the thumb caused an inhibiton of partial epileptic seizures through increasing serotonin, noradrenaline and metenkephalin containing thalamus that made an inhibition sircuit.

p084
Cell recognition molecules may trigger the epileptogenesis during development in the hippocampus of the epileptic mutant EL
MURASHIMA YL, YOSHII M, INOUE N
Tokyo Metropolitan University Graduate School of Human Health Science, Arakawa-Ku, Tokyo, Japan

POSTER ABSTRACTS

Purpose: Cell recognition molecules: Neural cell adhesion molecule (NCAM), extra-cellular matrix glycoprotein tenascin-R (TN-R) are involved in the control of axon targeting, neural cell adhesion, migration and differentiation, so that the plasticity of neural circuits in the central nervous system. Polysialylation is a post-translational modification of the NCAM, which have been suggested to be functionally involved in the pathophysiology of epilepsy. Also the classic cadherins, calcium-dependent cell adhesion molecules are known to function in development in synaptic organization and stabilization. And reelin is an extra-cellular matrix protein important for neural positioning. The purpose of the present study is to examine how cell recognition molecules contribute to the epileptogenesis by using epileptic mutant EL mice.
Method: EL mice and their control animal, DDY mice were used. EL mice show secondary generalized seizures. In the interictal period during development, changes of NCAM, polysialic acid-NCAM (PSA-NCAM), Cadherin, TN-R and Reelin were investigated by Western blotting in the seizure generalization site, hippocampus.
Results: In EL mice, levels of the PSA-NCAM, Cadherin, Tenascin R and Reelin significantly increased during early developmental stages (3-7weeks) and then, decreased at 10 weeks and remain very low thereafter. The sharp withdrawal was observed before experiencing frequent seizures. In contrast, the expression of NCAM expressions showed no remarkable changes.
Conclusion: In the brain of EL, PSA-NCAM and Cadherin, Tenascin R and Reelin are upregulated before experiencing repetitive seizures, which may trigger the ictogenesis and epileptogenesis through the contribution to abnormal plastic phenomena. And NCAM may compensate the hyper-excitability during development.

p085
Initiation and propagation of epileptiform activity in hippocampl slice based on multilectrode array recording
LIU JS, GONG XW, LI JB, ZHANG PM, LIANG PJ, LU QC
Shanghai Jiao Tong University, Shanghai, China

Purpose: To investigate the network properties of epileptiform activity, as well as the pharmacological effects of antiepileptic drugs (AEDs) on the interictal-like discharges in hippocampal slices.
Method: Epileptiform bursts were induced by either raising the bath K^+ concentration or omitting Mg^{2+} from perfusion medium. Simultaneous recordings of epielptiform discharges across the hippocampal slices were obtained from multi-electrode array (MEA). The effect of sodium valproate (VPA) on epileptiform discharges was investigated.
Results: Synchronous epileptiform burst discharges appeared rhythmically in slices. Interictal-like epileptiform discharges in hippocampal slice predominantly aroused from stratum pyramidal layer of CA3a-b, then propagated bidirectionally into CA1 and CA3c region. Severing the connections between CA3 and CA1 abolished epileptiform burst in CA1 without disrupting the initiation of seizures in CA3. In high-K^+ model, speed of retrograde epileptiform field potentials (EFP) propagation (from CA3a-b to CA3c) was higher than that of antegrade one (from CA3a-b to CA1), while no difference was found in the epileptiform propagation elicited by 0-Mg^{2+}. High-K^+-induced epileptiform activity showed slower antegrade but faster retrograde propagation compared with $Mg2^+$-free model. Valproate (VPA) in a low concentration (100 μM) suppressed the propagation rather than rate or duration of high-K^+-induced population bursts.
Conclusion: Our results suggest the existence of multiple pacemakers in the CA3a-b region for the generation of interictal-like epileptiform discharges in the hippocampus, and postulate that distinct mechanisms predominate two pathways for the epileptiform propagation in the hippocampal loop. VPA at clinically relevant levels might prevent the occurrence of seizures by suppressing the propagation of interictal discharges.

POSTER ABSTRACTS

p086

Low blood glucose increases absence seizure susceptibility

KIM TH[1], REID CA[1], BERKOVIC SF[2], PETROU S[1]
[1]Florey Neuroscience Institute, Parkville, Australia, [2]University of Melbourne, Melbourne, Australia

Purpose: Absence epilepsies are a common disease with a strong genetic aetiology. Certain environmental factors can influence absence occurrence but a complete understanding of absence precipitation is lacking. Here we investigate if lowering blood glucose increases spike-wave activity in mouse models with varying seizure susceptibility.
Methods: Three mouse models were used; an absence seizure model based on the knock-in of a human $GABA_A$gamma2(R43Q) mutation (DBA(R43Q)), the spike-wave discharge (SWD)-prone DBA/2J strain, and the seizure resistant C57Bl/6 strain. Electrocorticogram recordings were made to measure SWDs from mice prior to and following injection of various doses of insulin. Blood glucose was independently measured to determine the reduction in levels following insulin injection.
Results: A ~45% reduction in blood glucose levels (6.7±0.3 mM to 4.0±0.4 mM, n=10, p< 0.05) was sufficient to double SWD occurrence in the DBA(R43Q) model (19.9±5.9 to 50.3±5.9 SWD/h, n=10, p=0.001) and in the SWD-prone DBA/2J mouse strain(1.1±0.5 to 1.8±0.4 SWD/h, n=7, p=0.01). Larger reductions in blood glucose further increased SWDs in both these models. However, even with large reductions in blood glucose no discharges were observed in the seizure-resistant C57Bl/6 mouse strain (n=6). Injection of glucose reversed the impact of insulin on SWDs in the DBA(R43Q) model (48.5±14.2 to 20.5±9.8 SWD/h, n=5, p=0.02), supporting a reduction in blood glucose as the modulating influence.
Conclusion: Low blood glucose can reduce seizure threshold in genetically predisposed animal models and should be considered as a potential environmental risk factor in absence epilepsy patients.

p087

Gender effect on the amygdala volume in temporal lobe epilepsy

PINTO ML, SILVA I, LIN K, JACKOWSKI AP, CENTENO RS, JUNIOR HC, YACUBIAN EM, AMADO D
UNIFESP, São Paulo, Brazil

Objective: To investigate gender differences on the amygdala volume in patients with temporal lobe epilepsy with mesial temporal sclerosis (TLE-MTS).
Methods: One hundred and twenty-four patients with refractory unilateral or bilateral TLE- MTS who were considered possible candidates for epilepsy surgery underwent a comprehensive pre-surgical evaluation were enrolled in this study and submitted to an MRI scan. Amygdala was manually segmented in sixty-seven women (27 right-TLE; 32 left-TLE and 8 bilateral-TLE) and 57 men (22 right-TLE, 30 left-TLE and 5 bilateral-TLE).
Results: Significant ipsilateral amygdalar volume reduction was observed for right and left TLE patients. No gender effect for the amygdala volume was observed. Contralateral amygdalar asymmetry was observed for right and left TLE patients. However, no amygdala asymmetry was observed for bilateral-TLE.
Conclusions: No gender differences were observed for the amygdala volume. Ipsilateral amygdala volume reductions observed in TLE patients might be related to differential rates of cerebral maturation between hemispheres.

POSTER ABSTRACTS

p088

Enduring effects of early life stress on neuronal firing patterns in thalamic reticular nucleus: implications for limbic epilepsy

ALI I[1], SALZBERG M[2], JONES NC[3], O'BRIEN P[3], FRENCH C[3], PINAULT D[4], MORRIS MJ[5], O'BRIEN TJ[6,7]

[1]Dept of Medicine, University of Melbourne, Melbourne, Australia, [2]Department of Psychiatry, University of Melbourne, Melbourne, Australia, [3]Department of Medicine, University of Melbourne, 4th Floor Clinical Sciences Building, Royal Melbourne Hospital, Grattan Street, Parkville, Melbourne, Australia, [4]INSERM U666, Louis Pasteur University, Faculty of Medicine, Strasbourg, France, [5]Department of Pharmacology, University of New South Wales, School of Medical Sciences, Sydney, Australia, [6]Department of Medicine, University of Melbourne, 4th Floor Clinical Sciences Building, Royal Melbourne Hospital, Grattan Street, Melbourne, Australia, [7]Department of Neurology, Royal Melbourne Hospital, Grattan Street, Parkville, Melbourne, Australia

Purpose: Early life stress (ELS) accelerates kindling induced epileptogenesis in adult male Wistar rats. Recent studies suggest critical involvement of thalamocortical circuits in kindling, in particular inducing a change in the neuronal firing patterns of thalamic reticular nucleus (TRN). We hypothesized that ELS would aggravate the changes in TRN neuronal firing patterns of amygdala kindled rats.
Method: ELS was induced using maternal separation (MS) for 3 hours daily from postnatal days 2-14, while control rats were separated (EH) for 15 minutes during the same period.
At 7 weeks of age, bipolar electrodes were implanted in the left amygdala. Kindling stimulations were delivered at after discharge threshold twice daily till 5 class V seizures were observed. Thereafter extracellular single neuronal recordings were performed in vivo under neuralept anaesthesia.
Results: There was no significant difference in TRN neuronal firing between non-kindled MS and EH rats. However, kindling led to significant reductions in interictal firing frequency and increased percentage burst firing of TRN cells compared to non-kindled rats. The changes in percentage burst firing were more pronounced in kindled MS rats as compared to kindled EH rats (30.5 ± 7.3, 14 cells, 4 MS rats Vs 12.95 ± 5.3, 10 cells, 3 EH rats P=0.08).
Conclusion: Neuroplastic changes in thalamocortical circuitries play a role in the progression of amygdala kindling, and that these are enhanced by MS. This has implications for how ELS exposure could play a role in pathophysiology of mesial temporal lobe epilepsy, in particular the development of secondary generalised seizures.

p089

The impact of tissue plasminogen activator (tPA) expression on limbic epileptogenesis in mice

NG AQ[1], SASHINDRANATH M[2], MEDCALF R[2], COOK A[1], HAMILTON J[1], O'BRIEN TJ[1], JONES NC[1]

[1]University of Melbourne, Melbourne, Australia, [2]Monash University, Melbourne, Australia

Purpose: Inflammatory mediators are implicated in temporal lobe epileptogenesis, and increasing evidence suggests that tissue plasminogen activator (tPA), an endogenous serine protease increases susceptibility to seizures. This study investigated the effects of endogenous tPA on vulnerability to limbic epilepsy utilising the mouse amygdala kindling model.
Method: Adult tPA deficient (tPA -/-) male mice and wildtype controls were implanted with a bipolar electrode into the left amygdala. Mice underwent either conventional kindling until fully kindled (five Class V seizures), or sham kindling. Brains were then excised and analysed for tPA activity using an amidolytic assay.

POSTER ABSTRACTS

Results: There was no difference in after-discharge thresholds between tPA -/- (n=8) and wildtype (n=5) mice (147±27 microamps vs. 120±25 microamps; p=0.50) or the number of stimulations required to reach fully kindled state [p=0.54]. tPA -/- mice appeared to experience shorter seizures throughout kindling, although this did not reach significance [p=0.07]. A trend for increased tPA activity was observed in the cortex of wildtype kindled mice compared to sham kindled mice. No activity was observed in tPA -/- mice. We are currently evaluating the rate of epilepsy progression in tPA over-expressing transgenic mice.
Conclusion: Genetic deletion of tPA appears to result in shorter seizures during kindling in mice. Further experiments will determine whether genetic over-expression of tPA can promote vulnerability to kindling. If tPA is found to facilitate seizures, this will have important implications for its use as treatment for acute stroke and may also lay a foundation for a novel tPA-targeted therapy for epilepsy.

p090
Rats with epilepsy induced by pilocarpine present a pineal gland lesion and reduced nocturnal melatonin levels
LIMA E[1], PERES R[1], BARATELI V[2], ROSA R[2], AFECHE S[1], CAVALHEIRO E[1], NAFFAH-MAZZACORATTI MDG[2], AMADO D[2], CIPOLLA-NETO J[1]
[1]Universidade de São Paulo - USP, São Paulo, Brazil, [2]UNIFESP, São Paulo, Brazil

Purpose: To verify that animals with epilepsy induced by pilocarpine present a pineal lesion and alterations in the nocturnal melatonin levels.
Methods: The animals were divided in 2 groups: SALINE - control animals n=33 and SE - animals that received pilocarpine and presented status epilepticus (SE) n=33. Sixty days after the SE the animals were killed in different times, with 5 animals for time at 7:00 pm; 10:00 pm; 01:00 am; 04:00 am and 7:00am and the pineal gland were removed to verify the nocturnal melatonin levels by high performance liquid chromatography (HPLC) with electrochemical detection. Another animals were killed during the day, 8 animals per group, and the pineal gland was removed to analyze pineal gland lesion by pineal dissociation.
Results: The melatonin levels were significantly lower at 4:00 am in the animals that presented SE (1.430 ng/gland p=0,001) when compared to control animals (3.231 ng/gland). Moreover, the pineal gland of the epilepticus rats present a higher cell death (14,96±0,40n°cell p=0,0001) and DNA fragmentation (4,16±0,23 DNA frag. P=0,0001) when compared to control animals (3,95±0,89 n°cell) and (1,4±0,17 DNA frag).
Conclusions: In this study we observed an important decreased of nocturnal melatonin levels in the pineal gland in animals with epilepsy and these alterations could be related with the pineal lesion found in these animals. These datas confirming our hypothesis that animals with epilepsy present a decreased nocturnal melatonin levels.
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p091
MEMRI evaluation of coriaria lactone-induced neuronal activation in rhesus hippocampus
YANG T, HONG Z, YAN B, LI H, GONG Q, ZHOU D
West China Hospital, Sichuan University, Chengdu, China

Purpose: To investigate the hippocampal neuronal activation in a Coriaria Lactone-induced rhesus model of acute seizure using manganese-enhanced magnetic resonance imaging (MEMRI) and the effect of VGCCs by diltiazem, an L-type calcium channel blocker, in the hippocampal neuronal activation .

POSTER ABSTRACTS

Method: Six rhesus macaques were divided into three groups to receive MnCl2 or Coriaria Lactone and MnCl2 or Coriaria Lactone, MnCl2 and diltiazem treatment. MnCl2 was given systemically during the course of seizure induced by Coriaria Lactone. Four hours after MnCl2 injection, T1-weighted MRI was performed followed by analysis of manganese enhancement.
Results: MEMRI studies revealed signal hyperintensity(100.305±2.564) in the hippocampus after Coriaria Lactone and MnCl2 treatment compared with Mn2+ infusion alone ($p < 0.05$. And the MEMRI signal in the hippocampus (71.342±1.727) can be attenuated by diltiazem, an L-type calcium channel blocker ($p < 0.05$).
Conclusion: It indicated that Coriaria Lactone-induced neuronal activation increased remarkably in hippocampus and the activation of glutamatergic neurons through NMDAR and VGCCs play an important role in the pathogenic mechanisms of seizure induced by Coriaria Lactone. MEMRI can be used to investigate the role of calcium channels in the neurological conditions.

p092
Precursor rhythmic neuronal activities in S2 somatosensory and insular cortices during the initiation of genetically determined absence-related spike-and-wave discharges
ZHENG TW[1,2], MORRIS MJ[3], JOVANOVSKA V[1], VAN RAAY L[1], GANDRATHI AK[1], REID CA[4], O'BRIEN TJ[1], PINAULT D[2]
[1]Department of Medicine (RMH), University of Melbourne, Parkville, Australia, [2]INSERM U666, Physiopathologie et Psychopathologie de la Schizophrénie, IFR37, Faculté de Médecine, Université de Strasbourg, Strasbourg, France, [3]Department of Pharmacology, University of New South Wales, School of Medical Sciences, Kensington, Australia, [4]Centre for Neuroscience, University of Melbourne, Parkville, Australia

The origin of bilateral synchronous spike-and-wave discharges (SWDs) that accompany absence seizures has been widely debated. Studies conducted in genetic rodent models suggest that SWDs originate from a restricted focus in the somatosensory cortex. However, the nature of this "focus" remains unclear. This study aimed to characterize the interictal, preictal and ictal neuronal activity in the primary and secondary cortical regions (S1, S2) and in the adjacent insular cortex (IC) in Genetic Absence Epilepsy Rats from Strasbourg (GAERS) using multi-site cellular and network recordings. SWDs were preceded by field potential 5-9 Hz oscillations, which were detected first in S2 and IC relative to S1. These oscillations were also recorded in non-epileptic control (NEC) rats but did not trigger SWDs. In GAERS, SWDs could be triggered following a 2-s train of 7Hz electrical stimuli with a lower current intensity in S2 than in S1. In S2 and IC, subsets of neurons displayed rhythmic firing (5-9 Hz) in between seizures. During every cycle prior to the spike component of the SW complex,
short-lasting high-frequency oscillations consistently occurred in IC ~20 ms before S1. S2 and IC layers V and VI neurons fired during the same time window, while in S1 layer VI neurons fired before layer V neurons. The present findings demonstrate the presence of precursor cellular and network rhythmic activities in S2 and IC cortices during the generation of absence-related SWDs.

p093
Further evidence of GABRG2 variants in severe myoclonic epilepsy of infancy
CHUNG S-K
School of Medicine, Swansea University, Swansea, United Kingdom

POSTER ABSTRACTS

Purpose: Severe myoclonic epilepsy of infancy (SMEI), also known as Dravet syndrome, is characterized by multiple seizure types that typically begin during the first year of life. The voltage-gated sodium channel alpha1-subunit (*SCN1A*) gene is the most commonly mutated gene accounting for over 70% of SMEI cases. Recently, mutations in gamma-aminobutyric acid (A) receptor gamma 2-subunit (*GABRG2*) gene, which is also associated with other forms of epilepsy, have been described in rare cases of SMEI. This study investigated the pathological role of *GABRG2* in SMEI patients that were gene-negative for *SCNA1* mutations.
Method: The mutation screening was performed using direct sequencing of entire exonic regions of *GABRG2*.
Results: Of the 23 patients screened, we have identified new variants in *GABRG2* including one heterozygous missense variant.
Conclusion: This study further supports the genetic heterogeneity and involvement of the GABAergic system in Dravet syndrome.

Genetics

p094
Molecular diagnosis of generalized epilepsy with febrile seizure plus (gefs+): novel mutations of SCN1A gene detected in malaysian patients
TAN EH, RAZAK SA, ABDULLAH JM, YUSOFF AAM
University Science of Malaysia, Kubang Kerian, Kelantan, Malaysia

Purpose: The aim of this study was to establish a method for detection of *SCN1A* gene mutation, to correlate with the clinical presentation and molecular findings of Malaysian GEFS+ patients.
Method: Denaturing high performance liquid chromatography (DHPLC) and direct sequencing were used to detect mutations in the *SCN1A* gene in 30 Malaysian GEFS+ patients. Genomic DNA was extracted from whole blood. Polymerase chain reaction (PCR) was carried out to amplify 26 exons of *SCN1A* based on intronic sequence. Subsequently, PCR products were analyzed by DHPLC. The samples with an aberrant profile peak from DHPLC analysis were confirmed by direct sequencing.
Results: Direct sequencing of *SCN1A* revealed twelve sequence variants, as well as 2 novel mutations were found in our patients at coding regions, c.5179A>G and c.4765A>G. The transition in exon 26 of *SCN1A* predicts a substitution Asn1733Asp in the loop between transmembrane segments S5 and S6 of the sodium channel protein. Missense mutation at c.4765A>G in exon 25 effecting a change from Histidine to Arginine at codon 1586. The compound heterozygous mutation found in patients may have cumulative effects that explained the severity of the disease which there caused GEFS+ with a heterogeneous phenotype that included mental retardation, developmental delay, ataxia, anxiety disorder and ADHD.
Conclusion: In conclusion, PCR-DHPLC approach provided a rapid, sensitive and specific screening method in the molecular diagnosis of GEFS+ and this study also suggested that mutations in *SCN1A* gene are one of the prevalent causes of GEFS+ in Malaysia.

p095
The GAT3 gene is associated with epileptogenesis
KIM YO[1], KIM MK[2], JANG SY[2]
[1]*Department of Pediatrics, Chonnam National University Medical School, Gwangju, Korea, Republic of,*
[2]*Department of Neurology, Chonnam National University Medical School, Gwangju, Korea, Republic of*

Purpose: Gamma-aminobutyric acid (GABA) is a major inhibitory neurotransmitter in the brain.

POSTER ABSTRACTS

GABAergic neurotransmission is terminated by the uptake of GABA into the presynaptic terminal and the surrounding astroglial cells by sodium-dependent transporters, such as GAT3. Therefore, the overexpression of GAT3 in the brain may cause neuronal hyperexcitability, a principal mechanism of epileptogenesis. We aimed to elucidate whether the genetic variations in the gene encoding GAT3 are associated with epileptogenesis.
Method: Five polymorphisms in coding region (cSNPs) of the GAT3 gene were genotyped for 400 patients with epilepsy and 207 controls using direct sequencing and real-time PCR, and the distribution of cSNPs was compared.
Results: The homozygous mutant allele (CC) of cSNP-GAT3E5 was distributed significantly higher in control (27.5%) than in patient group (18.3%) (p=0.009, Fisher's exact test). The relative risk (odds ratio) for susceptibility to epilepsy was 1.702 (95%CI=1.145-2.531, p=0.009) in individuals carrying homozygous wild-type (TT) or heterozygous allele (CT) compared with individuals carrying CC.
Conclusion: The mutant allele (C) of c SNP-GAT3E5 may exert a protective effect against epileptogenesis in the manner which follows a pattern of autosomal recessive inheritance. Further study for elucidating the functional value of the mutant allele is needed.

p096
Association of variants in CYP2C19 and ABCB1 with phenytoin maintenance doses in Thai patients with epilepsy
INSARO M[1], CHINVARUN Y[2], YODNOPKLAO P[3], NILAYON D[4], TANTISIRA M[1], KIJSANAYOTIN P[1]
[1]Department of Pharmacology and Physiology, Faculty of Pharmaceutical Sciences, Chulalongkorn University, Bangkok, Thailand, [2]Neurology Unit, Pramongkutklao Hospital, Bangkok, Thailand, [3]Department of Medicine, Surin Hospital, Surin, Thailand, [4]Neurology Unit, Department of Medicine, Police General Hospital, Bangkok, Thailand

Purpose: Epilepsy is one of the major neurological problems in Thailand with prevalence rate of 5.7-7.2:1000 population. Phenytoin is widely used as one of the first line drug to treat epilepsy in Thai hospitals. This study aims to investigate the association of phenytoin maintenance doses with genetic variants and non-genetic factors in Thai patients with epilepsy.
Method: Eighty six Thai epileptic patients treated with phenytoin (56% were male, mean age of 44.40 ± 14.30 years) were included in this study. Genomic DNA were used to genotype single nucleotide polymorphisms (SNPs) in genes encoding cytochrome P450 which involve in phenytoin major metabolic pathway and an efflux transporter, P-glycoprotein. Four candidate SNPs including *CYP2C19*2*(c.681G>A), *CYP2C19*3* (c.636G>A), *CYP2C9*3* (c.1075A>C), and a SNP in ATP binding cassette subfamily B (ABCB1 c.3435C>T) were genotyped by using Taqman SNP genotyping assay. Multiple linear regression statistics was used to identify the association of phenytoin maintenance doses (mg/day/kg) with genetic and non-genetic variants.
Results: The minor allele frequencies of the studied variants in Thai patients were as follows: *CYP2C19*2*=0.27, *CYP2C19*3*=0.01, *CYP2C9*3*=0.02, ABCB13435T=0.43. All genotype frequencies were consistent with Hardy-Weinberg equilibrium (P>0.05). A multiple linear regression model revealed significant association of phenytoin maintenance doses with the presence of *CYP2C19*2* and ABCB1 C3435T variants and gender. The model explain 18% of the variability in phenytoin maintenance doses (R2=0.182, p=0.037).
Conclusion: This study suggests that genetic variants in *CYP2C19* and *ABCB1* influence variability in phenytoin maintenance doses in Thai patients with epilepsy.

POSTER ABSTRACTS

p097

Base substitution spectrum of SCN1A point mutation in epilepsy

HE N, GUO J, LIAO W-P

Institute of Neuroscience and the Second Affiliated Hospital of Guangzhou Medical College; Key Laboratory of Neurogenetics and Channelopathies of Guangdong Province and the Ministry of Education of China, Guangzhou, Guangdong, China

Purpose: *SCN1A* gene that encodes voltage-gated sodium channel α-subunit of $Na_v1.1$ is currently the most clinically relevant epilepsy gene. *SCN1A* mutations have been reported in a wide spectrum of epilepsy syndromes ranged from mild generalized epilepsy with febrile seizures plus (GEFS+) to severe Dravet syndromes and their subtypes. The pattern of the *SCN1A* point mutations, such as the ratio of transition to transversion, is an important
clue to the etiopathology of mutagenesis and epilepsy, which may also provide insight into the process of genome evolution and possibly further the model of evolutionary process for the sequences. Here we aim to formulate the base substitution spectrum of *SCN1A* xzpoint mutations.
Method: We investigated the *SCN1A* mutation profile of 539 point mutation sites of total 691 patients collected from the published data and our own unpublished studies. The characteristics of the base substitution were analyzed.
Results: The transitions count for 47.9% of all point mutations, while transversions for 30.3% and deletions for 16.7%. The commonest two kinds of substitutions occurred in *SCN1A* were C to T transition (18.7%) and G to A transition (15.9%). For each type of base (A, T, C, G), transition occurred approximately 2-6 times more frequently than each kind of transversion. The ratio of substitution at GC base pair to that at AT base pair was 2.1.
Conclusion: The universal bias in favour of transition over transversion is identical in the *SCN1A* point mutation in epilepsy patients, possibly as a result of the underlying chemistry of mutation.

p098

Genetic localization of familial cortical tremor with epilepsy gene to chromosome 8q23.3-24.22

LI N[1,2], JIANG H[1,2], SHEN L[1,2], LI X[3], HUANG X[3], WANG J[1], WU Y[1], CHEN C[1], GUO J[1,2], HU Z[4], XIA K[4], ZI X[2,3], TANG B[1,2,4]

[1]Xiangya Hospital of Central South University, Changsha, China, [2]Neurodegenerative Diseases Research Center, Central South University, Changsha, China, [3]The Third Affiliated Hospital of Central South University, Changsha, China, [4]State Key Laboratory of Medical Genetics of China, Changsha, China

Purpose: Familial cortical tremor with epilepsy (FCTE) is a rare autosomal dominant idiopathic epilepsy syndrome, characterized by adult-onset fine finger tremor, infrequent seizures, neuroelectrophysiological features of cortical reflex myoclonus, good response to anticonvulsants, non-progressive course and benign prognosis. Case reports were mainly from Japan, Netherlands and France. Causative gene(s) of FCTE was not disclosed yet. We have examined the genetic conditions of one Chinese pedigree affected with FCTE to shed light on the genetics of this disorder.
Method: Detailed clinical information was collected. Linkage analysis using microsatellite genetic markers was performed firstly to examine the loci 8q23.3-q24.1, 8q24 and 2p11.1-q12.2 of BAFME, FAME and ADCME, which display similar symptoms as FCTE. When linkage to chromosome 8q was indicated, fine microsatellite markers were used to map the causative gene. Candidate genes were subsequently selected for mutation screening.
Results: Linkage analysis identified a critical region spanning 22.43 cM between markers D8S555 and D8S558 in 8q23.3-8q24.22, with a maximum two-point LOD score of 7.21 at recombination fraction θ 0.0 for marker D8S199. Potassium channel gene KCNQ3 located in the region was subjected to mutation screening, but no sequence variants were found.

POSTER ABSTRACTS

Conclusion: This is the first report of FCTE family case found in China. Genetic studies establish the presence of FCTE locus on 8q23.3-8q24.22, and exclude *KCNQ3* as causative gene of FCTE from the Chinese pedigree. The results allow us to better understand the molecular basis of FCTE and other idiopathic epilepsy syndromes.

p099

Novel mutation of SCN1A in familial generalized epilepsy with febrile seizures plus

TANG B[1,2,3], LI N[1,3], ZHANG J[4], GUO J[1,3], YAN X[1,3], XIA K[2]

[1]Xiangya Hospital of Central South University, Changsha, China, [2]State Key Laboratory of Medical Genetics of China, Changsha, China, [3]Neurodegenerative Diseases Research Center, Central South University, Changsha, China, [4]Children's Hospital of Hunan Province, Changsha, China

Purpose: Genetic epilepsy with febrile seizures plus (GEFS+) is a familial childhood-onset idiopathic syndrome with autosomal dominant inheritance, characterized by febrile seizures persisting beyond six years of age and by development of various epilepsy phenotypes with afebrile seizures in the later life. High phenotypic heterogeneity and genetic heterogeneity are observed in the GEFS+ family individuals. To date, at least six genes including *SCN1A*, *SCN1B*, *GABRG2*, *SCN2A*, *SCN9A* and *GABRD* have been identified to cause GEFS+, with the first three ones (*SCN1A*, *SCN1B* and *GABRG2*) more frequently involved in the etiology. To identify the causative genes for three Chinese pedigrees affected with GEFS+, we performed mutation analysis of *SCN1A*, *SCN1B* and *GABRG2* genes.
Method: Clinical data of three Chinese GEFS+ families was collected. Candidate genes *SCN1A*, *SCN1B* and *GABRG2* were amplified by polymerase chain reaction (PCR) and then were subjected to direct sequencing in both directions.
Results: A novel mutation c.5383G>A in *SCN1A* was detected in one of GEFS+ pedigrees. Except four single nucleotide polymorphisms (SNPs), no causative sequence variants were found in *SCN1A*, *SCN1B* or *GABRG2* for two other families.
Conclusion: The study discloses a novel mutation in *SCN1A* gene and extends its mutations spectrum related to GEFS+. In Chinese GEFS+ families, causative variants may lie in other candidate genes besides *SCN1A*, *SCN1B* or *GABRG2*, or be probably not routine point mutations.

p100

Impact of genetic polymorphisms on pharmacokinetic and pharmacodynamic pathways of valproic acid

HO J-L[1], LIOU H-H[2], HUNG C-C[1]

[1]Department of Pharmacy, College of Pharmacy, China Medical University, Taichung, Taiwan, Republic of China, [2]Pharmacology and Neurology, National Taiwan University Hospital and College of Medicine, National Taiwan University, Taipei, Taiwan, Republic of China

Purpose: To evaluate the effect of genetic polymorphisms on valproic acid serum concentration or daily dosage and better individualize the medication for patients with epilepsy.
Method: The association between genetic polymorphisms and valproic acid serum concentration or daily dosage was detected in patients with epilepsy at the department of Neurology of National Taiwan University Hospital (NTUH). After signed informed consent, 10 ml blood was drawn to extract genomic DNA. A total of 12 single nucleotide polymorphisms in 6 candidate genes were genotyped by Polymerase chain reaction (PCR)-restriction fragment length polymorphism (RFLP) and Real-time PCR SNP analysis.

POSTER ABSTRACTS

Results: There were 108 valproic acid-received patients enrolled in the study. The mean dose of valproic acid was 1228±57 mg and the mean serum concentration was 67.75±2.14 mg/L. The results demonstrated that the *FABP2* c.163G>A, GRIN2B – 200T>G polymorphisms were associated with lower effective dose of the valproic acid. On the other hand, the effective dose of the valproic acid were higher in patients with UGT1A6 c.19T>G, c.541A>G and c.552A>C polymorphisms. Moreover, patients with AA genotype in *UGT2B7* -842G>A and TT genotype in *UGT2B7* c.802C>T were with higher valproic acid serum concentration.
Conclusion: This study suggests that polymorphisms in several genes encoding for drug targets, absorptive proteins and metabolizing enzymes are associated with valproic acid treatment outcome.

p101
Having an answer: does knowing your epilepsy gene really help families?
VEARS DF[1], DUNN K[2], WAKE SA[2,3], SCHEFFER IE[1,2]
[1]Epilepsy Research Centre, The University of Melbourne, West Heidelberg, Australia, [2]Department of Paediatrics, University of Melbourne, Royal Children's Hospital, Parkville, Australia, [3]Genetic Health Services Victoria, Royal Children's Hospital, Parkville, Australia

Purpose: Research groups worldwide have identified many genes associated with epilepsy. Genetic testing has recently entered the commercial realm yet there is no information on the impact of gene identification for individuals and families with epilepsy. This study explored the experience of receiving a genetic result in people with familial epilepsy and an epilepsy gene mutation some years after receiving a positive result.
Method: Using a targeted sampling strategy, a wide selection of family members who had received a positive genetic test were invited to participate. In-depth, semi-structured interviews were conducted, audio-taped and transcribed. The data underwent thematic analysis.
Results: 20 individuals from 3 families with the epilepsy syndromes ADNFLE and GEFS+, and genes CHRNA4, GABRG2, SCN1B, were interviewed. The mean time of receiving the genetic result prior to interview was 10.9 years (range 5-14 years). Three major themes were identified: clinical utility of gene testing; 'talking about the family genes' and the impact of living with epilepsy. Participants want knowledge about their epilepsy gene, how it helps them and their families, and translation to clinical care. Their experience of epilepsy influences the impact of receiving genetic results.
Conclusions: We provide the first insights into the impact of receiving a positive genetic test on an individual in a family with epilepsy. Our results will inform the development of guidelines for genetic result disclosure, both in clinical and research settings, to improve genetic counselling for individuals and families.

p102
Coexistence gene mutation in SMEI are identified by using DNA array
SUGAWARA T[1,2], YOSHIDA S[1,3], WADA K[4], HIROSE S[5], KANEKO S[1], THE EPILEPSY GENETIC STUDY GROUP JAPAN
[1]Department of Neuropsychiatry, Hirosaki University Graduate School of Medicine, Hirosaki, Japan, [2]Research Institute of Bio-System Informatics, Tohoku Chemical Co., Ltd., Morioka, Japan, [3]Department of Integrated Human Sciences, Hamamatsu University School of Medicine, Hamamatsu, Japan, [4]Department of Disability and Health, Division of Health Sciences, Hirosaki University Graduate School of Health Sciences, Hirosaki, Japan, [5]Department of Pediatrics, Fukuoka University School of Medicine, Fukuoka, Japan

Purpose: More than 70% of SMEI patients have gene mutations of *SCN1A*. Therefore, *SCN1A* is assumed to be responsible gene of SMEI. However, it is unknown whether other gene mutations are implicated in the pathogenesis of SMEI. In this study, we analyzed gene mutations in SMEI patients to elucidate the relationship between gene mutation and SMEI.

POSTER ABSTRACTS

Method: We designed a custom resequencing array, in which 14 epilepsy genes (*SCN1A*, *SCN1B*, *CHRNA4*, *CHRNA7*, *CHRNB2*, *GABRA1*, *GABRD*, *GABRG2*, *CACNB4*, *CLCN2*, *KCNQ2*, *KCNQ3*, *CACNA1A*, *CACNA1H*) were tiled, and we developed DNA array (prototype) for genetic diagnosis of epilepsy. We investigated mutations of epileptic gene in SMEI patients by using this DNA array.
Results: We found several missense mutations not only SCN1A but other mutations (*CHARNA4*, *CACNA1H*) in about 10% of SMEI patients. These mutations were not found from 37 control (not epilepsy) samples.
Conclusion: It is possible that coexistence gene mutations are also implicated in the pathogenesis of SMEI. Because of small number of control samples, significance of these mutations in the pathogenesis of epilepsy is yet to be clarified. But novel mutations can be identified with the DNA array in epilepsy patients.

p103
Severe infantile multi-focal epilepsy: a focal epileptic encephalopathy due to SCN1A mutations
BELLOWS ST[1], ARSOV T[1], MCMAHON JM[1], GILL D[2], DAMIANO J[1], ZUBERI SM[3], MULLEY JC[4,5], BERKOVIC SF[1], SCHEFFER IE[1,6]
[1]Epilepsy Research Centre and Department of Medicine (Neurology), University of Melbourne, Austin Health, Melbourne, Australia, [2]The Children's Hospital at Westmead, Sydney, Australia, [3]Fraser of Allander Neurosciences Unit, Royal Hospital for Sick Children, Glasgow, United Kingdom, [4]Epilepsy Research Program, SA Pathology at Women's and Children's Hospital, Adelaide, Australia, [5]School of Molecular and Biomedical Sciences, University of Adelaide, Adelaide, Australia, [6]Department of Paediatrics, University of Melbourne, Royal Children's Hospital, Melbourne, Australia

Purpose: To confirm that severe infantile multifocal epilepsy (SIMFE) is a recognizable electroclinical syndrome due to sodium channel gene *SCN1A* mutations in a significant proportion of cases. In our large study of infantile-onset epileptic encephalopathies, we identified a new genetic focal epileptic encephalopathy SIMFE (Harkin et al. Brain 2007;130:843-852). SIMFE is characterised by multifocal seizures, multifocal epileptiform discharges and later cognitive slowing. 3/5 cases had *SCN1A* mutations.
Method: Electroclinical characterization of patients with SIMFE. All *SCN1A* exons and exon-intron junctions were amplified from genomic DNA by PCR and sequenced in both directions using the ABI3730XL sequencing platform.
Results: 4 new patients with SIMFE were identified. All had multiple focal seizure types with varying semiology. Mean age of seizure onset was 9.5 months (5-19 months). Developmental regression or plateau occurred later than seizure onset (1.5-8 years). Cognitive ability ranged from severe intellectual disability to low average intellect. EEG studies typically showed multifocal epileptiform activity with no (or exceptional) generalised spike wave discharges. Brain imaging findings were normal (3) or mild ventriculomegaly (1). 3/4 (75%) had SCN1A mutations: 1 de novo missense, 1 truncation and 1 splice site mutation. Parental DNA was unavailable in 2 cases.
Conclusion: 6/9 (67%) patients with SIMFE have *SCN1A* mutations. These findings confirm the distinctive phenotype of SIMFE is due to *SCN1A* mutations in a high proportion of cases. *SCN1A* mutations are associated with focal seizures of mild type (temporal and occipital seizures) and also with a focal epileptic encephalopathy, SIMFE.

p104
Acetylcholine receptor gene alpha 7 (CHRNA7) in idiopathic generalised epilepsy (IGE): mutation detection
DAMIANO JA, BERKOVIC SF, LAWRENCE KM, MULLEN SA, ARSOV T
Epilepsy Research Centre, Department of Medicine, The University of Melbourne, Austin Health, Heidelberg, Australia

POSTER ABSTRACTS

Purpose: Alpha-7-nicotinic-acetylcholine receptor (CHRNA7) is one of five acetylcholine receptors expressed in the brain. Given that two of these receptors have previously been implicated in epilepsy, and that CHRNA7 is deleted in 1% of patients with IGE (carrying the 15q13.3 microdeletion), this gene is considered to be a strong candidate gene for IGE. Previous studies have reported CHRNA7 variants in Juvenile Myoclonic Epilepsy and schizophrenia. Here we present results from our screen of patients with IGE for mutations in CHRNA7 and its partial duplication CHRFAM7A.
Method: 92 patients with IGE and 92 control DNA samples were screened. CHRNA7 exons 1-4 were bi-directionally sequenced and exons 5 -10 were initially screened for mutations with DHPLC and samples with variant denaturing profiles were subsequently sequenced. Long range PCR specific for CHRNA7 exons 5-10 was used to assign the mutations to either CHRNA7 or CHRFAM7A.
Results: We identified one missense mutation (Gly234Ser) in CHRNA7 in a patient with generalised tonic-clonic seizures alone with seizure onset at 23 years and schizophrenia. The second mutation (Val161Leu), assigned to CHRFAM7A, was found in a patient with IGE with absence seizures from 13 and generalized tonic-clonic seizures from 18 years. Both mutations affect highly conserved amino acid residues and were not found in 184 control chromosomes.
Conclusion: Our findings support the view that mutations in CHRNA7 and CHRFAM7A may be relevant to IGE and could be relevant to the mechanism in cases with 15q13.3 microdeletion. Further studies are necessary to confirm the functional significance of these findings.

p105
Epilepsy-migraine syndrome due to a missense mutation in SCN1A
OLIVER K[1], ARSOV T[1], KIVITY S[2], AFAWI Z[3], DAMIANO J[1], KORCZYN A[4], BERKOVIC S[1]
[1]Epilepsy Research Centre, Department of Medicine, University of Melbourne, Austin Health, Heidelberg West, Australia, [2]Epilepsy Unit, Schneider Children's Medical Center of Israel, Petach Tikvah, Israel, [3]Department of Neurology, Tel-Aviv Sourasky Medical Center, Tel-Aviv, Israel, [4]Sieratzki Chair of Neurology, Tel-Aviv University, Tel-Aviv, Israel

Objective: SCN1A is a well-known epilepsy gene, especially within the genetic epilepsy with febrile seizures plus(GEFS+) spectrum. More recently *SCN1A* has been identified as the third familial hemiplegic migraine (FHM) gene. Here we describe an *SCN1A* mutation positive family with a unique epilepsy-migraine syndrome.
Method: This Ashkenazi Jewish family was ascertained as part of an ongoing genetic study into epilepsy. Detailed clinical histories were taken from family members. Genomic DNA was extracted from blood samples and *SCN1A* exons and exon-intron junctions were amplified by PCR. PCR products were sequenced in both directions using the ABI3730XL sequencing platform and analysed using mutation detection software (Codon Code).
Results: The pedigree was consistent with autosomal dominant inheritance. Affected individuals had epilepsy (n=1), migraine (n=1) or both (n=5). Seizures began with a numb or tingling feeling in the hand evolving to a tonic-clonic seizure. Onset age ranged from 8-15 years. No febrile convulsions were reported and interictal EEG recordings were normal. Six family members described separate classical migraine attacks; none fulfilled the International Headache Classification (ICHD - II) criteria for FHM.
Sequencing identified a novel heterozygous missense *SCN1A* mutation, 5674C>G(Arg1892Gly), in the proband and subsequently in her two affected sons.
Conclusion: Previously one *SCN1A* mutation positive family with co-occurring FHM and epilepsy has been reported. The migraine described in this family does not meet FHM criteria and the focal epilepsy reported is unlike those usually associated with *SCN1A*. This family broadens the phenotypes associated with *SCN1A* and further supports the idea of a molecular link between epilepsy and migraine.

POSTER ABSTRACTS

p106

Familial adult myoclonic epilepsy (FAME): recognition of mild phenotypes and refinement of the 2q locus

CROMPTON DE[1,2], LAWRENCE KM[1], BELLOWS ST[1], SADLEIR LG[3], BAHLO M[4], HARTY R[3], DUNNE JW[5], ARSOV T[1], BERKOVIC SF[1], SCHEFFER IE[1,6]

[1]Epilepsy Research Centre, Department of Medicine, University of Melbourne, Austin Health, Heidelberg West, Australia, [2]Institute of Human Genetics, University of Newcastle upon Tyne, Newcastle, United Kingdom, [3]Department of Paediatrics, Wellington School of Medicine, University of Otago, Wellington, New Zealand, [4]Walter and Eliza Hall Institute of Medical Research, Melbourne, Australia, [5]W.A. Adult Epilepsy Centre, Royal Perth Hospital, Perth, Australia, [6]Department of Paediatrics, University of Melbourne, Royal Children's Hospital, Parkville, Australia

Purpose: Familial Adult Myoclonic Epilepsy (FAME) is a dominant syndrome of cortical tremor, myoclonus and epilepsy. Considerable clinical heterogeneity exists. FAME loci have been identified on chromosomes 2, 5 and 8, but the underlying genes remain unknown. We undertook a family study to further delineate the FAME phenotype and refine the genetic locus.
Method: A New Zealand/Australian FAME family was ascertained. Structured clinical histories, neurological examinations and neurophysiological data were obtained, together with DNA samples. Linkage was performed using a 250K SNP platform (Affymetrix), and refined with microsatellite markers.
Results: 55 affected individuals were studied. Postural hand tremor typically began in adolescence (range 4·60 years, median 15.5). Proximal myoclonus was present in 44/55 (80%) of individuals (median onset 17 yrs, range 5·60). Tremor and myoclonus were slowly progressive, but usually caused only minor disability.
Generalized tonic-clonic seizures (GTCS) occurred in 8/55 (15%) individuals (range 18·76 yrs, median 43.5). Epileptiform EEG abnormalities were rare (2/36, 5%). Somatosensory evoked potentials and EEG-EMG back averaging confirmed features of cortical reflex myoclonus. Linkage analysis demonstrates mapping to *FAME2* (2p11.1·2q12.2) narrowing the 2p locus.
Conclusion: Significantly fewer individuals with GTCS or epileptiform EEG abnormalities were seen, compared to previous *FAME2* families. Most such families were studied because multiple members had GTCS or partial onset seizures, potentially introducing ascertainment bias. In our family, we find a milder disorder, often resembling essential tremor. This may more faithfully reflect the usual *FAME2* phenotype. Gene identification is ongoing.

p107

The contribution of SCARB2 and PRICKLE1 gene mutations in progressive myoclonus epilepsies

DIBBENS LM[1], BAYLY MA[1], ANDERMANN E[2], VEARS DF3, MACPHERSON J[4], ANDERMANN F[2], COLE AJ[5], LEACH JP[6], GORRIE G[6], MICHELI F[7], LEHESJOKI A·E[8], BERKOVIC SF[3]

[1]Epilepsy Research Program, SA Pathology at the Womens and Childrens Hospital, North Adelaide, Australia, [2]Neurogenetics Institute, Montreal Neurological Hospital and Institute, Montreal, Canada, [3]Epilepsy Research Centre and Department of Medicine (Neurology), University of Melbourne, Austin Health Centre, Melbourne, Australia, [4]Wessex Regional Genetics Laboratory, Salisbury, United Kingdom, 5Massachusetts General Hospital Epilepsy Service and Department of Neurology, Harvard Medical School, Boston, Canada, [6]Department of Neurology, Southern General Hospital, Glasgow, United Kingdom, [7]Instituto de Neurosciencias, Hospital de Clinicas Jose de San Martin, Buenos Aires, Argentina, [8]Folkhalsan Institute of Genetics, Department of Medical Genetics and Neuroscience Centre, University of Helsinki, Helsinki, Finland

POSTER ABSTRACTS

Purpose: Progressive myoclonus epilepsies (PME) are a group of rare disorders characterized by disabling myoclonus, seizures and ataxia. Two broad groups are seen: (1) those associated with severe dementia and (2) those in which intellect is essentially preserved. The latter group includes Action Myoclonus Renal Failure (AMRF) syndrome in which patients also have renal complications. Mutations in the lysosomal *SCARB2* gene cause AMRF and also cause PME without renal failure. Mutation of the *PRICKLE1* gene is another recently identified cause of PME. We sought to determine the frequency of *SCARB2* and *PRICKLE1* mutations in PMEs.
Method: Our cohort of unsolved PMEs includes 6 patients with a phenotype suggesting AMRF. We analysed the patients for *SCARB2* and *PRICKLE1* gene variation by high resolution melt curve analysis and variants were confirmed by sequencing.
Results: To date we identified *SCARB2* mutations in 4 of the 6 suspected AMRF cases, including an Argentinian family with 4 affected children. One further case with a *SCARB2* mutation but with normal renal function was found. Cases of French Canadian and Scottish origin were each found to contain a founder *SCARB2* mutation causing AMRF. We have not yet identified any further *PRICKLE1* mutations in cases of PME.
Conclusion: *SCARB2* mutations in PME cases without renal complications are not rare. The likelihood of identifying a *SCARB2* mutation in a patient presenting with PME and renal complications is very high. No further *PRICLKE1* mutations have been found in PME patients, suggesting they are a rare cause of PME.

p108
Mutation of channel SCN3A in a patient with severe myoclonic epilepsy of infancy and autism
LI B-M, WANG P, SHI Y-W, YU M-J, DENG W-Y, GAO M-M, LIU X-R, DENG Y-H, YI Y-H, LIAO W-P
Institute of Neurosciences, Guangzhou Medical College, Guangzhou, China

Purpose: Mutations in the sodium channel genes *SCN1A* and *SCN2A* have been identified in epilepsy with febrile seizures plus, but *SCN3A* has not previously been investigated as a candidate gene for epilepsy. To explore the possible relationship between the *SCN3A* gene and epilepsy with febrile seizures plus. The *SCN3A* gene was screened for mutations in 15 patients with epilepsy with febrile seizures plus and mental developing retardation.
Method: The information of clinical manifestation, EEG, brain imagin, psychiatric develpoment of these 15 patients were collected and analyzed. DNA was extracted from peripheral blood of these patients. All 26 coding exons of *SCN3A* were amplified by polymerase chain reaction(PCR). We directly sequenced the PCR products. The novel mutational sites were verified by setting up the neurological normal controls (80 cases).
Results: Missence coding variant c.3282C>A(p.D998E) was identified in a patient, analysis of DNA from both his parents demonstrated that the *SCN3A* variant was inherited from the patient´s father, who is also heterozgyous. The novel coding variant was not present in 80 neurological normal controls. This patient was diagnosed with SMEI according to criteria of the International League against Epilepsy (ILAE) and autism according to DSM-IV and ICD-10, he had mental developing retardation, but his father has no history of seizures and is develpomentally normal.
Conclusion: *SCN3A* mutations might contribute to epilepsy with febrile seizures plus. It is necessary to screen *SCN3A* exon in mentally retarded patients of epilepsy with febrile seizures plus.

POSTER ABSTRACTS

p109

PCDH19 mutations cause epilepsy and mental retardation in females (EFMR)

DESAI T[1], DIBBENS LM[2,3], BAYLY MA[2], ARSOV T[1], KNEEN R[4], GIBBS J[5], SISODIYA S[6], GECZ J[2,3], MCKENZIE F[7,8], BERKOVIC SF[1], MULLEY JC[3], VON SPICZAK S[9], RONAN A[7,8], SCHEFFER IE[1]
[1]The University of Melbourne, Melbourne, Australia, [2]SA Pathology at Women's and Children's Hospital, Adelaide, Australia, [3]University of Adelaide, Adelaide, Australia, [4]Alder Hey Children's NHS Foundation Trust, Liverpool, United Kingdom, [5]The Countess of Chester Health Park, Chester, United Kingdom, [6]UCL Institute of Neurology, London, United Kingdom, [7]Hunter Genetics, Newcastle, Australia, [8]University of Newcastle, Newcastle, Australia, [9]University Medical Center, Kiel, Germany

Purpose: Epilepsy and mental retardation limited to females (EFMR) is a disorder that occurs in both familial and sporadic cases and is caused by mutations in the protocadherin 19 gene (*PCDH19*). We sought to determine the frequency of *PCDH19* mutations in girls presenting with seizures before 3 years of age who also had developmental delay and/or intellectual disability.
Method: We studied 119 unrelated female patients. We analysed *PCDH19* gene variation by high resolution melt curve analysis and variants were confirmed by sequencing. Where available, we analysed the inheritance of the mutation in relatives.
Results: We identified *PCDH19* mutations in 3/119 unrelated females: 2 missense mutations and 1 frameshift mutation. Mean age of seizure onset in the girls with EFMR was 9.3 months (3-17 months). The predominant pattern was of clusters of seizures, often triggered by fever. Seizure types included febrile, tonic, tonic-clonic seizures. Status epilepticus occurred in one girl at 4 years. Cognitive ability ranged from mild to moderate intellectual disability. Autistic features and behavioural problems were present in 2 patients. The two patients with missense mutations both have severely affected sisters who share their *PCDH19* mutations.
Conclusion: Approximately 2.5% of females who experience seizures in infancy with a history of intellectual disability have a mutation in the *PCDH19* gene. The presentation is characteristic and can be distinguished from Dravet syndrome. The finding of 2 sister pairs highlights the importance of *PCDH19* mutational analysis when sisters present with early onset seizures and developmental delay.

p110

Epilepsy with occipital features due to SCN1A missense mutations

ARSOV T[1], KIVITY S[2], DAMIANO J[1], AFAWI Z[3], OLIVER K[1], MCMAHON J[1], LAWRENCE K[1], SCHEFFER IE[1], BERKOVIC SF[1]
[1]University of Melbourne, Department of Medicine AH/NH, Heidelberg, Australia, [2]Epilepsy Unit, Schneider Children's Medical Center of Israel, Petach Tikvah, Israel, [3]Department of Neurology, Tel Aviv Sourasky Medical Center, Tel Aviv, Israel

Purpose: Mutations in SCN1A are an important cause of Dravet syndrome and genetic (generalized) epilepsy with febrile seizures plus (GEFS+). With increasing recognition that SCN1A may cause focal seizures and association with rare cases of benign occipital epilepsy of childhood, we studied a cohort of patients with occipital seizures.
Method: We screened 40 unrelated patients with epilepsy with occipital features. All SCN1A exons and exon-intron junctions were amplified by PCR and sequenced using the ABI3730XL sequencing platform.

POSTER ABSTRACTS

Results: SCN1A mutations were found in 2/40 cases. A de novo missense mutation Ala1429Val occurred in a patient with febrile seizures (onset 6 months) and focal seizures (onset 13 years) characterized by head and eye deviation before secondarily generalizing, triggered by flashing lights. Definitive epileptiform EEG abnormalities were not found. The second case was an inherited Lys876Thr mutation in a large Arab-Israeli family with GEFS+. The screened individual had a history of FS from age 12 months, with a pattern of benign partial epilepsy of childhood from 6 years and EEG showing occipital-parietal slowing. Of the 15 other affected family members with Lys876Thr mutation, two had additional occipital features including nausea and vomiting.
Conclusion: We confirm that SCN1A can cause epilepsy with occipital features. Both our cases and earlier reports had febrile seizures, highlighting the link between GEFS+ and benign occipital epilepsies, both in sporadic and familial cases. This observation is of diagnostic and counselling importance and highlights the overlap of generalized and focal epilepsies that have traditionally been separated.

p111
Frequencies of the polymorphisms in ABCB1 and association of these polymorphisms with multidrug-resistant epilepsy
HONG Z, YUE L, ZHU G, YU P, WU X
Institute of Neurology, Huashan Hospital, Fudan University, Shanghai, China

Purpose: To determine the frequencies of polymorphisms at C3435T and tagging SNPs in ABCB1 and to explore the association of these polymorphisms with pharmacoresistance.
Methods: We recruited patients with epilepsy who were treated with optimal AEDs. Demographic and clinical information were collected for all patients. Drug resistance was defined as four or more seizures/year despite three or more AEDs at maximally tolerated doses, while drug response was defined as seizure free for the previous year. With retrospective case-control study method, we analyzed the frequencyies of these polymorphims and association of them with multidrug-resistant epilepsy.
Results: Two hundred and eighteen people with epilepsy were recruited, of which 132 were drug-resistant, 86 drug responsive, 123 males and 95 females. Frequency of TT genotype and T allele was significantly higher in pharmacoresistant patients than that of responsive patients, while AA genotype and A allele was lower in drug-resistant patients(p=0.013, p=0.045, respectively); For rs1002204, drug-resistant patients were more likely to have TT genotype(p=0.038) and T allele(p=0.033). Compared with other haplotypes, patients with T-T haplotype had a significantly higher risk of drug resistance(OR=1.774, 95%CI:1.172-2.686,p=0.006). With multiple logistic regression analysis, rs10234411 TT genotype was an independent risk factor for pharmacoresistance. TT genotype was associated with a 4.414-fold increased risk for pharmacoresistance when compared to non-TT genotype(OR=4.414, 95% CI: 1.483-13.141,p=0.008). Besides, partial seizure and patients with more than 6 seizures before drug treatment showed an independently increased risk of drug resistance.
Conclusion: TT genotype in ABCB1 rs10234411 was significantly associated with poor AEDs response and rs1002204 TT genotype probably was correlated with drug resistance.

p112
Skewed X-inactivation may mask inheritance of a CDKL5 mutation
YENDLE SC[1], HARVEY AS[2], BIENVENU T[3], SCHEFFER IE[1,2,4]
[1]Epilepsy Research Centre, Department of Medicine, The University of Melbourne, Austin Health, Melbourne, Australia, [2]Department of Neurology, Royal Children's Hospital, Melbourne, Australia, [3]Université Paris Descartes, Institut Cochin, CNRS (UMR8104), Inserm, U1016, Paris, France, [4]Department of Paediatrics, The University of Melbourne, Royal Children's Hospital, Melbourne, Australia

POSTER ABSTRACTS

Purpose: Mutations in the X-linked cyclin-dependent kinase-like 5 (CDKL5) gene are associated with a severe encephalopathy in girls presenting with early onset refractory seizures and severe intellectual disability. CDKL5 mutations arise de novo or rarely occur in the setting of presumed gonadal mosaicism. We report the first case of skewed X chromosome inactivation of an inherited CDKL5 mutation.
Method: Detailed phenotyping was performed and CDKL5 sequenced on DNA. X-Chromosome Inactivation (XCI) analysis was studied using standard methodology for the androgen receptor locus on the DNA of peripheral blood cells of the proband and her mother.
Results: 9 year old girl with multiple seizure types and severe intellectual disability had presented at 5 months with epileptic spasms and regression. She had a novel CDKL5 missense mutation p.I508T (c.1523T>C) inherited from her unaffected mother. This amino acid Ileu 508 is strictly conserved in Rattus norvegicus and Mus musculus. XCI studies showed a random XCI pattern (RsaI/HpaII 66%:34%; RsaI/CfoI 65%:35%) in the proband. Her unaffected mother exhibited 100% skewed X inactivation.
Conclusion: The finding of an inherited CDKL5 mutation with variable X chromosome skewing is of critical importance for genetic counselling for families; the parents have a risk of a further affected daughter and the proband's normal sister has a significant risk of inheriting the 100% skewed mutation with implications for her offspring. This is the first reported case of an inherited CDKL5 mutation due to skewed XCI in a carrier mother, and shows that XCI may influence the presence of CDKL5 encephalopathy.

Translational research

p113
Coupling of cerebral blood flow and metabolism during focal hypothermia
FUJIOKA H[1], FUJII M[1], KOIZUMI H[1], NOMURA S[1], IMOTO H[1], SUEHIRO E[1], OWADA Y[2], SAITO T[3], YAMAKAWA T[4], SUZUKI M[1], CONSORTIUM OF ADVANCED EPILEPSY TREATMENT (CADET)
[1]Department of Neurosurgery, Graduate School of Medicine, Yamaguchi University School of Medicine, Yamaguchi, Japan, [2]Department of Organ Anatomy, Graduate School of Medicine, Yamaguchi University School of Medicine, Yamaguchi, Japan, [3]Applied Medical Science Engineering, Graduate School of Medicine, Yamaguchi University School of Medicine, Yamaguchi, Japan, [4]Graduate School of Life Science and Systems Engineering, Kyushu Institute of Technology, Fukuoka, Japan

Purpose: Although focal brain hypothermia has been a candidate for epilepsy treatment, the therapeutic feasibility remains to be elucidated. Of particular importance in this regard is to clarify cooling-induced neurovascular and metabolic changes. To address this issue, cerebral blood flow (CBF) measurement and microdialysis were performed during deep focal hypothermia in epileptic patients.
Method: A PID-controlled, thermoelectrically-driven cooling device (cooling area: 40×40 mm) was intraoperatively applied on the cortical epileptogenic foci in intractably epileptic patients (n=6), and CBF measurement and microdialysis were performed. Focal seizures were induced by microinjecting a penicillin solution on the eloquent area of rat SI-MI cortex (n=5), and a cooling device (6×6 mm) was implanted.
Results: Although CBF in the epileptic foci was transiently reduced during 15°C cooling, it was reversible and immediately recovered following rewarming. Cooling-induced metabolic changes were qualitatively similar across patients. Of glycolytic metabolism, lactate levels were significantly reduced ($p < 0.05$), while glucose and pyruvate levels remained relatively unaffected. Extracellular levels of glutamate were significantly decreased during cooling ($p < 0.05$) with the effect remained even after rewarming ($p < 0.05$). Cooling to 20°C significantly reduced severity of focal seizures in rats ($p < 0.05$) with minimal influence on neurological functions.

POSTER ABSTRACTS

Conclusion: It has been reported that autoregulation and metabolic coupling in epilepsy are impaired. This study showed, for the first time, coupling of CBF and metabolism with cooling to 15°C. Decreased lactate and glutamate levels suggested neuroprotective effects. Together with the behavioral investigation in animals, the results suggested a feasibility of hypothermal device-based therapy.

p114

A prospective study of the use of HLA-B*1502 genotyping in preventing carbamazepine-induced Stevens-Johnson syndrome and toxic epidermal necrolysis

CHEN P[1], SHEN C·Y[1], LIN J·J[2], ONG C·T[3], WU S·L[4], TSAI P·J[1], CHEN W·H[1], CHUNG W·H[5], HUNG S·I[6], CHANG C·F[7], CHEN C·H[1], LIN S·Y[1], CHUAN H·P[1], HUANG L·C[1], CHEN Y·J[1], CHEN Y·T[1], THE TAIWAN SJS CONSORTIUM

[1]Academia Sinica, Taipei, Taiwan, Republic of China, [2]Chu Shang Show Chwan Hospital, Chu Shang, Nan-Tou, Taiwan, Republic of China, [3]Chia-Yi Christian Hospital, Chia-Yi, Chia-Yi, Taiwan, Republic of China, [4]Changhua Christian Hospital, Changhua, Chang-Hua, Taiwan, Republic of China, [5]Department of Dermatology, Chang Gung Memorial Hospital, College of Medicine, Chang Gung University, Taipei, Taiwan, Republic of China, [6]Institute of Pharmacology, School of Medicine, National Yang-Ming University, Taipei, Taiwan, Republic of China, [7]Pharmigene, Inc., Taipei, Taiwan, Republic of China

Purpose: Carbamazepine(CBZ)-induced Stevens-Johnson syndrome(SJS) and its related disease, toxic epidermal necrolysis(TEN), are strongly associated with the human leukocyte antigen (HLA) B*1502. This study aimed at preventing CBZ-induced SJS/TEN by identifying individuals at risk using HLA-B*1502 genotyping.
Method: Patients, recruited from 26 hospitals, who had not previously received CBZ and would nomrally have been treated with CBZ, underwent prospective HLA-B*1502 screening and HLA-B*1502-positive patients were excluded from CBZ treatment. For ethical considerations, the study was designed as a nonrandomized trial and historical incidence was used as the control. The incidence of CBZ-induced SJS/TEN in the period of CBZ use without HLA-B*1502 screening was compared to that seen with prospective screening.
Results: Between January 2007 and December 2009, 4473 subjects were recruited from 26 participating hospitals in Taiwan. Screening showed that 7.8% of the patients carried the HLA-B*1502 allele and no CBZ was prescribed; instead, they were either given alternative medication or were left on their before-study medication. Of all the enrolled patients, 4.9% developed mild, transient skin rashes, but six had more wide-spread rashes and were hospitalized (final diagnosis: 3 maculopapular eruption, 2 hypersensitivity syndrome, 1 urticaria). None of the 2946 participating patients who were given CBZ developed SJS/TEN, in contrast to the estimated 7 cases of CBZ-SJS/TEN in the historical control (0.23% of CBZ users) (p value=0.0046; Fisher´s two-tailed exact test).
Conclusion: Our data suggest that application of HLA-B*1502 genotyping as a screening tool for patients taking CBZ can effectively reduce the incidence of these life-threatening adverse drug reactions.

Women and pregnancy

p115

Risk factor related with reproductive endocrinology disorder in Chinese women of child-bearing age with Epilepsy Department of Neurology

ZHOU J·Q, CHEN L·J, ZHOU L·M, CHEN Z·Y
The First Affiliated Hospital ,Sun Yat-Set University, Guangzhou, China

POSTER ABSTRACTS

Purpose: To identify the risk factors related with Reproductive endocrinology disorder in Chinese women of child-bearing age with epilepsy
Method: Collected the clinical data of 102 women with epilepsy.Grouped the patient according to seven aspects(seizure onset age,seizure type,seizure frequence,duration of epilepsy,AEDs type, age of start AEDs therapy and duration of therapy) and analyzed the contribution of these factors in development of PCOS and its components.
Results: The incidence of hyperandrogenemia in patients with an early onset age(≤14 years old) was higher than the ones with an onset age >14 years old. Onset age≤14 was the risk factor of hyperandrogenemia in Logistic regression analysis.The incidence of a/oligomenorrhea, polycystic ovaries, hyperandrogenemia and PCOS in the valproate-treated women were 40.63%,50.00%,15.65% and 34.38% respectively,which were higher than the no-therapy group and non-valproate treated group. Valproate therapy was the risk factor of PCOS and its components.
Conclusion: Valproate therapy was the risk factor of PCOS and its components in Chinese women of child-bearing age with epilepsy. Onset age≤14 was the risk factor of hyperandrogenemia.

p116
The effect of fetal valproate exposure on memory functioning in school aged children
BARTON S[1,2], NADEBAUM C[1,2], ANDERSON VA[2,3,4], VAJDA FJE[5], REUTENS D[6], WOOD AG[1,2]
[1]Monash University, Melbourne, Australia, [2]Murdoch Childrens Research Institute, Melbourne, Australia, [3]Royal Children's Hospital, Melbourne, Australia, [4]University of Melbourne, Melbourne, Australia, [5]Australian Pregnancy Register for Women with Epilepsy and Allied Disorders, Melbourne, Australia, [6]University of Queensland, Brisbane, Australia

Purpose: Women with epilepsy are usually recommended to continue taking anti-epileptic drugs (AEDs) throughout pregnancy, despite some medications being associated with elevated risk of birth defects. Valproate appears to pose a greater risk compared to other AEDs and has been associated with intellectual impairments. Animal models have shown that prenatal valproate exposure can also affect hippocampal development. This study aimed to investigate memory functioning in school-aged children who were exposed to valproate during pregnancy.
Method: One hundred and two school-aged children (aged six to eight years) exposed to AEDs *in utero* participated in the study. Children with birth malformations were excluded. Information on AED exposure was collected prospectively. Children underwent a neuropsychological assessment using standardised memory measures. Preliminary results are presented.
Results: Children exposed to valproate demonstrated significantly poorer performances on a story recall task, which remained after controlling for language and verbal intellectual skills. Relative to other AEDs, children exposed to valproate also performed more poorly on a list learning task and were more susceptible to retroactive interference. Valproate dose was negatively correlated with performance on verbal memory tasks. There was no difference between children exposed to valproate and other AEDs on tasks of visuospatial memory.
Conclusion: Preliminary findings suggest that fetal exposure to valproate has a specific impact on verbally mediated memory skills. Higher doses of valproate appear to pose a greater risk to memory functioning. The effect on retroactive interference is suggestive of mesial temporal lobe dysfunction, which should be an area for future investigation.

p117
Report on the second generation of antiepileptic drugs (AEDs) from the database of the Australian pregnancy register (APR)
VAJDA F[1], GRAHAM J[2], ROTEN A[2], LANDER C[3], O'BRIEN T[1], EADIE M[4]
[1]Dept of Medicine, University of Melbourne, Parkville, VIC, Australia, [2]Royal Melbourne Hospital, Parkville, VIC, Australia, [3]Royal Brisbane Hospital, Brisbane, QLD, Australia, [4]University of Queensland, Brisbane, QLD, Australia

POSTER ABSTRACTS

Purpose: To provide information on the efficacy and teratogenicity of new AEDs in pregnancy.
Method: Analysis of the database of the APR
Results: Since 1994 ten new AEDs have been introduced into Australia. They were all approved initially as add on medications, mainly for the control of partial epilepsy. Three of these have since been withdrawn, abandoned or used only as a last resort, felbamate, vigabatrin, fosphenytoin. Pregabalin and oxcarbazepine have been infrequently prescribed in pregnancy, thus only lamotrigine, topiramate, gabapentin and levetiracetam are suitable for evaluation. The data to be presented involves efficacy data against seizures, teratogenicity involving foetal physical malformations, in women with epilepsy who were untreated and in comparison with three traditional AEDs phenytoin, valproate and carbamazepine whose teratogenicity has been reported previously. The APR now has a database on 1500 women with epilepsy.
The data on new AEDs comprises monotherapy data for lamotrigine (262), levetiracetam (42), gabapentin (14) and topiramate (35)
On monotherapy, the incidence of malformations relevant to lamotrigine was (7), levetiracetam (0), gabapentin (0) and topiramate (0).
Conclusion: The second generation AEDS appear no more teratogenic than traditional drugs in monotherapy.
Polytherapy data are more difficult to interpret. The study continues with an extension of focus on foetal cognition and development.

p118
Status epilepticus and posterior reversible encephalopathy syndrome (PRES)
NARAYANA RV, PATI R, KUMAR S, MAHATO A
Sevenhills Hospital, Visakhapatnam, India

Purpose: Posterior reversible encephalopathy syndrome(PRES) was associated with multiple causes. It is also seen in women with pre-eclampsia/hypertension and eclampsia during pregnancy. we have seen patients with PRES -status epilepticus after cesarian section without hypertension / preeclampsia/eclampsia.
Method: In the last one year three patients were admitted with status epilepticus under obstetrician and gynaecologist and referred to neurology for evaluation.two of them underwent cesarean section under spinal anaesthesia and one patient underwent hysterectomy under spinal anasthesia .patients were evaluated for possible causes for status epilepticus.CTscan brain, MRI brain MR venography and EEG was done. Seizures treated with antiepileptic drugs.
Results: All of them presented with headache, vomting,seizures, visual disturbances and psychotic behaviour.Their neuroimaging showed bilareral subcortical whitematter edema more predominant on the posterior parieto-occipital areas consistent with PRES.their lesions resolved in two to four weeks time. None of the patients had hypertension/pre-eclampsia or eclampsia. Status epilepticus responded well to antiepileptic drugs.
Conclusion: PRES is a reversible condition Hypertension/pre-eclampsia and eclampsia are not seen and exact cause in our cases is not known we are not sure whether pregnancy itself/spinal anesthesia and surgery on the uterus is any thing to do with the brain lesions.

p119
Assessment of knowledge of women's issues and epilepsy: a survey of obstetrician in Kerala, South India
BHAT M[1], NIRMALA C[1], THOMAS SV[2]
[1]Medical College, Thiruvanthapuram, India, [2]Sree Chitra Tirunal Institute for Medical Sciences and Technology, Trivandrum, India

POSTER ABSTRACTS

Purpose: To assess the knowledge of obstetricians about the relevant topics and concerns of women with epilepsy (WWE) in Kerala state, South India. Also to compare the knowledge of obstetricians working in different hospital set-ups and to compare it with the existing literature.
Method: A total of 97 obstetricians (Teaching hospital-43, private hospital-32 and community health centres-21) were surveyed using a modified KOWIE (Knowledge of women's issues and epilepsy) questionnaire II with additional questions.
Results: 89 (91%) were well informed about the teratogenic effects of AEDs, 92 (95%) understood the need to continue AEDs during pregnancy, 95 (97%) knew the role of folic acid and vitamin K during pregnancy, 92 (95%) agreed that enzyme inducing AEDs decrease the efficacy of contraception and 91% understood the safety of breast-feeding. About 2/3rd knew that best AED during pregnancy is the one most appropriate for the seizure type. Only about 1/3rd of them understood the effects of endogenous steroid hormones on seizure threshold and fewer than half knew that AEDs cause osteomalacia. Only a few were aware of unique issues related to epilepsy and increased incidence of female sexual dysfunction (29.9%) and infertility (26.8%).
Conclusion: Majority of the obstetricians were well informed about the fetal complications of antenatal AED exposure but were relatively under informed of other complications such as osteomalacia, sexual dysfunction and infertility. An aggressive educational effort is necessary to close the gaps in knowledge. The knowledge of the doctors in India is comparable to that of developed country like USA.

p120
Early cognitive and behavioural outcomes following prenatal exposure to antiepileptic drugs
HARTMANN P[1], ANDERSON V[2], VAJDA F[3], REUTENS D[4], HOLDEN M[5], WOOD A[1,2]
[1]Monash University, Melbourne, Australia, [2]Murdoch Childrens Research Institute, Melbourne, Australia, [3]Australian Pregnancy Register for Women with Epilepsy and Allied Disorders, Melbourne, Australia, [4]University of Queensland, Brisbane, Australia, [5]University of Melbourne, Melbourne, Australia

Purpose: This study characterised cognitive and behavioural outcomes in children at one and three years of age who were exposed prenatally to antiepileptic drugs.
Method: 114 babies recruited via the Australian Pregnancy Register were assessed blind to drug using the Bayley Scales of Infant and Toddler Development (3rd Ed), with repeat neuropsychological assessment at three years of age. Maternal IQ was assessed using the Wechsler Abbreviated Scale of Intelligence. Here we present preliminary analyses of babies' data where group size was greater than ten.
Results: The average age at first assessment was 16.7 months (range=12-23). Group performance was compared with the population mean using one-sample t-tests. Exposure to valproate monotherapy (N=15) was associated with significantly reduced scores on language, motor and global adaptive behaviour scales. Language, motor and practical aspects of adaptive behaviour composite scores were reduced in babies exposed to valproate polytherapy (N=13). Lamotrigine monotherapy (N=35) exposure showed reduced motor composite scores. Carbamazepine monotherapy exposure (N=25) was associated with reduced language composite scores and practical adaptive skills. Maternal IQ did not correlate with cognitive outcomes in children.
In 23 three-year-olds seen to date, there were significant correlations between full scale IQ at age three and one-year-old performance on Bayleys language and motor scales.
Conclusion: To date there have been no data on very early identification of risk in children prenatally exposed to antiepileptic drugs. Our preliminary findings suggest it is possible to detect risk associated with later outcome. Further analyses are required to explore the contribution of potential confounds.

POSTER ABSTRACTS

Status epilepticus

p121
A case of status epilepticus in a patient with ginkgo nut intoxication
PARK H-K, YANG K-I, OH H-G
Department of Neurology, College of Medicine, SoonChunHyang University, Cheonan-si, Korea, Republic of

Purpose: Ginkgo nuts have been eaten as food since ancient times in Korea. The ginkgo nuts is also taken as an herbal medicine for its antitussive and expectorant properties although the potential toxicity of the ginkgo nut is not as well-known. Several reports indicate that consumption of ginkgo nuts might induce seizure activity.
Method: We present a case of status epilepticus after consuming a large number of ginkgo nuts.
Results: A 58-year-old man was admitted with repetitive generalized tonic-clonic seizures and prolonged unconsciousness. He had eaten approximately 300 ginkgo nuts before the onset of symptoms. There were no focal neurologic signs. Initial brain CT was normal. We diagnosed as status epilepticus and intravenous loading of phenytoin (1000 mg) was started. However, since recurrent seizure attack was occurred after 4 hours, midazolam (0.1 mg/kg/hr) was injected. Further seizure attack was not happened. His mentality recovered after 2 days of treatment. He was discharged without sequlae 10 days after admission.
Conclusion: It has been reported that 4-O-methoxypyridoxine (4-MPN) in ginkgo nuts cause seizure. 4-MPN indirectly inhibits the enzyme activity of glutamate decarboxylase, resulting in decreased levels of gamma-amino-butyric acid (GABA). And reduced concentration of GABA may lead to seizure. Therefore, gingko nuts should be used cautiously or avoided patients who might be predisposed to seizure or who are using other medicines known to provoke seizure.

p122
Neurodegenerations as etiologic factors of status epilepticus at infancy and early childhood
KHOLIN A[1,2], MIKHAILOVA S[2], IL'INA E[2], LEMESHKO I[1], VORONKOVA K[1], MUKHIN K[1], PETRUKHIN A[1]
[1]Russian State Medical University, Moscow, Russian Federation, [2]Russian Children Clinical Hospital, Moscow, Russian Federation

Purpose: Status epilepticus (SE) at infancy and early childhood is non-rare, but sometimes non-well diagnosed problem. SE in neurodegenerations is characterized by poor prognosis and high risk of aggravation by AEDs
Method: Were investigated 267 children with debut of SE before the age of three years in child neurology department of Russian Children Clinical Hospital at the period 2000-2010.
Results: At group of 267 children with debut of SE at infancy and early childhood in 15,7% of cases (n=42) neurodegenerative disorders have been diagnosed. Cases of biotinidase deficiency (n=9), Menkes disease (n=2), histidinemia (n=1), methylmalonic acidemia (n=1) и GM-1 gangliosidosis type I (n=1) caused status of serial tonic spasms. Myoclonis SE were caused by neuronal ceroid lipofuscinosis type II (n=7), Alpers disease (n=5), GM-2 gangliosidosis type II or Sandhoff disease (n=3), neuronal ceroid lipofuscinosis type I (n=2), GM-1 gangliosidosis type II (n=1), glicogenosis type III (n=1), Krabbe disease (n=1), metachromatic leukodystrophy (n=1), Canavan disease (n=1), Zellweger syndrome (n=1). Hemiconvulsive SE were seen in MELAS syndrome (n=3). One case of Tay-Sachs disease caused migrating SE of alternative tonic and hemiconvulsive, versive and pharyngo-oral, henifacial and inhibitory components. Also was identified a familial case of Kreutzfeld-Jakob disease (Met129Val mutation in PRNP) with series of tonic spasms and spike-wave stupor.
Conclusion: In cases of SE at infancy and early childhood it is very useful to obtain genetic consulting. Early diagnostic of biotinidase deficiency is very important for opportunity to favorable prognosis.

POSTER ABSTRACTS

p123

A retrospective analysis of children admitted with convulsive status epilepticus to Children's Hospital at Westmead

GUPTA S, GILL DA, DALE RC
Children's Hospital at Westmead, Westmead, Australia

Purpose: To characterize children with convulsive status epilepticus (CSE) in terms of their demographics, aetiology, morbidity and mortality.
Method: A retrospective analysis of the clinical and demographic data of 88 patients, who were admitted to our Children's Hospital, with CSE. We obtained these data from accident and emergency, ambulance/NETS and intensive care notes. We excluded the patients who presented with non convulsive status epilepticus. The period of study was from March 2002 till June 2009.
Results: A total of 88 patients presented with CSE with nine patients presenting more than once during the study period. The average age was 5.5 years with 30% being in less than 2 year age group. 48 patients had epilepsy prior to presentation. The most common etiology in our group was acute on remote symptomatic followed by remote symptomatic. Prolonged febrile seizure group was uncommon in our cohort. The commonest acute symptomatic cause was CNS infection. 76% status events required PICU admission. The mean duration of hospital stay was 13 days (range 2days-1.5yrs). There were 2 deaths during the acute phase, both in the acute symptomatic category. 6 patients died on follow up due to severe neurological disabilities. 61 patients had significant cognitive sequelae; however most had pre-existing neurological problems. The groups that fared well were prolonged febrile seizure and idiopathic epilepsy related
Conclusion: Status epilepticus is a complex heterogeneous condition with considerable morbidity and mortality. This data will help in appropriate allocation of resources to reduce the effects of this disorder in childhood.

p124

In search of neuroprotective drugs against experimentally induced status epilepticus in young rats

AHMAD M[1], AL DEEB S[2], TARIQ M[2]
[1]King Saud University, Riyadh, Saudi Arabia, [2]Riyadh Military Hospital, Riyadh, Saudi Arabia

Purpose: All currently available antiepileptic drugs do not provide satisfactory protection against seizures and associated neurodegenerative changes. Based on our pilot study of 11 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 short listed drugs were in the order PTX>PGM>QCN. High dose of PTX protected young animals against status epilepticus induced seizures and associated neurodegenerative changes.

POSTER ABSTRACTS

p125
A case of lingual epilepsia partialis continua persisted through two and a half years
CHO Y-J, LEE MK, LEE BI, HEO K
Department of Neurology, Yonsei University College of Medicine, Seoul, Korea, Republic of

Purpose: Epilepsia partialis continua (EPC) is considered as the status epilepticus equivalent of simple partial motor seizure. Every body part can be involved, but isolated lingual EPC has been rarely reported.
Method: We present a patient with continuing lingual EPC for more than two years, which was successfully controlled by surgery.
Results: A 30-year-old woman was admitted for surgical management of intractable lingual EPC lasted for two and a half years. Previously, she had been healthy except having a possible SLE which was controlled with low-dose steroid. The seizures started with secondarily GTCS following tongue and right facial clonic jerks which soon progressed to daily continuous tremulous tongue twitching. Although various antiepileptic drugs had some effect on seizures; however, she still had daily continuous tongue tremor with intermittent facial involvement, easily aggravated by eating or talking. Scalp EEGs failed to reveal any epileptiform discharge and serial brain MRIs disclosed multiple high-signal intensity lesions in the left frontal lobe on T2WI. Increased perfusion and metabolism was observed in the left opercula on ictal SPECT and FDG-PET. Intracranial video-EEG monitoring clearly demonstrated ictal discharges over left frontal operculum close to a MRI lesion, which was coincided with right facial twitching on electromyographic recording. Lesionectomy and cortisectomy under electrocorticography were performed. The pathology was focal cortical dysplasia without balloon cells (type IIa). A complete seizure freedom was achieved immediately after surgery, and remained in seizure-free state for 6 months.
Conclusion: We report a rare case of lingual EPC occurred everyday for 30 months.

Adult epileptology

p126
Convulsive syncope: diagnostic accuracy and prognosis
CHAN J, DUNNE JW, LEE JC, LAWN ND
Department of Neurology, Royal Perth Hospital, Perth, Australia

Purpose: Convulsive syncope (CS) is often misdiagnosed as an epileptic seizure. Clinical assessment is the gold standard in diagnosis. Patient outcome may be a marker of initial diagnostic accuracy.
Method: Patients at a hospital-based First Seizure Clinic were prospectively studied from 1999 to 2008. Patients with a clinical diagnosis of CS or first-ever seizure were followed-up to determine the nature of any further events. Clinical features, investigations and outcomes were compared.
Results: 273 patients with CS were compared to 1325 patients with first-ever seizure. Most were referred from the Emergency Department (67%). Causes of CS included vasovagal (91%), situational (5%), orthostatic (2%) and cardiac arrhythmia (2%). EEG was abnormal in 17 CS patients: generalised epileptiform abnormalities (2), focal slowing (9) or generalised slowing (8). Neuroimaging, performed in 81%, showed potentially epileptogenic lesions in 8 (4%). In comparison to first-ever seizure patients, CS patients were significantly more likely to be female (56% vs 39%) and younger (median 30 years vs 36 years), less likely to have epilepsy risk factors (10% vs 25%) and were just as likely to had been incontinent, had an event-related injury or have a family history of epilepsy. At one-year, recurrent syncope had occurred in 41 CS patients (15%). Only 3 CS patients (1%) re-presented with a seizure compared to 41% of first-ever seizure patients ($p< 0.001$).
Conclusion: Follow up of patients with convulsive syncope presenting to a First Seizure Clinic showed a very low rate of subsequent seizures consistent with an accurate initial clinical diagnosis.

POSTER ABSTRACTS

p127
Risk factors for the development of seizures and epilepsy among post stroke patients in a Philippine tertiary hospital: a retrospective cohort study
FABIANA NL
University of the Philippines-Philippine General Hospital, Manila, Philippines

Purpose: Stroke accounts for >50% of the newly-diagnosed cases of epilepsy, but only < 10% of all stroke patients experience seizures and epilepsy. This study aimed to determine the risk factors in developing seizure and epilepsy after stroke including age, type and location, recurrence and severity of deficits.
Method: This is a retrospective cohort study on adult post-stroke patients who developed seizure and epilepsy seen in a Philippine tertiary hospital in 2007 - 2009. Control population consisted of those who did not develop seizures and epilepsy.
Results: Seizures occurred in 5.9% of the 505 cases. All of post-stroke seizure patients developed epilepsy, majority with late onset first seizure. Most had cortical infarction and Generalized Tonic Convulsive (GTC) seizures. Most electroencephalogram (EEG) findings were focal or generalized slowing. Cortical location increases the risk of post-stroke seizure by 2.5 (p=0.52) and the development of epilepsy by 4.6% (p= 0.008). Simultaneous involvement of cortical-subcortical regions increases seizure occurrence by 3.2 times (p=0.062).Subarachnoid hemorrhage (SAH) increases risk of seizure by 41.6 (p=0.003). For each subsequent stroke, the risk of seizure increases by 1.5 (p=0.032) and the risk of epilepsy by 1.6 (p= 0.077).
Conclusion: Seizures occur more commonly with recurrent strokes, SAH, and involvement of cortical and simultaneous cortical-subcortical areas. Cortical involvement and recurrent strokes have the greatest risk of epilepsy. This underscores the importance of secondary stroke prevention and the need for further studies on the impact of epilepsy on stroke outcome.

p128
Symptomatic seizures following acute Japanese and herpes simplex encephalitis: experience from a tertiary care centre in South India
SINHA S, RAO S, RAVI V, NAGARATHNA S
NIMHANS, Bangalore, India

Purpose: Acute viral encephalitis (V.E.) often manifest with seizure. Focused studies regarding symptomatic seizures in acute V.E. are less. We studied the profile, and risk factors of acute symptomatic seizures in acute V.E. and analyze the prevalence of seizure recurrence in those with acute symptomatic seizures.
Method: This study involved 75 patients (age: 27.44 ±18.47 years; M:F:: 38:37) of confirmed acute V.E. viz. Herpes simplex encephalitis (HSE: 48) and Japanese encephalitis (JE: 27). Diagnosis was based on supportive clinical, radiological, CSF serology profiles and EEG changes (HSE). Detailed analysis was performed in 55 patients with acute symptomatic seizures.
Results: Acute symptomatic seizures was noted in 55/75 patients (73.3%): HSE: 35/48 (72.9%) and JE: 20/27 (74.1%). The seizure types were: generalized (38.7%); focal (34.6%). Status epilepticus was noted in 10 (13.3%), while cluster seizures were observed in 9 (12%) patients. EEG (n=19) in JE revealed background slowing (n=15). EEG in HSE (n=35) showed background slowing - 32, lateralized abnormality -15 (PLEDs: 6) and bilateral fronto-temporal findings -19. Imaging abnormalities were: HSE: CT-10/35; MRI-31/33; JE: CT-12/20; MRI-5/10. All received parenteral phenytoin and 25.5% required a second AED. None of the parameters were significantly different between patients with and without acute symptomatic seizures. Among patients with acute symptomatic seizures, 21.8% developed recurrent seizures at follow up.

POSTER ABSTRACTS

Conclusion: Acute symptomatic seizure was noted in 3/4th of patients with acute V.E. and a fifth of them developed remote symptomatic epilepsy. However, no risk factor could be identified with acute symptomatic seizures in this cohort.

p129
Quality of life for Mongolian patients with epilepsy
BAYARMAA D[1], ARIUNAA J[2]
[1]Alfa-Dolgion Neurology Hospital, Shastin Central Hospital, Ulaanbaatar, Mongolia, [2]Shastin Central Hospital, Neurology Department, Ulaanbaatar, Mongolia

Purpose: To investigate the features and related factors of quality of life for people with epilepsy in Mongolia.
Method: A prospective study with clinical, demographic and psychosocial details was collected at Epilepsy Mini Center of Alfa ·Dolgion Neurology Hospital of Ulaanbaatar city. Data were collected from September 2010 · May 2010. QOLIE ·31 P was translated into Mongolian and completed by 184 ambulatory patients aged 18·35 years with localisation·related epilepsy. Were analyzed scores from each component of the QOLIE ·31 P.
Results: Was strong correlation between seizure frequency with seizure worry and psychological distress score. Significant high score dominated in seizure worry, medication effect and mental activity.
Conclusion: Mental activity and social functioning with seizure concerns mostly affects the quality of life Mongolian patients with epilepsy. Using this questionnaire in our daily practice we do touch and discuss social and mental problems, which our patients were facing. This study made one new step to show that Mongolian physicians need to know not only epilepsy as a disease but about the patients concerns of epilepsy, including awareness of seizures, seizure effects, emotions and anxiety, social activities, medication effects. Recognition of patient's all distress will be useful for total epilepsy care and improve the quality life of patients.

p130
Intracranial EEG ictal onset: which frequency, high or low?
CHUNG JM[1], TSUCHIYA N[2], TUDUSCIUC O[2], MAMALAK A[3], ELIASHIV D[1]
[1]Cedars-Sinai Medical Center Dept of Neurology, Los Angeles, USA, [2]California Institute of Technology, Pasadena, USA, [3]Cedars-Sinai Medical Center Dept of Neurosurgery, Los Angeles, USA

Purpose: To identify ictal onset frequencies with wide spectrum EEG frequency analysis.
Methods: Four patients with medically refractory partial epilepsy undergoing intracranial macroelectrode monitoring (2 depth electrodes, 2 subdural grids) were analyzed. Digital EEG data was sampled at 2 kHz at various intervals. Multiband frequency and power analysis were performed to characterize the predominating frequency during the interictal, pre·ictal, ictal, and postictal periods.
Results: Seven seizures—four seizures collected from subdural grid electrodes and three seizures from depth electrodes—were analyzed. The power spectrogram of frequency band between 0 · 100 Hz demonstrated suppression 2 seconds prior to ictal onset adjacent to the ictal onset electrode. In each case, ictal onset was localized to one contact and was characterized by a significant increase of 10 · 30 Hz frequencies preceding the increase of 30 · 100 Hz frequencies by 3 seconds before propagation. The synchronization of these alpha·beta frequencies was not seen during the interictal period. Focal surgical resections were performed in the areas correlated to the alpha·beta synchronization and HFO prior to and during the patients' clinical seizures. The patients have seizure·free outcomes confirming the localization.

POSTER ABSTRACTS

Conclusion: Previous studies of HFO from intracranial EEG recordings consistently show the frequencies at ictal onset above gamma range. However, most of these studies utilize microelectrodes and/or single neuron recording techniques. In our study, HFO were preceded by lower frequency activity. HFO may not be the first ictal manifestation in some cases and lower frequency ictal frequencies should not be overlooked. Larger studies are needed.

p131
Early ictal SPECT injection in the localisation of the epileptogenic zone
WONG TH, BIRKETT J, MCANALLY L, BARTLEY M, THEA KHUN T, LARCOS G, BLEASEL A
Westmead Hospital, Westmead, Australia

Purpose: To analyse the benefit of an early ictal SPECT injection in the localisation of the epileptogenic zone.
Method: A retrospective analysis of SPECT scans obtained with a nurse at the bedside was compared to 26 consecutive SPECT performed by nurses alerted by seizure alarm. The result was analysed with Mann-Whitney and Chi-Square Tests.
Results: A total of 31 bedside SPECT were performed between March 2003 and March 2010 in patients as part of the workup for epilepsy surgery. 15/31 (48.4 %) bedside SPECT were performed while patients were hyperventilating (HV). The time to SPECT ranged from 0-54 sec for the HV group (mean of 22.1 seconds) and 3-24 seconds for the non-HV bedside group (mean of 13.0 seconds). The 26 non-bedside SPECT injection times ranged from 26-136 seconds (mean 69.7 sec). Concordance between epileptogenic zone and subtraction interictal/ictal SPECT was; 62.5% for non-HV bedside SPECT, 60% for HV bedside SPECT and 46 % for non-bedside SPECT (no statistical significance). The mean duration of seizure was 54 seconds in bedside SPECT vs 116 seconds in non-bedside SPECT seizures. Logistic regression demonstrated statistical significance for localisation of seizure focus when SPECT injection was done within 60 seconds (61% vs 31%).
Conclusion: Non-bedside SPECT is useful in localisation of epileptogenic focus when it can be accomplished within 60 seconds of seizure onset. Bedside SPECT (HV or non-HV) has an important role in early injection time with short seizures.

p132
Presence of human herpes virus-6 DNA exclusively in temporal lobe epilepsy brain tissue of patients with history of encephalitis
NIEHUSMANN P[1], MITTELSTAEDT T[1], BIEN CG[2], DREXLER JF[3], GROTE A[4], SCHOCH S[1], BECKER AJ[1]
[1]Dept. of Neuropathology, University of Bonn Medical Center, Bonn, Germany, [2]Dept. of Epileptology, University of Bonn Medical Center, Bonn, Germany, [3]Dept. of Virology, University of Bonn Medical Center, Bonn, Germany, [4]Dept. of Neurosurgery, University of Bonn Medical Center, Bonn, Germany

Purpose: Temporal lobe epilepsy (TLE) is frequently associated with mesial temporal sclerosis (MTS). Many etiological aspects of TLE are still unresolved. Early age of initial precipitating injury is an important predictor of hippocampal pathology. Febrile seizures in children are frequently associated with human herpesvirus-6 (HHV-6) infections and more than 95% of children older than two years are HHV-6 seropositive. HHV-6 can establish lifelong latency in the CNS. A pathogenetic relationship of persistent HHV-6 infection and TLE has been suggested. Here, we analyzed HHV-6 presence in surgical tissue from well-defined subgroups of TLE-patients.
Method: Nested PCR, sequence analysis and immunohistochemistry were performed to identify HHV-6 DNA or antigen and to differentiate HHV-6 variants. The subgroups of pharmacoresistant TLE-patients were classified by clinical and neuropathological criteria as follows: Idiopathic MTS (n=10), lesion-associated (n=10), complex febrile seizures (CFS, n=6), history of encephalitis (n=9). Temporal lobe autopsy samples from patients without a history of neurological disorders served as controls (n=10).

POSTER ABSTRACTS

Results: HHV-6B DNA but no HHV-6 antigen was detected in 55.6% of specimens from patients with a history of encephalitis. In contrast, lesion-associated TLE and autopsy-control specimens were negative for HHV-6 DNA and antigen. Intriguingly, in non-lesional MTS (with or without history of febrile seizures) neither molecular nor immunohistochemical analyses revealed presence of HHV-6.
Conclusion: Our data argue against HHV-6 as major local pathogenetic factor in MTS hippocampi after CFS. The high detection rate of HHV-6B DNA suggests a potential pathogenetic role of HHV-6B in TLE patients with a history of encephalitis.

p133
Electrical status of slow wave sleep in adults
MCCONNELL H
Griffith University School of Medicine, Southport, Australia

Purpose: To review Electrical Status of Slow Wave Sleep in adults.
Method: Case study and literature review.
Results: Electrical Status of Slow Wave Sleep is a rare disorder that is usually diagnosed in children and frequently associated with a variety of neuropsychiatric sequalae. A 35 year old gentleman with a history of abuse and sever cognitive deficits is presented here. He presented with a history of infrequent daytime complex partial seizures and episodes of severe agitation on the background of severe language and intellectual impairment since childhood. His seizures occurred with and without secondary generalization. His EEG showed frequent seizure activity throughout slow wave sleep and his MRI was normal. Trials with various antiepileptic drugs have not been successful in affecting either his daytime neuropsychiatric manifestations or his nocturnal EEG abnormalities. A second adult patient with frequent epileptiform discharges during slow wave sleep but not fully meeting criteria for electrical status of slow wave sleep is also discussed. A review of electrical status of slow wave sleep in adults is presented with discussion of neuropsychiatric manifestations of this disorder.
Conclusion: Electrical Status of Slow Wave Sleep may occur in adults with Neuropsychiatric presentations.

p134
Cerebral venous thrombosis as a cause of new-onset seizures in young adults
KIM J
Hallym University Sacred Heart Hospital, Anyang, Korea, Republic of

Purpose: To report cases of new-onset seizures in young adults induced by venous thrombosis.
Method: A 23-year-old man and a 36-year-old man visited hospital, complaining of loss of conciousness and headaches.
Results: Their MRI showed acute infarction in high frontal areas and MR venograms demonstrated thrombosis of superior sagittal sinus. Risk factors of cerebral venous thrombosis were not found in both cases. They were treated with anticoagulation and follow-up MR venogram after three months revealed recanalization of superior sagittal sinus.
Conclusion: Cerebral venous thrombosis could be a rare cause of new-onset seizures in young adults.

p135
Surveillance of epilepsy in catchment area of Dhulikhel Hospital
PANT P, GAUTAM N, UPRETY S, KOJU R, GURUNG R, POKHAREL B, BEDI TRS
Kathmandu University School of Medical Sciences, Department of Internal Medicine, Dhulikhel, Nepal

Purpose: The objective of the present study was to carry out the surveillance to identify and diagnose the epilepsy cases in catchment of the Dhulikhel Hospital.

POSTER ABSTRACTS

Method: Surveillance of epilepsy was done through the five different outreach centers under Dhulikhel hospital .The health worker working in the center carried out a primary survey at household level to detect the epilepsy cases in the community.
Instead of using the usual open method of history-taking we devised a set of questions which would give us an idea whether a person had epilepsy or some other cause of blackouts and if it was epilepsy whether it was primary or secondary. We put these questions on an Excel spreadsheet so that the individual record could be easily turned into a database.
Results: Out of 41 patient seen 30(73.2%) were diagnosed as epilepsy, 8 (19.5%) were diagnosed as non epilepsy and 1 (2.4%) had single seizure. Among 30 cases only 1(3.3%) were diagnosed as primary and 27(90.0%) were diagnose as secondary epilepsy. However 2(6.7%) were diagnosis was uncertain. Newly diagnosed cases were 13(43.3%) out of 30. 12(40.0%) people with first attendance of probable epilepsy were prescribed drugs as follows, 6(50.0%) were given carbamazepine, 5(41.7%) were given phenytoin and 1(8.3%) was given sodium valproate.
Conclusion: Epilepsy is a common neurological problem, most of the time the diagnosis of epilepsy is uncertain, it has to differentiate from others causes of blackouts.
Simple questionnaire tools can be used to identity, diagnose, and treat epilepsy in the developing world.

Epidemiology

p136
Seizure-related injuries in newly diagnosed childhood epilepsy
TING D, KWONG LK
Tuen Mun Hospital, Hong Kong, Hong Kong

Purpose: To evaluate seizure-related injuries in children with newly diagnosed epilepsy
Method: Children aged less than 18 years with newly diagnosed epilepsy presenting to the Pediatric department in Tuen Mun hospital between 2002 and 2003 were recruited. All children were interviewed before January 2005. Children with seizure-related injury (cases) were compared to those without injury (controls) for identification of risk factors.
Results: One hundred twenty-two children were surveyed. No patient died during seizure. Eleven (9%) children suffered seizure-related injuries. Injury occurred at a mean age of 11.6 years and epilepsy was diagnosed at a mean age of 13.8 years in those with seizure-induced injury. Injury occurred at first seizure presentation or upon diagnosis of epilepsy in 72.7% patients. Ten (90.9%) children were not on anti-epileptic drugs at the time of injury. The mean age of seizure onset was 10.7 years in cases and 6.7 years in control (P=0.007). Seizures resulting in injuries were generalized tonic-clonic in 72.7% (P=0.045, OR=3.77, 95% CI=0.95, 14.98). Idiopathic etiology was observed in 54.5% and normal neurodevelopmental status in 72.7% of patients with injury. Age of seizure onset was the only independent variable retained on multivariate analysis. Soft tissue and dental injuries comprised 91.7% and 75% of injury occurred at home.
Conclusion: Risk of seizure-related injuries was substantial especially before epilepsy was diagnosed, but most of these injuries were minor. These unique data could help in parental counseling.

p137
Epidemiology of epilepsy in children less than five years of age in a developing nation
SUJATHA SG, THORA S, DUBEY R
Department of Pediatrics, MGM Medical College and my & CNBC Hospitals, Indore, India

POSTER ABSTRACTS

Purpose: Epidemiological studies are conducted in epilepsy in various centers of the world. Pattern differs in developing and developed countries. It is important to know about the pattern prevailing in our work area so as to focus our attention in that direction. This work is therefore designed to study the epidemiology in children of our area.
Method: This study was conducted between October 2008 to 2009 in children who were either visiting the outpatient Department or indoor patients, in our Department and were classified based on seizure types and *epileptic syndromes* as per the recent ILAE classification system.
Results: Total 54 patients were studied. 30(55.6%) patients had Generalized seizures and 23(42.6%) had partial seizures. One patient had multiple seizure types. Idiopathic epilepsy was found in 2(3.7%), Cryptogenic epilepsy in 8(14.8%), Symptomatic epilepsy in 42(77.7%) and 2(3.7%) were unclassified. In the syndromes, West Syndrome were 9(16.7%), Lennox-Gestaut syndrome was 1(1.8%), 1(1.8%) was Early infantile epileptic encephalopathy and 1(1.8%) was Dravet's syndrome.
Conclusion: *Defining the type of epilepsy and syndromic diagnosis should be considered mandatory* as they provide a firm foundation for short and long term therapeutic decisions and offer the best prognostic guidance.
Majority of our patients had symptomatic epilepsy with significant developmental delay, mostly due to preventable causes. The available public health measures should be carried out more effectively, giving more stress especially on perinatal care so as to decrease the proportion of these preventable epilepsies in the community and *help these children to live a normal life like others*.

p138
A study of newly diagnosed epileptic patients in Karachi
HEREKAR AD, NDC
Neuro Diagnostic Centre, Karachi, Pakistan

Purpose: To determine the characteristics of newly diagnosed epilepsy in Karachi population.
Method: This is a prospective study of 200 consecutively newly diagnosed epilepsy cases reporting to a private neurology centre in Karachi, Pakistan. The inclusion criteria were 2 or more siezures. All patients underwent EEG record.
Results: Two hundred patients were collected between 2008 to 2010. 125 patients were under 12 years of age Amongst the neurology patients reporting to a private neurology setup (Neuro Diagnostic Centre), patients suffering from epilepsy were studied for age of onset, gender and types of seizures and mental status. The commonest incidence of epilepsy was between 6 and 20 years. Children were found to be more affected as compared to adults. The mean age of onset of epilepsy was about 16 years. Localized epilepsy were found to be 58% while generalized epilepsy were 42%idiopathic generalized epilepsy was 28%, juvenile myoclonic epilepsy was 5%, childhood absence epilepsy 6%, West Syndrome 2%. Lennox Gastaut syndrome were 1%. West syndrome was 2%. Photosentive / photoconvulsive seizures were 4%. 28% of the cases were symptomatic and 72% were cryptogenic. 6% of close relatives (parents and siblings) had history of epilepsy.
Conclusion: The features of epilepsy in a Pakistani Population is largely similar to other under developed country. Genetic factors play an important role in causing epilepsy in the community.

p139
Prevalence of post-stroke seizures in Srinagarind hospital
TIAMKAO S, SRINAGARIND EPILEPSY RESEARCH GROUP
Faculty of Medicine, Khon Kaen University, Khon Kaen, Thailand

Purpose: Post-stroke seizures in Thailand have not been studied. Thus, the authors' main objective was to assess the prevalence of post-stroke seizures and secondary objective was to determine the factors associated with post-stroke seizures and mortality after stroke.

POSTER ABSTRACTS

Method: This was a retrospective, descriptive study. The population included stroke patients admitted to Srinagarind Hospital between 2000 and 2004. The patients were 15 years of age and older. The authors reviewed medical records, mailed out a questionnaire and conducted telephone interviews.
Results: The present study included 372 patients with stroke; of whom 15.6% had the seizures after the stroke. The length of follow-up was at least 5 years. Generalized tonic-clonic seizures were the most common type of post-stroke seizures. The time from the onset of stroke to the seizures was mostly (60.3%) less than 2 weeks (i.e., early post-stroke seizures). The associated factors of post-stroke seizures were: non-dyslipidemia (p=0.0007), intracerebral hemorrhage (p=0.015) and cortical area (p=0.05). The overall mortality rate at the time of the study was 39.5%, and 7.5% at 30 days and 22.8% at 1 year.
Conclusion: The prevalence of post-stroke seizures was higher than in previous studies but the associated factors of post-stroke seizures were similar.

p140
Sociodemographic and clinical characteristics of patients with epilepsy from Eastern Nepal
ROKA YB, BISTA BK, AHMAD SW, PARAJULI M
Neuro Hospital, Biratnagar, Nepal

Purpose: There are few studies on epilepsy from Nepal and the aim is to study the clinic-demographic profile of these cases presenting to this hospital with the aim to have a baseline data on seizure disorders in eastern Nepal which can be used for further studies or clinical trials and also for national data collection.
Method: 225 patients with seizure disorders were managed between December 2009 and June 2010 (new cases being recruited beyond today's date). The study is a prospective descriptive type based on a questionnaire that included sociodemographic and clinical variables like age, sex, type of epilepsy, duration and number of attacks, radiological and EEG findings, blood examination including serology for neurocysticercosis (NCC).
Results: The total number of cases is 225 (new cases being recruited beyond this date). The males were 59% and the rest females. Most of the cases were student with 50% followed by housewives (21%), office goers (20%) and farmers (10%). The most frequent clinical type was GTC in 68% followed by partial seizures in 20%. The most common symptom was headache in 57 % followed by learning disability in 35%. CT scan was done for all cases which showed calcification in 37% as the most common finding. EEG was abnormal in 65 % of cases. The CT and EEG correlated with in 22% of cases only.
Conclusion: The most common type of seizure was GTC. EEG was normal in 35% of cases and CT abnormal in70 % of cases.

p141
Traditional health practitioners in Jhenaidah district of Bangladesh: their knowledge on epilepsy, malaria, insomnia, and sexually transmitted diseases and possible role in primary health care
MOLLIK MAH[1,2]
[1]Peoples Integrated Alliance, Bogra, Not Applicable, Bangladesh, [2]Practical Academy on Wise Education & Research Foundation, Dhaka, Not Applicable, Bangladesh

POSTER ABSTRACTS

Purpose: Epilepsy, malaria, insomnia, and sexually transmitted diseases have been prevalent and even endemic in various parts of the world since ancient times. In recent years, attention has focused on these diseases because of the emergence of drug-resistant varieties of these diseases. As a result, it has become imperative to discover novel compounds to treat such diseases. Since medicinal plants form one of the best sources for obtaining pharmacologically active constituents, which can be used as remedy for diseases like epilepsy, malaria, insomnia, and sexually transmitted diseases. The objective of this present study was to conduct an ethnopharmacological survey amongst the traditional health practitioners of Jhenaidah district of Bangladesh; to obtain information on medicinal plants used by them as remedy for the above ailments. It is noteworthy in this regard that all the above-mentioned ailments are prevalent in Bangladesh, and the primarily rural population of the country relies on medicinal plants or plant parts prescribed by traditional health practitioners to treat the above ailments.
Method: Interviews were conducted of traditional health practitioners with the help of a semi-structured questionnaire and medicinal plant samples were photographed and identified at the Bangladesh National Herbarium.
Results: Information on the use of twenty-nine medicinal plants or plant parts was obtained.
Conclusion: Since the rural patients appeared to be generally satisfied with the treatment offered through these medicinal plants, it is important to conduct proper scientific studies towards discovery of compounds of interest in these plants, which can be used as safe and effective medicines.

p142
Incidence of epilepsy in us nursing homes by predisposing conditions
LEPPIK IE[1], EBERLY L[2], BIRNBAUM A[2], HARMS S[2], SVENDSEN K[2], GARRARD J[2], VIRNIG B[2]
[1]University of Minnesota, Golden Valley, USA, [2]University of Minnesota, Minneapolis, USA

Purpose: Although the point prevalence of epilepsy/seizure (epi/sz) in nursing homes (NHs) is approximately 10%, there is little information regarding the incidence of epilepsy after admission to NHs.
Methods: We examined the first (baseline) Minimum Data Set (MDS), a standard mandated assessment tool for residents in US NH performed at admission and quarterly during the stay, for all persons age 65+ years (N=4,588,324), of all Medicare/Medicaid certified US NHs during 2003-2005 for indication of epi/sz. Epi/sz was defined as ICD-9 code 345 or 780.39, or other notation in the MDS. Those with no epi/sz and ≥1 follow-up MDS (N=3,613,926), were followed through 2007 or NH end-of-stay to identify new-onset epi/sz.
Results: Of the 3,613,926 residents followed forward, there were 1,642 new-onset epi/sz cases/100,000 patient-years (100KPY). At baseline, 690,064 had a diagnosis of stroke, and these had incident epi/sz rates of 2,762/ 100K PY. Of 19,171 with head injury incident epi/sz was 4,566 /100K PY. Of 8,092 with brain tumor incidence epi/sz was 12,256/100K PY. Of 1,004,011 with diabetes incident epi/sz was 1,840/100KPY ; Of 205,320 with Parkinson´s Disease incident epi/sz was 1,766/100KPY ; of 2,349,465 with hypertension incident epi/sz was 1,667; of 1,410,514 with dementia (any type) incident epi/sz was 1,644/100KPY Among 407,801 residents with no “usual” predisposing diagnoses, epi/sz incidence was lower, 1,245/100KPY. Rates also differed significantly by US states.
Conclusion: Incidence of epi/sz during a NH stay is markedly higher than among community elderly, and varies widely by baseline conditions that are precursors to seizures.

p143
Prevalence of epilepsy in Korea: nationwide study from the national health insurance statistics
LEE S-Y[1], JUNG K-Y[2], KIM DW[3], EPIDEMIOLOGY COMMITTEE OF KOREAN EPILEPSY SOCIETY
[1]Kangwon National University, Chuncheon, Korea, Republic of, [2]Korea University, Seoul, Korea, Republic of, [3]Konkuk University Medical Center, Seoul, Korea, Republic of

POSTER ABSTRACTS

Purpose: There has been no population based epidemiologic data for Korean epilepsy. Our government-run medical security system that covers all the people and medical facilities enables nationwide epidemiologic study. We purposed to get the basic epidemiologic data of epilepsy for academic use and public health policies., based on the medical service utilization data from national health insurance database.
Methods: We used database of Korean Health Insurance Review and Assessment Service and Korean National Health Insurance Cooperation. We identified epilepsy patients by diagnostic codes used for medical insurance claim and anticonvulsant usage. We analyzed the one year prevalence of each age group, gender, economic status and region, the pattern of anticonvulsant medication, and the medical cost for 2007.
Results: Overall prevalence of the patients who took anticonvulsant under the diagnosis of epilepsy was 2.41/1000. The prevalence was higher in men than in women. Age specific prevalence was lowest in 30s and 40s. Epilepsy was 4 times more prevalent in low income persons who receive medical aid. The prevalence was highest in Jeju Island and lowest in Ulsan metropolitan city. In younger generation, new anticonvulsants were more frequently used. Total annual cost was 0.46% of total medical expenditure and 0.27% of total expenditure on health.
Conclusion: The overall prevalence of epilepsy in Korea is comparable to the results from contemporary studies performed in developed country. The prevalence is influenced by demographics, economic state and geography.

p144
The prevalence of seizure-related injury, near drowning and vehicular crashes in a community sample of patients with epilepsy
TAN M[1], FICKER D[2], O'BRIEN TJ[3], COOK MJ[1], PEARCE N[4], D'SOUZA WJ[1,4,5]
[1]Department of Medicine, St Vincent's Hospital, Melbourne, Australia, [2]University of Cincinnati Academic Health Center, Department of Neurology, Cincinnati, USA, [3]Department of Medicine, Royal Melbourne Hospital, University of Melbourne, Melbourne, Australia, [4]The Centre for Public Health Research, Massey University Wellington Campus, Wellington, New Zealand, [5]The Menzies Research Institute, University of Tasmania, Hobart, Australia

Background: The epidemiology of injury, near drowning and vehicular crashes in patients with epilepsy (PWE) has mostly been researched in hospital samples, which are prone to selection bias.
Objectives: To estimate the prevalence of seizure-related injury, near drowning and vehicular crashes in a community-treated sample of PWE.
Methods: The Australian national prescription database was used to recruit patients with epilepsy onto the Tasmanian Epilepsy Register (TER). Baseline assessment included assessment of lifetime and twelve month prevalence of injury, near drowning and vehicular crashes by validated interviewer-administered questionnaire.
Results: Of the 1180 initially enrolled on the TER, 1101 were subsequently available (43 withdrew, 36 died) with 997 (90.6%) completing interviewing: 581 Focal, 184 Generalised, 85 Non-epileptic seizures (NES), 46 Uncertain Epilepsy (US), 101 Uncertain partial or generalised epilepsy (UPG), 866 were included in the injury and near drowning analyses (excluding NES and US). In addition, 679 were only included in the driving analyses (age > 16 years, no driving disability). The estimated lifetime and 12 month prevalence was: any injury 213 (24.6%), 65 (7.5%); bathing or swimming 82(9.5%); 18 (2.1%), vehicular crash 82 (12.1%), 7 (1.0%).
Conclusion: Seizure-related co-morbidity with respect to injury, near drowning and vehicular crashes is a significant public health issue for PWE. Studying the risk factors surrounding these events could help improve the safety, occupational, recreational and mobility choices available to PWE.

POSTER ABSTRACTS

p145

Prescription patterns of antiepileptic drugs in Taiwan: a nationwide population-based study in the years 2003-2007

HSIEH L-P[1,2], HUANG C-Y[2]

[1]Department of Neurology, Cheng-Ching Hospital, Taichung, Taiwan, Republic of China, [2]Department of Industrial Engineering and Enterprise Information, Tunghai University, Taichung, Taiwan, Republic of China

Purpose: The aim of the study was to evaluate prevalence of use and prescribing of antiepileptic drugs (AEDs) in epilepsy and other indications in a nation-wide population using a prescription database.
Method: The patients who had at least two prescribed and dispensed AED were selected into the study from the random-sampling files that included about 600,000 populations. The diagnostic coding of NHIRD is according to the International Classification of Diseases, Ninth Clinical Modification (ICD-9-CM).
Results: Prevalence of AED use (for 1000 inhabitants) increased form 12.6 in 2003 to 13.8 in 2007. About the prevalence of new AEDs use increased progressively form 1.0 in 2003 to 3.8 in 2007, but the prevalence of old AEDs were decreased (11.6 to 10). Newer AEDs increased from 14.6% of total AED use in 2003 to 25.5% in 2007, with a corresponding decrease of older AEDs from 85.4% in 2003 to 74.5% in 2007 that still represented the majority of total AED volume dispensed. Carbamazepine and valproic acid were the most common AEDs, about the new generation AEDs, Gabapentin was the first. New AEDs were mostly used in patients aged 65 years and older. The more widespread use of newer AEDs was for mood disorders and pain. Older AED currently remain first line drug for epileptic disorders.
Conclusion: An increasing use of AEDs has been recently observed over a 5-year period in NHIRD, mostly explained by newer compound used for conditions other than epilepsy.

Prognosis

p146

Predictors of outcome in the resective surgical treatment of epilepsy

YANG X, LU Q

Shanghai Jiao Tong University, Shanghai, China

Purpose: In order to identify the prognostic factors for selection of ideal patients for epilepsy surgery and also to define features of different pathological types of focal cortical dysplasia.
Method: We restropectively reviewed clinical, EEG, MRI, histopathology, surgical variables and seizure outcome data in 99 patients from Shanghai Renji Hospital between 2003.1 and 2008.12 with at least 1 year of postoperative follow-up. Seizure outcome was categorized based on the modified ILAE classification.
Results: Of 99 operated patients, by univariate analysis, hippocampal sclerosis with medial temporal lobe epilepsy ($p< 0.01$), MRI with invisible focal lesion concordant with EEG ($p< 0.05$), ictal EEG regional patterns ($p< 0.05$) and EcoG regional patterns ($P< 0.05$) were associated with favorable surgery outcome.Recurred within 6 months postoperatively($P< 0.01$),incomplete resection ($p< 0.05$) and surgery complications ($P< 0.05$) were associated poor outcome. By multivariate analysis, hippocampal sclerosis with medial temporal lobe epilepsy ($P< 0.01$) and MRI with visible focal lesions ($P< 0.05$) were independent presurgical predictor of favorable outcome ($P< 0.01$) and recurred with 6 month was the only significant independent predictor associated with poor outcome ($P< 0.01$). Of 52 FCD patients, the prognosis was better in FCDII types compared with FCDI types ($P< 0.01$) and FCD Ib had the worst surgery outcome.

POSTER ABSTRACTS

Conclusion: Our study shows that patients with hippocampal sclerosis and abnormal MRI with epileptic lesions are strongly associated with favorable surgical outcome .Different histopathological types of FCD have distinct clinical and imaging characteristics and FCD type II has better surgical outcome.

p147
Does early electroencephalography (EEG) finding of clinical neonatal seizures have a predictive value to long-term outcome?
SANG T, JIANG Y, YANG Y, LIU X, QIN J
Department of Pediatrics, Peking University First Hospital, Beijing, China

Purpose: There are still debates on if aggressive treatment should be given when the neonate seizure first occurs. The aim of this study was to determine if electroencephalographic(EEG) characteristics at early stage of newborns with neonatal seizures indicated the long-term neurodevelopmental outcome, so as to give an evaluation of prognosis——normal or retardation, with or without post-neonatal seizures(PNS) ——at a certain extent.
Method: Of 247 infants with clinical neonatal seizures of diverse etiologies admitted to the neonatal ward from January 1997 to December 2009, 158 were followed up at the age of 4 months to 10-year-old. The study was based on the Video-EEG record combined with clinical information and nervous system physical examination when they were in hospital. The follow-up data included neurodevelopmental outcome (evaluated by Gesell Developmental Screening Test or Wechsler Preschool and Primary Scale of Intelligence) and PNS outcome.
Results: Developmental outcomes were significantly associated with background activity of EEG (*Sig*< 0.001) and cranial image (*Sig*=0.005). The presence of PNS only correlated to background activity of EEG significantly (*Sig*=0.001).
Conclusion: Abnormal neurodevelopmental outcome occur more likely in the patients with abnormal EEG background activity or with abnormal cranial image than that in patients with normally or mildly abnormal EEG background and without abnormal cranial image. Background activity of EEG is the best predictive factor for long-term prognosis of neonatal seizures (both neurodevelomental outcome and PNS outcome), whereas epileptic discharge is not an independent risk factor of unfavorable outcome in our cohort.The extended follow-up study is undergoing.

p148
A study of predictive factors for post stroke seizures
DHAMIJA RK, KAINTURA A, KUMAR M
Lady Hardinge Medical College, New Delhi, India

Purpose: The clinical profile and predictive factors of seizures associated with stroke is uncertain in developing countries, where stroke is common in young. Hence timely identification of patients at risk to develop seizures is crucial for management. The present study was therefore conducted to analyze the predictive factors of seizures associated with stroke.
Methods: Patients admitted with clinical diagnosis of stroke and developing seizures in hospital or during a follow up of 6 months were evaluated for type of seizures as well as for timing of seizures. Clinical Profile, Stroke mechanism, EEG, Echo, Metabolic profile and Cerebral imaging studies were analyzed to study predictive factors for occurrence of seizures.

POSTER ABSTRACTS

Results: The study included 127 patients of ischemic stroke and 61 patients of hemorrhagic stroke. Out of 19 patients who had post stroke seizures, 36.84% of patients had early onset and 63.16% had late onset seizures. 31.57% of patients who had seizures went on to develop recurrent seizures. 47.36% of these post stroke seizures were having ischaemic stroke while 52.64% had haemorrhagic stroke. 26.31% of post stroke seizure patients had generalized tonic clonic seizures whereas 42.10% had partial seizures and 31.57% had partial onset generalized seizures. Patients with recurrent stroke, hypertension with large infarct on CT were found to be at more risk of developing seizures. Patients who developed recurrent seizures had large cortical lesions with severe neurological deficit at presentation.
Conclusion: Patients with large cortical infarcts, previous stroke and hypertension are at higher risk of developing seizures after stroke.

Paediatric epileptology

p149
Adolescent methamphetamine administration impairs spatial memory in rats with neonatal status epilepticus
HUANG L-T[1], WANG J-Y[2]
[1]Department of Pediatrics, Chang Gung Memorial Hospital-Kaohsiung Medical Center, Chang Gung University College of Medicine, Kaohsiung, Taiwan, Republic of China, [2]Department of Physiology, Graduate Institutes of Medical Sciences, Taipei Medical University, Taipei, Taiwan, Republic of China

Purpose: Neonatal status epilepticus (NeoSE) may cause functional impairment in adulthood. The purpose of this study was to compare the effects of adolescent methamphetamine (MA) on spatial memory in rats with or without prior NeoSE.
Method: NeoSE was induced at postnatal day 10 (P10) by lithium-pilocarpine (LiPC). At adolescence (P43), rats received saline or MA for 5 days. After a 7-day withdrawal of MA, rats underwent daily (P55 ~ P60) Morris water maze testing for spatial memory and tissue for biochemical analyses.
Results: The escape latency in MWM was significantly longer in LiPC/MA group (NeoSE with adolescent MA administration) than in other groups, suggesting an impaired spatial memory. The LiPC/MA group showed an impaired spatial memory which paralleled lower mRNA levels of tissue plasminogen activator (tPA) and serum- and glucose-regulated kinase-1 (SGK1) in the hippocampus than other groups. Mossy fiber sprouting only in the CA3 region in LiPC/MA group but not in other groups, indicating synapse reorganization. Tissue content of malondialdehyde was also the highest in LiPC/MA group while gene expression of superoxide dismutase 2 significantly increased in Sal/MA group and further increased in LiPC/MA group.
Conclusion: These results indicate that adolescent MA administration impairs spatial memory and is accompanied by synaptic plasticity and increased oxidative stress in the hippocampus in rats suffered with prior insult of NeoSE.

p150
Mozart K.448 acts as an add-on therapy in children with electrophysiologically or clinically refractory epilepsy
LIN L-C, YANG R-C
Kaohsiung Medical University Hospital, Kaohsiung, Taiwan, Republic of China

Purpose: Our previous work revealed that epileptiform discharges in epileptic children decreased during and right after exposure to Mozart's Sonata for two pianos in D major, K.448 (Mozart K.448). In this study, we evaluated the long-term effect of Mozart K.448 on children with electrophysiologically or clinically refractory epilepsy.

POSTER ABSTRACTS

Method: Twenty-three children with epilepsy who were (I) clinically well-controlled by antiepileptic drug (AED) but electrophysiologically refractory for at least 6 months (N=12), or (II) patients with clinically refractory seizures (N=11) were included in this study. Epileptiform discharges in the electrophysiologically refractory group were recorded before and at 1, 2, and 6 months. Seizure frequencies were recorded before and monthly during the study period in the clinically refractory seizure group. During the study period, both groups received Mozart K.448 exposure once a day before bedtime for 6 months, and all of the patients remained on the same AED.
Results: The epileptiform discharges in the electrophysiologically refractory group of patients were significantly decreased when compared with the electroencephalogram (EEG) of each patient before music exposure. The effect appeared as early as one month and the average decrease in epileptiform discharges at 1, 2, and 6 months was 67.70±30.91%, 79.54± 29.88%, and 85.15±28.13%, respectively. Eight of eleven patients with clinically refractory epilepsy showed excellent responses, two of them were seizure free and six of them had very good responses.
Conclusion: We conclude that Mozart K.448 could be used as an effective add-on therapy in the treatment of patients with electrophysiologically or clinically refractory epilepsy.

p151
ACTH therapy for two cases of epileptic spasms without hypsarrhythmia
SHIMONO M[1], ISHII M[1], SENJU A[1], SHIOTA N[2], KUSUHARA K[1]
[1]University of Occupational and Environmental Health, Japan, Kitakyushu, Japan, [2]Department of Health Care & Support Center, Environment & Safety, UBE Industries, Ube, Japan

Purpose: Epileptic Spasms without Hypsarrythmia (ESWH) is an important and newly identified epileptic syndrome in childhood. This syndrome, however, was not included in the 2009 ILAE epileptic syndromes list.
Method: We present two male cases of ESWH: case 1 was a 1-year-old, and case 2 was a 6-year-old. Both were treated successfully with adrenocorticotropic hormone (ACTH) therapy in 2009.
Results: Both had sudden flexion of the axial muscles that resulted in head nodding, which started at 17 months (case 1) and 14 months (case 2) of age. Their development was almost normal at the onset of the seizures, but gradually grew delayed thereafter. Their inter-ictal electroencephalography (EEG) showed generalized polyspikes. Single or combined administration of anti-epileptic drugs including VPA, ZNS, CZP, CLB, TPM, and LTG was ineffective. Thus, we provided ACTH therapy combined with a few anti-epileptic drugs, which resulted in successful seizure control.
Conclusion: While ESWH is a distinct type of epilepsy from either West syndrome or Lennox-Gastaut syndrome, its prognosis and treatments are similar to those of the idiopathic West syndrome. Early treatment with ACTH might be an essential factor for a favorable outcome.

p152
A clinical case study of hypomelanosis of Ito and epilepsy
ZHANG J
Hunan Children's Hospital, Changsha, Hunan, China

Hypomelanosis of Ito, also known as the lack of incontinentia pigment achromians, is mainly with skin and nervous system symptoms and may be associated with many side symptoms such as head and facial dysplasia, congenital malformation of heart and reproductive systems, etc. Because it is related to multiple systems and organs it is now considered a multi-system involved skin disease. Ito (Ito´s) first reported this disease in 1951 and he considered it might be due to autosomal dominant inheritance. Now people are inclined to think that the possible pathogenesis of the disease is mutations caused by the X chromosome inactivation or activation that caused abnormal transition of mesoderm and ectoderm precursors during embryonic development, and as a result, leading to skin section line development disorder, neuronal migration disorder, neurodysplasia, and forming multi-system abnormalities.

POSTER ABSTRACTS

At present the available epidemiological data for such kind of neurocutaneous syndrome disease is very limited. One case was identified in Changsha, China and is reported here. At first the whole process of diagnosis and treatments for the patient will be described in detail. Then, discussions are presented on how to identify Hypomelanosis of Ito from another two similar neurocutaneous syndromes: incontinentia pigmenti and tuberous sclerosis. The last part of this paper is recommendations on general procedures of diagnosis and treatments for this disease.

p153
Utility of prolonged video EEG recording in assessment of refractory epilepsies
FUNG L-WE, CHONG J, TSUNG L-YL
Department of Paediatrics, The Chinese University of Hong Kong, Shatin, Hong Kong

Purpose: The assess the utility and results of patients with refractory epilepsies who undergone prolonged EEG monitoring as part of the workup for possible epilepsy surgery
Method: Medical records of patients with refractory epilepsies who underwent prolonged (> 24hours) video EEG monitoring in 2007-2009 were reviewed. Their demographic data, primary diagnosis, duration of epilepsy, recording results, subsequent management plan and outcome were retrieved.
Results: Thirty patients underwent video EEG recording during the study period. Three patients had two recordings during this period. (17 male and 10 female patients) Age ranged from 8 month to 18.1 years old (Median = 12 year old). Duration of epilepsy before this recording ranged from 4 months to 14.5 years (Median =3.9 years) The duration of recording ranged from one day to eight days. (Median = 2days) In 24 of the recordings, habitual seizures were captured. The results of the recordings facilitate further investigations for surgical workup (including ictal SPECT/ inter-ictal PET) for four patients, surgical planning made for four patients and three patients had undergone surgery. Two undergone resective surgery and the other had corpus callosotomy. The former are now seizure free.
Conclusion: Video EEG is a useful tool for clarifying the exact seizure type in pediatric patients, as seizures may be subtle and accurate description from care-takers could be difficult. This may in turn facilitate further management options

p154
Oxcarbazepine oral suspension in epilepsy: our experience in Taiwanese infants and children, and a review of the literature
WEI S-H[1], FAN P-C[2]
[1]E-Da Hospital, Kaohsiung County, Taiwan, Republic of China, [2]National Taiwan University Chidren Hospital, Tapei, Taiwan, Republic of China

Purpose: There are rare studies regarding the use of Oxcarbazepine(OXC) in oral suspension. Moreover, most clinical studies have assessed the efficacy and safety of OXC in Western pediatric patients rather than Asian ones. We, thus, investigated the efficacy, tolerability, and side effects of OXC oral suspension in infants and children with various types of epilepsy in Taiwan.
Method: A retrospective review of the efficacy, tolerability, and side effects of OXC oral suspension in a tertiary medical center in Taiwan was conducted in the pediatric patients younger than 9 years old, diagnosed with partial epilepsy, which was divided into idiopathic partial, symptomatic partial or multifocal subtypes.

POSTER ABSTRACTS

Results: A total of 36 patients (mean age 29.5±4.7 months, 19 males and 17 females) were enrolled. Of them, 24 (66%) were seizure free, 6 (17%) had seizure reductions of more than 50%, 5 (14%) seizure reductions of less than 50% and 1 (3%) no effect after OXC treatment. The effectiveness was significantly related to the epilepsy subtype ($p < 0.01$) and the number of combined antiepileptic drugs before OXC treatment ($p < 0.01$). The patients with idiopathic and symptomatic partial epilepsy responded better to OXC oral suspension than those with multifocal epilepsy. Nine patients (25%) had minor or moderate side effects.
Conclusion: OXC oral suspension is effective and well tolerated both as monotherapy or adjunctive therapy in the Asian infants and children with partial epilepsy. We also demonstrate the published articles about pediatric patients from different populations or countries treated with OXC tablets or oral suspension.

p155
Study about prevalence of idiopathic generalized epilepsies in children
KUMAR A, PRAKASH S, KUMAR A, KUMAR R
Indira Gandhi Institute of Medical Sciences, Patna, Bihar, India

Purpose: To know the prevalence of idiopathic generalized epilepsies in children and to study about its characteristics and to compare with other studies.
Methods: and Observations·Inclusion criteria·
(1)Age between 1·14 years of age.
(2) Clinical semiology suggestive of generalized seizure.
(3) EEG · normal/generalized epileptiform activity.
(4) CT/MRI ·Normal or abnormality unrelated to epileptiform activity/syndrome.
(5) Seizure in sleep with criteria 2, 3 & 4.
(6)EEG shows predominantly generalized epileptiform activity with occasional focal activity but satisfying criteria 2 & 4.
Exclusion criteria·
(1) Age < 1 yr and >14 years.
(2) Non progressive encephalopathy cases with epilepsy.
(3) Clinical semiology not clear/ favors focal seizure like history of aura. 4 Symptomatic cases.
5 EEG· Clear focal activities.
Results: Over a period of 3 years 5000 epileptic patients seen. Between 1·14 years of age group were 900 (18%). 180 cases (20%) included in the study. Boys were affected more than girls, particularly in JME. The prevalence of IGE was similar to that found by Eriksson, Shah and Genton. Waaler and Murthy had a lower prevalence of IGE.The prevalence of juvenile absence epilepsy in our study was consistent with results from most studies.
Conclusion: JME is most commonest generalized childhood epilepsy, followed by childhood absence epilepsy and juvenile absence epilepsy. These syndromes has excellent prognosis. EGMA is uncommon in the childhood population. The problems with classification as pure generalized tonic clonic seizures often are that minor absence or myoclonic seizures may precede GTC's by several years and may also go unnoticed causing misclassification as other idiopathic generalized epilepsy.

p156
Neuropsychological change over time in children with benign rolandic epilepsy
KWON S, SEO HE, BYUN JC
Kyungpook National University Hospital, Daegu, Korea, Republic of

Purpose: Children with benign rolandic epilepsy (BRE) may display different degrees of neuropsychological deficits such as cognitive, ADHD, and other behavioral deficits. The aim of this study was to assess the change of neuropsychological function in children with BRE who was not medicated.

POSTER ABSTRACTS

Method: A total of 7 children with BRE, not on the antiepileptic drugs were involved in the study from 2008 to 2009. All children underwent a comprehensive neuropsychological battery including Korean versions of Wechsler Intelligence Scale for Children III (K-WISC-III), Frontal Executive Neuropsychological Test (K-FENT), Rey Complex Figure Test (RCFT), Wisconsin Card Sorting Test (WCST), Attention Deficit Scale (K-ADS), Child Behavior Checklist (K-CBCL) at the first and 6 month visits.
Results: The subjects exhibited no significant change in cognitive function, but surprisingly a significant improvement in scores of Executive IQ (+17.1) and Memory IQ (+12.7) as well as subtests of K-FENT (color reading interference of stroop test, learning/immediate recall of AVLT), delayed recall of Rey CFT over time ($p < 0.05$). This may result from learning effect, even though the follow-up evaluation was done 6 months later. However, they seemed to have attentional difficulties, though statistically not significant, social and emotional difficulties on ADS and CBCL.
Conclusion: The data has relatively limited predictive value due to small sample size, but at least show the evidence that contrary to the presumed benign nature of BRE, the condition may cause attentional, social and emotional problems. Despite the findings, further studies are needed to elucidate the core nature of BRE.

p157
Malignant epilepsies starting in the neonatal period and suppression-burst EEG pattern
YAMAMOTO H[1], OKUMURA A[2]
[1]St. Marianna University School of Medicine, Kawasaki, Japan, [2]Juntendo University School of Medicine, Tokyo, Japan

Purpose: As seizures in the neonatal period have generally been identified only by direct clinical observation, there is usually a lack of objectivity whether seizures are categorized as epilepsies or non-epilepsies. A major characteristic of neonatal seizures is electro-clinical dissociation and some electro-graphic seizures do not produce clinical symptoms. It is difficult to correctly identify real epilepsies or epileptic syndromes in the neonatal period without ictal electroencephalogram (EEG). Some epileptic syndromes starting in the neonatal period, such as early myoclonic encephalopathy, Ohtahara syndrome, or migrating partial seizures in infancy are categorized as malignant epilepsies. A suppression-burst EEG pattern (SBP) is usually seen in neonates with serious brain damage or neonatal epileptic syndromes.
Method: Our report is on malignant epilepsies in Japan starting in the neonatal period and we will propose a precise definition for SBP which has not correctly been identified in the literatures.
Results: Epileptic encephalopathies with SBP in neonatal period are known to evolve into relatively few types of epileptic syndromes.
Conclusion: We will emphasize the importance of ictal EEGs for diagnosis and treatment of malignant epilepsies and epileptic syndromes in the neonatal period.

p158
Quest for a pragmatic, patient oriented approach: modified ILAE classification in a pediatric epilepsy clinic
SRIVASTAVA K, RAJADHYAKSHA S
Bharati Vidyapeeth University Medical College, Pune, Maharashtra, India

Purpose: The previous attempts of classifying children with epilepsy using 1981/1989/2001 and 2009 proposals had many shortcomings. In clinical practice, a flexible module is recommended, so we combined the approach suggested by 2009 classification (syndrome à etiology à seizure type) while retaining the broad categories of axis 3, 2001 scheme.
Method: 1530 patients below 18 years of age were referred between 2006-2010.
Unequivocal epilepsy was diagnosed in 777 patients (50.78%) and classified as per the above mentioned approach.

POSTER ABSTRACTS

Results: Electro-clinical syndromes were identified in 163 (21%), distinctive constellations in 7 cases. In the remaining 607 patients, non-distinctive constellations recognized were GEFS+ (33), undetermined IGE (18) and BNFNS (5). Out of the remaining 551 cases, 380 cases could be classified on basis of underlying etiology (presumed genetic- 91, metabolic- 27, structural-20, perinatal insult-115, underlying mental retardation of un-identified cause -37, stroke-24, CNS infection-31 etc.) Undetermined epileptic encephalopathy was identified in 36 patients. In 171 cases, no etiology was found and the predominant mode of seizure onset was determined- focal in 141 cases, generalized in 25, while in 6 cases, the seizure type remained unclassified.
Conclusion: When the syndrome was not known, non-distinctive constellations and etiology helped and similarly, when etiology could not be determined, seizure type helped in management and prognostication to a large extent. Improvisation with assimilation of the advantages and inputs of the previous classification will obviate the limitations inherent in previous attempts. We found the above-mentioned approach to be pragmatic and patient-oriented.

p159
The expression of TRPV1 in cortical lesions of tuberous sclerosis complex and focal cortical dysplasias type IIb
SHU H-F[1], ZHANG C-Q[1], YU S-X[1], SONG Y-C[1], LIU S-Y[1], YANG H[1], YIN Q[2]
[1]Department of Neurosurgery, Xinqiao Hospital, Third Military Medical University, Chongqing, China, [2]Department of Rehabilitation, Southwest Hospital, Third Military Medical University, Chongqing, China

Purpose: Tuberous sclerosis complex (TSC) and focal cortical dysplasia type IIb (FCD_{IIb}) are malformations of cortical development (MCDs) that are recognized causes of intractable epilepsy. TSC and FCD_{IIb} are characterized by extensive laminar disruption, dysmorphic neurons (DNs), and the presence of balloon cells (BCs) or giant cells (GCs), which are closely related with the severity of seizure. However, the cellular mechanism(s) underlying the epileptogenicity remain(s) largely unknown. Transient receptor potential vanilloid receptor 1(TRPV1), a member of the transient receptor potential family, is the capsaicin receptor and is known to be invovled in the peripheral nociception. Recently, accumulating evidence indicates that TRPV1 may also play a significant role in the epileptogenicity and maybe a potential antiepileptogenic target. In the attempt to understand the mechanism(s) of epileptic activity in MCDs, we examined the expression and cellular distribution of TRPV1 in the cortical lesions of FCD_{IIb} and TSC.
Method: TRPV1 was studied in epilepsy surgery cases with FCD_{IIb} (n = 10) and TSC (cortical tubers; n = 10) using immunocytochemistry, confocal analysis, and western blotting.
Results: We demonstrated TRPV1 receptors were predominatly expressed in the misshapen cells, including DNs and BCs/GCs. The increased TRPV1 expression within the dysplastic cortex, compared to normal control cortex, was confirmed by western blot analysis.
Conclusion: These findings suggest that the TRPV1 may be invovled in the intrinsic and increased epileptogenicity of MCDs.

p160
Clinical characteristics of patients with West syndrome associated with occipital focal cortical dysplasia
AKASAKA N[1], TOHYAMA J[1], OHASHI T[1], KOBAYASHI Y[1], SASAGAWA M[2], MURAKAMI H[3], MASUDA H[3], KAMEYAMA S[3]
[1]Department of Pediatrics, Nishi-Niigata Chuo National Hospital, Niigata City, Japan, [2]Department of Psychiatry, Nishi-Niigata Chuo National Hospital, Niigata City, Japan, [3]Departmen of Neurosurgery, Nishi-Niigata Chuo National Hospital, Niigata City, Japan

POSTER ABSTRACTS

Purpose: Focal cortical dysplasia (FCD) in occipital lobe often causes West syndrome in addition to partial seizure. We aimed to delineate the clinical picture of patients who have cortical dysplasia in occipital lobe.
Methods: We examined three patients with FCD in occipital lobe, and West syndrome. We evaluated their clinical, neuroradiological findings, result of treatment and their prognosis.
Results: Patient 1 and 2 could not be detected FCD on initial MRI or CT, and follow up MRI at 7 years and 10 years of age could demonstrate FCD, respectively. Patient 1 and 2 showed infantile spasms as their initial ictal events. In early stage of disease, all three patients presented partial seizures, which accompanied with abnormal eye movements. Infantile spasms were well controlled with antiepileptic drugs or adrenocorticotropic therapy, but their partial seizures were intractable. In patient 1, surgical resection for focal cortical dysplasia was performed at 12 years of age, and the partial seizures disappeared, however, the improvement of intelligence quotient was not obtained. Other two patients did not hope surgical intervention, and their developments had gradually been delayed.
Conclusions: In the case of West syndrome with intractable partial seizures, it is likely to exist of pathological lesions such as FCD in occipital lobe even if initial MRI appears normal, and a careful observation is necessary to detect these lesions as early as possible. Early surgical intervention for children with intractable epilepsy associated with FCD could result in cessation of seizures and improvement of developmental problem.

p161
Electroclinical features of late-onset spasms: a report of 9 cases
HWANG YS, HWANG H, LIM BC, KIM KJ, CHAE JH
Seoul National University Children's Hospital, Seoul, Korea, Republic of

Purpose: The aim of this study was to assess the electroclinical features of patients with late-onset (12 months or older) spasms.
Method: We retrospectively reviewed the medical records and video-EEG recordings of the 9 cases (5 males, 4 females) diagnosed as having late-onset spasms at Seoul National University Hospital from 2003 to 2009.
Results: The median onset of spasm was 3.0 years (range, 13mo—8yr). The symptomatic etiologies were identified in 5 cases. In 4 cases, predominant axial tonic spasms caused head nodding or body fall instead of flexion or extension of extremities. Hypomotor or atypical absence seizures were accompanied in 4 cases either separately or following spasms. The interictal EEG revealed hypsarrhythmic pattern in only 1 case. While diffuse or bilateral frontotemporal slowing with some interhemispheric synchrony were the most frequent interical background abnormalities, focal or asymmetric slowing was observed in 2 cases. The ictal EEG revealed diffuse high voltage delta waves or sharp wave bursts followed by generalized voltage attenuation. Spasms were controlled in only 3 cases (2 with vigabatrin, 1 with topiramate). All cases including spasm free cases showed moderate to severe developmental delay or cognitive impairment.
Conclusion: Late-onset spasms was characterized by atypical ictal semiology, frequent association of hypomotor or atypical absence seizures, absence of typical hypsarrhythmic pattern, and poor treatment outcome. Focal EEG abnormalities in a subgroup of cases suggest focal pathology may contribute to late-onset spasms.

p162
Clinical experience with levetiracetam monotherapy in pediatric epilepsy in Chungbuk, Korea
KIM WS[1], SIM JY[1], PARK HJ[2]
[1]Chungbuk National University, Cheongju, Korea, Republic of, [2]Eul Ji University, Daejeon, Korea, Republic of

Purpose: This study was designed to evaluate the safety and the reduction in seizure frequency of Levetiracetam monotherapy for pediatric epilepsy in Chungbuk in Korea.

POSTER ABSTRACTS

Method: We prospectively reviewed the medical records of 49 pediatric epilepsy patients (male 33 and female 16) taken only Levetiracetam. They were followed up for over at least 4 months after prescribed Levetiracetam from June 2007 to October 2008 at Chungbuk national university hospital in Chungbuk in Korea.
Results: 44 patients (89.8%) out of 49 showed reduction in seizure frequency of more than 50%, and 43 patients (87.8%) more than 75%.Of 8 patients who had generalized seizures including 6 patients with juvenile myoclonic epilepsy, 2 patient with absence seizures, 8 patients showed seizure reduction of more than 75% (100.0%).Of 41 patients who had partial seizures, 36 patients showed seizure reduction of more than 50% (87.8%) and 35 patients (85.4%) of more than75 %.There were differences between generalized seizure and partial seizure in those with more than 75% seizure reduction rate.The mean maintenance dosage of drug was 27.1 ±8.0 mg/kg/day. 7 (14.3%) patients showed adverse reactions : increased seizure frequency in 3 (42.9%) patients who discontinued levetiracetam, memory impairment in 1 patients and fatigue, irritability and inattention in 1 patient.
Conclusion: Levetiracetam is believed to be effective, and safe when used a monotherapy for various childhood epilepsies.

p163
Rapid onset obesity, hypothalamic dysfunction, hypoventilation, and autonomic dysregulation - a clinical syndrome with autonomic crisis masquerading as epilepsy and encephalopathy: a report of two cases
TAJUL ARIFIN T, KHOO TB, KHOO PC, HUSSAIN IHMI, DAYANG SR, ASIAH K, NORZILA MZ
Institute of Paediatrics, Hospital Kuala Lumpur, Kuala Lumpur, Malaysia

Purpose: . ROHHAD (**R**apid-onset **O**besity, **H**ypothalamic dysfunction, **H**ypoventilation, and **A**utonomic **D**ysregulation) is a rare clinical syndrome in children with less than 50 cases reported. Children with ROHHAD typically present with rapid-onset obesity, followed by signs of hypothalamic dysfunction and finally the autonomic dysregulation and hypoventilation. The cause is still unknown.
Methods: We report two cases of ROHHAD that presented with recurrent episodes of cardiorespiratory arrest, encephalopathy and seizures.
Results: Two unrelated patients, a 6 year old girl and 11 year old boy, with ROHHAD and negative PHOX2B gene mutation were reviewed. They presented with rapid onset obesity, recurrent episodes of apnea, cyanosis, seizures and encephalopathy from the age of 2 and 1.4 years respectively.
For the first patient, her typical episode consisted of profuse sweating, hypothermia, tachycardia followed by bradycardia, eye twitching, gaze deviation, tonic seizures followed by cardiorespiratory collapse. Her bradycardia was refractory to adrenaline but responded to buccal Midazolam. For the second patient, his typical episode consisted of sudden behavioral change, confusion with thrashing, hypersalivation and encephalopathy. These were initially treated as frontal lobe epilepsy. Their interictal EEGs were normal and postictal EEGs only showed diffuse slow waves. Their initial brain MRI scans were normal. They were treated with various AEDs without any benefit.Their symptoms stabilized following the addition of Clonidine.
Conclusion: The above cases illustrated that autonomic crisis can masquerade as epileptic seizures and are refractory to antiepileptic drugs. Early recognition of this clinical syndrome and commencement of appropriate treatment is important to prevent catastrophic outcome.

p164
Impact of pediatric epilepsy surgery on quality of life: a case controlled study from Northern India
SHUKLA G, MOHAMMED A, GOYAL V, SRIVASTAVA A, GUPTA A, SURI A, CHANDRA PS, GARG A, GAIKWAD S, BAL CS, BEHARI M
All India Institute of Medical Sciences, New Delhi, India

POSTER ABSTRACTS

Purpose: To evaluate the impact of epilepsy surgery on the quality of life (QOL) of children and their parents amongst patients consecutively selected for surgical treatment of refractory epilepsy over a 4 year period.
Method: A prospective study using 2 scales, the Impact of Childhood Illness Scale (ICIS) and the Impact of Pediatric Epilepsy scale (IPES) was carried out to assess the impact after a minimum period of 6 months post-surgery. The two scales were administered to two groups; parents of children undergoing surgery for refractory epilepsy (Group 1), and from parents of age and gender matched controls, awaiting epilepsy surgery (Group 2). The results were compared using the Mann Whitney test.
Results: Twenty-one patients were selected in each group (Group 1 -15M; Group 2-13M), with a mean age (12 + 5 years) and duration of epilepsy (8 + 5.5 years). A favorable seizure outcome (Engel's class 1&2) was observed in 18 (85.7%) patients in Group 1. Statistically significant differences (Group 1 vs. Group 2) were found in the parental scores for "severity of impact" cumulative scores ($p < 0.05$) and the "impact on child development" sub-scores($p < 0.05$) on the ICIS scale. Parental responses were however similar for both groups when the IPES and a visual analog scale was administered.
Conclusion: Pediatric epilepsy surgery carries a significant positive cumulative impact on QOL, especially in the domain of child development and a favorable seizure outcome. The reasons for different parental responses to the two QOL scales utilized will be discussed.

p165
Remission of symptomatic epilepsy in post-liver transplant children in Singapore
LAINO AGG, ONG HT, AW M, QUAK SH
National University Hospital, Singapore, Singapore

Purpose: To describe the features of epilepsy in post-liver transplant recipients and determine its rate of remission after treatment.
Method: A retrospective review of liver transplants at the National University Hospital between 1991-2009 identified children who developed epilepsy post-operatively. The demographics, seizure etiology and characteristics, electroencephalogram (EEG) findings and treatment provided were analyzed. The mean follow-up after transplant was 10 years (range: 7-13 years).
Results: Seventy-three transplants were performed with biliary atresia as the most common indication in 67% of recipients. Five children developed epilepsy post-operatively. All had late seizure onset from 5 months to 8 years post-transplant. Hypoxic ischemic encephalopathy secondary to sepsis and posterior reversible encephalopathy syndrome were the causes of seizures in 2 patients on cyclosporine. The 3 recipients on tacrolimus had no secondary cause for seizures. All initial EEGs revealed focal epileptiform discharges. Two recipients were successfully weaned off treatment despite persistent EEG epileptiform discharges. Monotherapy using Gabapentin or Valproic acid for 3 years resulted in complete remission in 2 patients. Myoclonic jerks occurred twice weekly in one patient. The other 2 patients with breakthrough seizures were poorly compliant to treatment.
Conclusion: Seizures in this study occurred significantly later than recipients worldwide where seizures occurred within one month post-transplant. EEG discharges did not predict failure to attain seizure-free state. Late-onset epilepsy in post-transplant children required the use of anti-epileptic drugs with only 40% ultimately achieving seizure remission.

p166
Infantile spasm control and developmental progression following short course therapy with steroids/vigabatrin
WANIGASINGHE J[1], JAYASENA YAA[2], DILANKA S[1]
[1]University of Colombo, Colombo, Sri Lanka, [2]Lady RIdgeway Hospital, Colombo, Sri Lanka

POSTER ABSTRACTS

Purpose: To document spasm control, subsequent evolution and developmental progress after treatment of infantile spasms with short course of steroids/vigabatrin.
Method: Study was performed in Sri Lanka. Children presenting with IS were treated according to United kingdom Infantile Spasms Study (UKISS) protocol. Seizure cessation was documented on last (14th) day of therapy. The children were prospectively followed up for recurrence of spasms and developmental outcome. Seizure diary was utilized to assess spasm control and developmental gain was recorded at 3, 6 and 12 months post treatment.
Results: Thirty patients were treated. Median age at onset was 5 months. Normal development prior to onset of spasms was seen in 12. Focal seizures preceded onset in 6; occurred concurrently with IS in 4.Twenty seven were given steroids (18-prednisolone and 9- IM ACTH), three were given vigabatrin.
Twenty were spasm free (66%), while another 5 had > 50% reduction on 14th day of treatment. Hypsarrhtymia was seen in 28 but continued only in 2 after treatment.Follow up was 3 months for 4, 6 months for 7 and 12 months for 17 children. Seven developed relapse of spasms. Median timeto relapse was 3 months (0.5-18 months). Frequency of spasms during relapse was lower (< 50%) than at the onset.Fifteen had regression of milestones with onset of spasms. Development showed improvement in all but two children.
Conclusion: IS responded well to initial treatment with steroids/ vigabatrin. In spite of relapse or continued spasms, the development progressed in a majority.

p167
A child who falls with a tap on the head
GOVINDARAJAN R
Cleveland Clinic Florida, Weston, Florida, USA

A 9-year-old girl presented with reflex atonic seizures since age 2 years evoked only by a tap on back of the head. Myoclonic semiology could not be ruled out. Inter-ictal EEG recorded generalized bursts of posterior-central dominant sharp waves. She had history of hypoglycaemic encephalopathy secondary to congenital hyperinsulinism. Her father has hot-water epilepsy. Seizures have been well controlled with oral valproate. Reflex seizures evoked by a tap are mostly myoclonic. Rare cases of atonic seizures have also been described.Differential include hyperekplakia, startle and somatosensory seizures.

p168
Devastating epileptic encephalopathy following generalized febrile status epilepticus
JADHAV VM
Arya Child Epilepsy and Neurology Clinic, Kolhapur, India

Purpose: Devastating epileptic encephalopathy following generalized febrile status epilepticus was seen in few children who had normal neurological investigations during the acute episode and were completely normal before. They experienced frequent seizures, remained severely retarded and did not regain language function thenafter. Purpose of this study was to find out why some children developed devastating epileptic encephalopathy and some recovered completely without any significant neurological morbidity.
Method: This five years retrospective study consists of 32 children between 3 to 9 years who experienced generalized febrile status epilepticus and were completely normal neurologically before. CSF study, CSF PCR for herpes and Japnese B, 1.5 T MRI were normal. Interictal EEGs showed diffuse slowing.

POSTER ABSTRACTS

Results: Nine children required IV Midazolam drip to control the status. Four children developed rebound seizures immediately after withdrawing midazolam drip and one recurred after 72 hours of discontinuation. Anti epileptics could be withdrawn safely in 27 children by 3 months who had no neurological morbidity. Five children continued to experience intractable daily seizures predominantly of complex partial variety and occasionally secondarily generalized in the range of 5-25 episodes a day. They remained severely retarded, and did not regain the lost language function with maximum follow up of 5 years. Drooling and dysphagia was seen in all children. The most efficacious drugs in long term were topiramate, oxcarbazepine and clobazam.
Conclusion: The mystery remains why only five chidren developed devastating epileptic encephalopathy. Prolonged encephalopathy and intractability was seen in this group. Age did not influence the outcome;

p169
Comparison of melatonin and sleep-deprivation for induction of sleep during paediatric sleep EEG acquisition
SINGH H, BRASS J, RINEY K
Department of Neurosciences Mater Children Hospital, Brisbane, Australia

Purpose: To determine whether melatonin administration is an effective alternative to sleep deprivation for inducing sleep during paediatric sleep EEG recordings.
Method: A retrospective review was undertaken of sleep EEGs carried out at the Mater Children's Hospital, Brisbane, from August 2008 (when melatonin-induced sleep EEG's were first introduced) until May 2010. For melatonin-induced EEG, 3-6mg melatonin was administered 20 minutes prior to the EEG recording. For sleep-deprivation, parents were asked to reduce the child's overnight sleep by 50% the night before the sleep EEG. Data extracted included age and sex of patient, method of sleep acquisition (melatonin vs sleep deprivation), duration from onset of EEG to onset of sleep and whether failure to achieve sleep occurred. If the patient had pre-existing behavioural difficulties or pervasive developmental disorder (PDD), this was noted.
Results: Sixty eight sleep EEG recordings were attempted in the time period of this study, 34 with melatonin and 34 with sleep-deprivation. The melatonin group included more patients with PDD than the sleep-deprived group (11/34 vs 1/34). Overall, 24/34 (71%) of the melatonin group and 28/34 (82%) of the sleep-deprived group achieved sleep. When children with PDD are excluded from both groups, equal proportions of the melatonin and sleep-deprived groups achieved sleep (20/23, 83% vs 27/33, 82%).
Conclusion: Melatonin is as effective as sleep deprivation for acquiring sleep during EEG recording in children without PDD. Melatonin administration is safe and is not accompanied by the adverse behavioural and family consequences that accompany overnight sleep deprivation of young children.

p170
Febrile infection-related epilepsy syndrome (FIRES) does not respond to rituximab or vagus nerve stimulation: a case report
HOWELL KB, HARVEY AS, BAILEY CA, MAIXNER W, FREEMAN JL
Royal Children's Hospital, Melbourne, Australia

POSTER ABSTRACTS

We report the case of a previously well, developmentally normal 14-year-old boy with acute onset of refractory status epilepticus in the setting of an encephalitic illness. Initial MRI brain was normal. No infective pathogen was identified. CSF neopterin was markedly elevated. Anti-glutamic acid decarboxylase (GAD) antibodies became positive during the illness. A diagnosis of FIRES was made early in the illness. Given the presumed autoimmune basis of FIRES, immunotherapy with corticosteroids, intravenous immunoglobulin and plasma exchange were used early. Aggressive anticonvulsant therapy was instituted. Only barbituates at doses high enough to induce burst-suppression controlled the repetitive independent bilateral focal seizures. Two therapies whose use was not previously reported in FIRES were tried - the anti-CD20 agent Rituximab and left vagus nerve stimulation. Neither modified the refractory seizure disorder. Intensive care treatment was withdrawn three weeks into the illness and the patient died. Postmortem examination of the brain was essentially normal, without inflammatory changes.

p171

Developmental GABAA receptor-mediated depolarization/excitation may contribute to immediate and delayed adverse effects of neonatal anesthesia

MARTYNYUK A, CAO W, SHAH H, EDWARDS D, GRAVENSTEIN N, SEUBERT C
University of Florida, Gainesville, USA

Purpose: Numerous clinical reports of anesthesia-associated paradoxical hyperexcitatory events in combination with retrospective analyses of human patient data raise concerns about the safety of anesthesia in neonates and infants. The underlying mechanisms of the anesthesia-related defects remain poorly understood. We hypothesize that anesthetics that enhance GABAA receptor activity may exacerbate GABA-induced depolarization resulting in seizures, neurotoxicity, and alteration of other developmental processes.
Method: The acute and delayed effects of sevoflurane and isoflurane were studied in postnatal days 4-21 (P4-P21) rats by measuring electroencephalographic activity, activated caspase-3 levels, hippocampal long-term potentiation (LTP) and prepulse inhibition (PPI) of acoustic startle response (sensorimotor gating function). Anesthesia was induced and maintained with 6% (3.4%) and 2.1% (1.2%) sevoflurane (isoflurane) over 3 min and 0.5-6 hrs, respectively.
Results: Electroencephalographic seizures were detected in more than 50% of P4-P7 rats during 2.1% sevoflurane anesthesia. Such seizures abated in older rat pups. Anesthesia of P4 rats with sevoflurane for 6 hrs caused a significant increase in level of activated caspase-3 and impaired LTP induction, measured 1 and 14-17 days after exposure to sevoflurane, respectively. These rats exhibited significantly impaired PPI of startle measured at P19-P24. Bumetanide (5 µmol/kg, I.P.), a specific inhibitor of the Na^+-K^+-2Cl- co-transporter, administered prior to sevoflurane, prevented seizures, neurotoxicity and sensorimotor gating deficit. Anesthesia with isoflurane caused less seizure-like activity but disrupted PPI of startle and increased apoptosis - effects that responded to pre-treatment with bumetanide.
Conclusion: These findings support a potential contribution of $GABA_A$ -mediated depolarization/excitation to complications of neonatal anesthesia.

p172

Vigabatrin-related neurotoxicity in an infant: first reported human case detailing clinical, histopathological and radiological findings

MALONE SA[1,2], KAMPPI H[1], WALSH M[1], WALLACE G[1,2], DEXTER M[3], LAWSON J[4], SUGO E[5], BYE A[4]
[1]Mater Children's Hospital, Brisbane, Australia, [2]Royal Children's Hospital, Brisbane, Australia, [3]Westmead Hospital, Sydney, Australia, [4]Sydney Children's Hospital, Randwick, Sydney, Australia, [5]Prince of Wales Hospital, Sydney, Australia

POSTER ABSTRACTS

Purpose: This is the first human case of Vigabatrin (VGB) related neurotoxicity evidenced by clinical, histopathological and radiological findings.
Method: The patient developed infantile spasms at age 3 months. Initial treatment with VGB (maximal dose 135 mg/kg) was unsuccessful, associated with reduced alertness and activity, and treatment was ceased after 1 month. Spasms recurred at 6 months and were medically refractory. A second course of VGB (maximum 120 mg/kg) commenced at 7 months.
Results: MRI brain imaging was at 3T: at 3 months, normal; but, when performed 5 weeks following VGB recommencement, revealed symmetrical diffusion restriction in the bilateral thalami and dorsal longitudinal tracts of the brainstem, consistent with VGB toxicity. Extensive metabolic investigations were negative. MRI at age 11 months (7 weeks after cessation of VGB) revealed minor resolving abnormality in the dorsal brainstem. Following cessation of VGB the infant had improved tone and visual interaction. Surgery (right temporoparieto-occipital resection) was performed at age 11 months based predominantly on PET and EEG findings, and resulted in early post-operative seizure freedom. Histopathology revealed focal cortical dyplasia (type 1A/1B), but with co-existing vacuolation of white matter in densely myelinated fibre tracts, supportive of VGB toxicity. Post-operative MRI (14 weeks after VGB cessation) revealed no changes attributable to VGB.
Conclusion: This is the first reported human case of Vigabatrin neurotoxicity detailing clinical, histopathological and imaging findings. Risk factors included young age and, as noted in animal studies, high dose treatment. MRI changes appeared more rapidly reversible than histopathological changes.

p173
Assessment of the visual field defect associated with vigabatrin therapy in epileptic children
CHUNG S
Kyung Hee University Hospital, Seoul, Korea, Republic of

Purpose: To investigate the visual field defect associated with vigabatrin treated epileptic children.
Method: One hundred thirty five epileptic children older than 8 years old (range: 8 years to 23 years) receiving vigabatrin in combination with other AEDs were included in this study. Of 135 patients, 28 were excluded because of a lack of cooperation in examination, and having underlying brain diseases. Visual field testing was performed by kinetic and/or static perimetry using Goldmann and Humphrey automated perimeter. In cases with showing suspicious or abnormal findings, re-examination was performed 6 · 12 months later.
Results: The ages at initial start of vigabatrin therapy ranged from 1.5 years to 18 years (mean age, 6.2 years). Duration treated with vigabatrin ranged from 24 to 65 months (mean, 36.2 months). In eleven (10.0%) of 110, showed a concentric visual field constriction (VFC), 8 of 11 patients were asymptomatic and 3 were symptomatic subjectively. Patients showing abnormal VFC were discontinued the vigabatrin treatment, and to perform the re-examination of visual field 6 · 12 months later according to patients' condition. VFC were disappeared without any other medical management.
Conclusion: We could not offer any conclusive results whether those were false positive because of poor cooperation in children or natural reversibility of vigabatrin-associated VFC. In conclusion, large scale studies are needed to assess the factors both for the occurrence and reversibility of vigabatrin induced VFC.

p174
Eight cases of malignant epilepsies with intractable epileptic seizures and mental retardation
FUKUDA M, YAMAMOTO H
St. Marianna University Hospital, Kawasaki, Japan

POSTER ABSTRACTS

Purpose: Although epilepsies in children are relatively benign disorder, we sometimes experience some patients with intractable epileptic seizures and severe mental retardation. These so-called malignant epilepsies (ME) include the age-related epileptic syndromes characterized by a variety of seizure manifestations, distinctive EEG patterns and poor outcomes, and intractable epileptic seizures refractory to conventional anti-epileptic drugs.
Method: We report 8 cases of ME which we experienced at our university hospital in the past three years. The subjects are 3 patients with West syndrome (WS), 2 patients with intractable epileptic seizures of unknown etiology and 1 patient with congenital brain malformation, subsequent neonatal seizures caused by hypoxic-ischemic encephalopathy and cortical dysplasia respectively (6 males and 2 females).
Results: The average age of seizure onset was 7 months. All cases were refractory to conventional anti-epileptic agents although 2 cases of WS responded to ACTH.
Conclusion: In this report we discuss advances in our understanding of the therapy in ME and also highlight recent progress in the efficacy of zonisamide.

p175
Prevalence of genetic and chromosomal syndromes and congenital brain malformations among epilepsy patients
TYSYACHINA MD[1,2], KUZINA NY[1], MUKHIN KY[1,2], PETRUKHIN AS[1], MELIKYAN EG[1]
[1]Child Neurology Clinic of Russian State Medical University, Moscow, Russian Federation, [2]Svt. Luka's Institute of Child Neurology and Epilepsy, Moscow, Russian Federation

Purpose: To study the prevalence of genetic and chromosomal syndromes and congenital brain malformations among epilepsy patients.
Method: We observed 2463 patients aged 6 months to 38 years, with epilepsy. Genetic and chromosomal syndromes and congenital brain malformations were found in 40 patients (1.6%) aged 3 months to 34 years.
Results: Onset of seizures was between 3 month and 16 years. The forms of epilepsy were: symptomatic focal epilepsies - 50%; various forms of focal epilepsy with good long-term prognosis and BEDC on EEG - 40%; and group of patients without epileptic seizures, with BEDC on EEG - 10%. Epileptic seizures were: focal - 77.8%; pseudo-generalized seizures, caused by SBS on EEG - 61.1%. Genetic and chromosomal syndromes and brain malformations were found following: chromosomal inversions, deletions and trisomies (9 cases), triplo-X syndrome (1 cases), ring chromosome 20 syndrome (1), Angelman syndrome (5), Rett syndrome (3), Down syndrome (3), Smith-Magenis syndrome (2), Miller-Dieker syndrome (2), Prader-Willi syndrome (1), Kabuki syndrome (1), Cornelia de Lange syndrome (1), Opitz-Frias syndrome (1), Borjeson-Forssman-Lehmann syndrome (1), Lowe syndrome (1), hypomelanosis of Ito (1), tuberous sclerosis (5), multinodular subependymal heterotopia (2).
Conclusion: In our study epilepsies with good long-term prognosis and BEDC on EEG, and only BEDC on EEG without epileptic seizures («idiopatic - like course») - were observed in 50% of cases. The remission of seizures in the general group was in 40% of patients. Benign outcome of epilepsy in this group can be caused by «hereditary impairment of brain maturation» [H. Doose, 2003].

p176
Vascular endothelial growth factor C (VEGF-C) system expression in cortical tubers of the tuberous sclerosis complex
ZHANG C-Q[1], SHU H-F[1], YIN Q[2], AN N[1], ZHAO B-Y[1], SONG Y-C[1], LIU S-Y[1], YANG H[1]
[1]Department of Neurosurgery, Xinqiao Hospital, Third Military Medical University, Chongqing, China, [2]Department of Rehabilitation and Physical Therapy, Southwest Hospital, Third Military Medical University, Chongqing, China

POSTER ABSTRACTS

Purpose: Tuberous sclerosis complex (TSC) is an autosomal dominant genetic disorder resulting from inactivating mutations in one of two genes, TSC1 and TSC2. Cortical tubers, a typical pathological hallmark of TSC in the brain, are frequently associated with pediatric intractable epilepsy. Recent evidence has shown vascular endothelial growth factor C (VEGF-C), the most important lymphangiogenic factor, plays significant roles in central nerve system. The aim of this study was to define the expression and cell-specific distribution of VEGF-C system in cortical tubers.
Method: The expression of VEGF-C system was studied in tissue homogenate of cortical tuber from patients with TSC using RT-PCR and western blot analysis. Immunostaining was used to observe changes in VEGF-C system expression in tubers versus age-matched control cortex (CTX).
Results: The mRNA and protein levels of VEGF-C,VEGFR-2 and VEGFR-3 were obviously upregulated in cortical tubers compared with CTX. Histologically, neuronal immunostaining of VEGF-C and its receptors was detected in CTX, mainly in pyramidal neurons. In cortical tubers, VEGF-C and both VEGFR-2 and VEGFR-3 were highly expressed in dysplastic neurons (DNs) and giant cells (GCs). Double labeling also revealed that the DNs and GNs expressing VEGF-C comprise a homologous population with characteristics of immature myelinated A-fiber neurons in the cortex. Moreover, most reactive astrocytes expressing VEGF-C were observed in tubers, whereas the immunoreactivity in vascular endothelial cells was weak
or undetectable
Conclusion: These findings suggest that VEGF-C system involves in the genesis of cortical tubers.

p177
Clinical outcome and long term prognosis of childhood epilepsy
YUNG A, WONG V
Department of Paediatrics and Adolescent Medicine, University of Hong Kong, Hong Kong, Hong Kong

Purpose: Epilepsy is a common paediatric condition. A minority of patients will develop medical intractability. The aim of our study is to look into the interim 2-year and long-term clinical outcome of a group of childhood-onset epilepsy patients.
Method: We studied our cohort of 536 children, with childhood-onset epilepsy, managed in the period January 1, 1979 - December 31, 2006, at the Epilepsy Clinic, Department of Paediatrics, University of Hong Kong. All patients were put on anti-epileptic drug (AED) and were followed up for ≥ 24 months after AED initiation. Patients with poor drug compliance or no AED treatment were excluded. We studied the patients at their 2-year follow up (FU) and at last, terminal FU. Outcome was defined as responders (seizure freedom for ≥12 months); uncontrolled epilepsy (UE) (seizure-free for < 12 months), and relapsers (initial seizure freedom for ≥12 months, followed by subsequent breakthrough seizures before 2 year or last FU).
Results: At 2 year FU for 505 patients with available data, 41.6% had UE, 44.8% responders and 13.7% relapsers. At terminal FU for a medial period of 10.0 years (range 2-30 years) for all 536 children, 31.9% had UE, 57.6% responders and 10.4% relapsers. Early clinical indicators associated with UE were identified.
Conclusion: We try to predict medical intractability using simple clinical data in the early clinical course. Worsening of post-treatment seizure frequency as compared to pre-treatment, young age of onset, high initial seizure frequency were associated with poor seizure outcome.

p178
Predictors of intractable epilepsy in childhood
MARTHATI MB
Andhra Medical College, Visakhapatnam, India

Purpose: To identify the predictors of medially intractable epilepsy in children.

POSTER ABSTRACTS

Method: A retrospective analysis of a cohort of 615 children with epilepsy was performed. Only children with more than 2 years of follow-up were included. Etiology and epilepsy syndromes were classified using the International League Against (ILAE) guidelines. Intractable epilepsy was defined as those with more than 1 seizure per month over 2 years with failure of three or more anti-epileptic drugs. Descriptive statistics were done; odds ratio (OR) was calculated for various parameters to determine predictors of intractable epilepsy. Univariate & multivariate analysis was performed:
Results: There were 615 children (age 1 month to 16 years), accounting for 24% of epilepsies attending the epilepsy clinic. The mean duration of follow up was 2.8 years (range 2·9 years). 63% of children had symptomatic epilepsies, 19% idiopathic and 17% cryptogenic. Refractory epilepsy was found in 22%; the predictors of intractable epilepsy in childhood epilepsies {OR (95% CI)} were male sex 1.5 (1.0·2.2), age of onset of epilepsy less than 1 year 2.8 (1.9·4.2), symptomatic epilepsies 3.848 (2.35·6.29), and neonatal seizures 2.85 (1.66·4.88).
Conclusion: The factors that predict intractability in childhood epilepsies are age of onset of epilepsy less than 1 year, symptomatic epilepsies and neonatal seizures. As perinatal insults is one of the major etiology of symptomatic epilepsies, it is an important preventable cause in children with epilepsy in developing countries.

p179
Epileptic encephalopathies related to mutations of the SCN1A gene: a single center experience
PILLAI S[1], DOUGLAS L[2], GILL D[1]
[1]TY Nelson Department of Neurology and Neurosurgery, The Children's Hospital at Westmead, Sydney, Australia, [2]Epilepsy Research Centre, The Children's Hospital at Westmead, Sydney, Australia

Introduction: Mutations in the *SCN1A* gene can lead to severe epileptic encephalopathy, the phenotype, however, may be variable. The most severe phenotype, SMEI, or Dravet syndrome, is associated with refractory epilepsy, developmental regression, and severe cognitive impairment. Further delineation of the phenotypic spectrum of early onset *SCN1A* related encephalopathies may lead to earlier recognition, treatment and prognosis.
Methods: All patients who had tested positive for a mutation of the SCN1A gene were included and the clinical phenotype, response to medications, and seizure frequency was recorded.
Results: 17 children tested positive for mutations in *SCN1A*. 12 patients had SMEI and 5 patients SMEB. Of the SMEB patients four had mild developmental delay only, and all but one of the 12 patients with SMEI had moderate or severe developmental delay. All but two children had normal development prior to seizure onset. A focal seizure was present in 12 patients at first presentation. Seizures were provoked by either fever, a febrile illness or immunization in 15 children. At most recent follow up, all patients have refractory epilepsy requiring treatment. One patient with SMEB at the age of 9 has stereotyped focal seizures only, with very rare GTCS. Two children with disabling pattern sensitive seizures improved markedly with the addition of stiripentol.
Conclusion: *SCN1A* mutations provided confirmation of the clinical suspicion of Dravet syndrome with patients having either the SMEI or SMEB phenotype. Developmental delay was milder in patients with the SMEB phenotype, and in those with seizure onset greater than 12 months.

p180
Study of the health-related quality of life in children with epilepsy
MELIKYAN EG[1], MUKHIN KY[2]
[1]Department of Neurology & Neurosurgery, National Clinical-Surgical N.I. Pirogov Center, Institute of the Medical Postgraduate Studies, Moscow, Russian Federation, [2]Department of Neurology & Neurosurgery, Russian State Medical University, Moscow, Russian Federation

POSTER ABSTRACTS

Purpose: To date, no Russian translations of HRQOL tools in children with epilepsy had been done. The aim was to evaluate the current measures of HRQOL in children with epilepsy and to produce a culturally appropriate translation of the chosen questionnaires.
Method: Analysis of the tools proposed for the assessment of HRQOL in children with epilepsy was provided. Forward and backward translations, expert panel review, and pilot testing produced a Russian translation.
Results: Following the research of the English literature at least twenty generic and seven specific measures of HRQOL in children with epilepsy were identified. Analysis of the psychometric properties HRQOL instruments suggested that two questionnaires were the most promising: 1/ Impact of Epilepsy Schedule (IES) and 2/ Quality of Life in Childhood Epilepsy Questionnaire (QOLCE). Both questionnaires seemed to be useful for the estimation of HRQOL's different dimensions, convenient, prospective and sensitive to change. Regarding possible differences in the perception of HRQOL between cultures, a systematic approach to translation and cultural adaptation of those questionnaires was used to ensure content equivalence and validity. In some cases modification of vocabulary, grammar, and sentence structure was necessary to maintain a semantic accuracy and grammatical fluency in
Russian language.
Conclusion: There is very good evidence of the reliability and validity of IES and QOLCE questionnaires in children with epilepsy. The Russian translation and cultural adaptation of these tools were conducted. The further validation of the translated scales in Russian population should be performed.

p181
Seizure and cognitive outcome in Thai children with benign Roland epilepsy
VISUDTIBHAN A, SAKPICHAISAKUN K, KHONGKHATITHUM C, THAMPRATANKUL L, CHIEMCHANYA S, VISUDHIPHAN P
Department of Pediatrics, Facutly of Medicine, Ramathibodi Hospital, Mahidol University, Bangkok, Thailand

Purpose: To determine clinical course, outcome and co-morbidity of children with BRE
Method: Children diagnosed with BRE at a university hospital between 2002 and 2009 were recruited. Medical records were reviewed to obtain demographic data, physical signs, investigation, treatment and clinical outcomes. The outcomes were categorized into 4 groups; 1) group I: no treatment, 2) group II: no seizure, current taking antiepileptic drug, 3) group III: seizure-free after discontinuation of antiepileptic drug, and group IV: multiple recurrent seizures despite taking medication. To determine the cognitive outcomes; Wechsler Intelligence Scale-III (WISC-III) and Wide Range Achievement Test (WRAT) would be determined whenever applicable.
Results: Forty-six patients (23 boys and 23 girls; mean age of seizure-onset 8.1 + 2.2 years) were included in this study. EEG showed typical centrotemporal spikes with tangential dipole in 43 tracings (96.5%). Seven children (15.2%) were lost to follow-up. There were 3 (6.5%), 31 (67.4%), and 5 (10.9%) patients who were categorized into group I, II and III, respectively. Among three groups, there were no statistical significance between age, gender and duration of seizures. Six of 37, who had WISC-III test, had subnormal IQ. Among 30 patients, who received WRAT; learning disability was evident in 20 (66.7%). There was no correlation either between the educational problems and number of seizures or between the educational problems and the absence of the tangential dipole.
Conclusion: This pilot cross-sectional study in Thai children demonstrated that children with BRE might have neuropsychological impairment and learning disability despite the favorable seizure outcome.

POSTER ABSTRACTS

p182

Characteristics of centrotemporal spikes in Panayiotopoulos syndrome

TOMONOH Y, FUJITA T, IHARA Y, NAKAMURA N, NIMOMIYA S, KODAMA R, IDEGUCHI H, INOUE T, YASUMOTO S, HIROSE S

Department of Pediatrics, Fukuoka University, Fukuoka City, Japan

Purpose: To identify the characteristics of centrotemporal spikes of patients with Panayiotopoulos syndrome (PS) who later progress to benign childhood epilepsy with centrotemporal spikes (BCECT).
Methods: We performed conventional EEG tests for 51 children with PS who had been treated at our department from 1996 to 2009. Centrotemporal spikes recorded were compared between patients who had progressed to BCECT and those who did not.
Results: Eleven children had EEGs showing centrotemporal spikes; two of them showed typical Rolandic discharges which consists of a small positive spike, a negative spike (biphasic) and a slow wave. One of the two with typical Rolandic discharges showed a yearly increase in spike number and evolved into BCECT. The other showed several Rolandic discharges whereas did not develop BCECT. The centrotemporal spikes of the remaining 9 children were irregular or not associated with the small positive spikes. None of them progressed to BCECT.
Conclusion: PS with increasing typical Rolandic discharges is likely to evolve into BCECT.

p183

Steroid pulse therapy for a case of frontal lobe epilepsy with antibodies to glutamate receptor (GluR)

IHARA Y[1], TOMONOU Y[1], FUJITA T[1], IDEGUCHI H[1], INOUE T[1], YASUMOTO S[1], TAKAHASHI Y[2], HIROSE S[1]

[1]Department of Pediatrics, Fukuoka University, School of Medicine, Fukuoka, Japan, [2]National Epilepsy Center Shizuoka Institute of Epilepsy and Neurological Disorders, Shizuoka, Japan

Objectives: We report a case of intractable frontal lobe epilepsy with the autoantibodies against glutamate receptors (GluR). Steroid pulse therapy is very effective, and this case presents a good clinical course and long-term prognosis.
Case report: The patient is an eight year old girl who, for several months, had been having seizures both when asleep and awake. During these attacks, she sometimes lost consciousness and was unable to move, and sometimes moved and looked about restlessly. Each episode lasted about a minute. Interictal EEGs showed diffuse irregular high voltage spike and waves, with rhythmic sharp waves seen predominantly in the frontal areas. These EEGs were improved by diazepam. We diagnosed frontal lobe epilepsy and we started the anti-epileptic drug, carbamazepine. Still, the seizures continued. Other antiepileptic drugs-valproic acid, zonisamide, clobazam, gabapentin, fenitoine, lamotorigine-were also ineffective. Seizure frequency remained unchanged. Autoantibodies against Glu R tested positive to IgM-ε2 in serum and to IgG-ε2 in CSF. We performed steroid pulse therapy, which proved very effective, gradually decreasing seizure frequency. The patient is now seizure-free; her EEG shows no abnormal discharges; and her mental state is normal.
Conclusion: Steroid pulse therapy was effective in the treatment of a case of intractable frontal lobe epilepsy. The mechanism behind the anticonvulsant action of corticosteroids had reported some factors. Our case showed positive autoantibodies to glutamate receptors. We should consider the possibility that there is a self-immunologic mechanism for intractable epilepsy, and that steroid therapy may be an effective treatment for it.

POSTER ABSTRACTS

p184

Drug withdrawal during inpatient evaluation for pediatric epilepsy surgery: outcomes in a newly established service

SINGH H, CLARK D, GURR S, RICHMOND J, MALONE SA
Royal Children's Hospital, Brisbane, Australia

Purpose: Drug withdrawal is often necessary in the Epilepsy Monitoring Unit (EMU) to facilitate seizures, however there is minimal literature on this subject. This is a review of outcomes from a newly established service with limited resources.
Method: Retrospective data review of patients with intractable epilepsy admitted for pre-surgical evaluation, including drug withdrawal, at the Royal Children's Hospital EMU. This EMU has limited access to SPECT (average 4-6 mornings/month). During periods of drug withdrawal patients have a carer present, intravenous access, continuous overnight oximetry, an individualized management plan for seizures, and daily review by an epileptologist +/- epilepsy fellow. Data analysed included seizure history, seizures during admission, status epilepticus (SE), the drug withdrawal strategy, success of admission (i.e. typical seizure achieved, +/- SPECT), and emergency treatments.
Results: There were 31 admissions (28 patients) from May 2008 to May 2010. Drug withdrawal was individualized; withdrawal was full on 3 occasions, partial in 28, and commenced on admission in 17. Overall seizure frequency increased during 15 admissions, decreased in 4, and remained unchanged in 12. Admission was successful in 25 patients (typical seizures), with SPECT achieved in 17. SPECT could not occur on 3 occasions due to lack of seizures in the alloted time. Acute benzodiazepine treatment was required on 7 occasions, including 3 episodes of SE. There were no ICU admissions.
Conclusion: Tailored drug withdrawal can be successful in the majority of patients, despite limited resources. There is a requirement for individualized patient care and monitoring to provide the safest outcome.

p185

Clinical profile of 13 children with severe myoclonic epilepsy of infancy in Hong Kong

FUNG C-W, WONG V
Department of Paediatrics and Adolescent Medicine, The University of Hong Kong, Queen Mary Hospital / Duchess of Kent Children's Hospital, Hong Kong, Hong Kong

Purpose: This study delineates the clinical profile of children suffering from severe myoclonic epilepsy of infancy (SMEI) or a similar phenotype without myoclonus (SMEIB) with a mutation in the SCN1A gene in Hong Kong.
Method: From 1985 to 2010, 13 children (7 males and 6 females), aged 2 to 29 y, with a phenotype of SMEI / SMEIB were confirmed to carry a mutation in the SCN1A gene (de novo mutation: n=10, 83%).
Results: All 13 patients (SMEI = 9, SMEIB = 4) had seizure onset before 1 year old (3 - 10 months). Majority (n=11, 85%) were precipitated by infection or vaccination. All but 2 had a history of status epilepticus, either febrile or unprovoked. All had unprovoked seizures after onset with ≥ 2 seizure types (generalized tonic clonic seizure, focal seizure, myoclonic seizure and atypical absence seizure). Focal clonic seizures involving either side of the body were present in 11 patients (85%). Physical examination was normal in 9 (69%). 2 had cerebellar ataxia and 2 demonstrated persistent intermittent myoclonus. All had normal development prior to seizure onset with progressive developmental decline resulting in either global developmental delay or mental retardation in 12 children (92%). 8 (62%) were confirmed
to have autism spectrum disorder after the onset of epilepsy. Response to clobazam, valproate and topiramate was promising. 10 (77%) had intractable seizures despite 2 to
3 anticonvulsants.

POSTER ABSTRACTS

Conclusion: Our cohort was similar to others. Autism spectrum disorder was frequent in our study, which was not commonly recognized.

p186
Sub-acute sclerotic panencephalitis (SSPE) is still prevalent in under-developed countries like Pakistan
HEREKAR AD
Neuro Diagnostic Centre, Karachi, Pakistan

Purpose: A prospective study with EEG abnormalities, the MRI changes and the presence of other criteria in SSPE patients.
Method: This is a prospective study of 10 consecutively clinically diagnosed SSPE patients reporting to a private neurology centre in Karachi, Pakistan between 2007 to 2009. The inclusion criteria were recurrent myoclonic jerks, mental decline, onset of age under 18 years and typical EEG changes. All patients underwent an EEG. Measles antibody titus were not available and thus other criteria were used for diagnosis.
Results: Paroxysmal synchronous discharges (PSDs) were found in all patients; however, it was noted that background EEG abnormality was found only after the first year into the disease as most patients at and immediately post onset had a normal EEG background. Two patients had visual difficulties with optic atrophy and 1 patient had abnormal generalized chorioathetoid movements together with myoclonus. Imaging abnormalities were found in 5 out of 10 patients, 1 hyper intense signal in both basal ganglia, abnormal white matter signals in two patients resembling leukodysrophy and multiple hyper intense signals involving the grey matter were found in 2 patients with MRI abnormalities.
Conclusion: SSPEs can be clinically diagnosed on the basis of other criteria even in the presence of non-availability of anti-body titres against measles virus in a third world country like Pakistan.

p187
Early identification and evaluation of neonatal seizures including neurodevelopmental impairments in the hospital admitted newborns

BANU SH[1], AKHTER R[2], KHAN NZ3
[1]Institute of Child Health & SSF Hospital, Neurosciences Unit, Dhaka, Bangladesh, [2]Institute of Child Health & SSF Hospital, Developmental Therapy ICH, Dhaka, Bangladesh, [3]Institute of Child Health & SSF Hospital, Development and Neurology Unit, Dhaka, Bangladesh

Background: Burden of neurodevelopmental impairments (NDIs) including seizures is very high in Bangladesh. There are multiple reasons for this including pregnancy and birth related problems, poor antenatal care, intra-uterine infection, intra cranial infection in early childhood with their consequents. A large proportion of them are probably preventable and/or modifiable by a simple mechanism in early period of life.
Objective: Early identification of neurodevelopmental impairments including seizures, intervention and systemic monitoring the outcomes using a quick assessment tool.

POSTER ABSTRACTS

Methods: Rapid neurodevelopmental assessment tool (RNDA) was developed and standardized by the Child Development and Neurology team in Dhaka (NZ Khan et al., 2010). Identification of babies at risk for NDIs in any of eight domains (gross motor, fine motor, vision, hearing, speech, behavior, cognition and seizures) is possible using this tool by multidisciplinary professionals. We used the tool for the babies admitted at the neonate special care unit of our hospital, situated in an area where the majority of mothers are poor, garments factoru workers.
All neonates were brought to the hospital with definite problems. RNDA was performed by a developmental therapist on recovery from their acute illness, and after discharge one monthly. Nuro-developmental intervention were provided. Re-assessment results were compared with the 1st one, their findings were analized by ANOVA to identify the most significant factor associated with poor outcomes.
Result: Total 119 babies were enrolled during August 2009 to April 2010, 62% were from the neighboring area, over 80% were born at home, 88% full-tem, 26.8% gave history of difficult labor, 49.5% had delayed spontaneous cry. Mean age at 1st assessment was 37 days. The immediate cause of hospital admission was pneumonia or septicemia in 40.3%, severe perinatal asphyxia (PA) and neonatal seizures (NS) in 39.5%, and babies of high-risk mothers in 20.2%. On 1st assessment 71% were identified at risk in different domains including 50% with seizures and 48% motor deficit. The state of NS and later onset seizures was analyzed. With appropriate intervention over 29% babies recovered their NDIs. Neonatal pneumonia or septicemia and severe PA had significant correlation with severe neurodevelopmental-deficits.

p188
Effects of topiramate on the problem solving abilities and language in newly diagnosed pediatric epileptic patients
KIM SJ, CHOI YM
Department of Pediatrics, Department of Clinical Speech Pathology, Chonbuk National University Medical School, Jeonju, Korea, Republic of

Purpose: The aim of this study was to investigate the effects of topiramate on language and problem solving ability in newly diagnosed pediatric patients with epilepsy.
Patients and Methods: Newly diagnosed 32 epileptic patients were assessed standard language tests and problem solving abilities. First test data were collected right before the topiramate monotherapy started and the treatment remained as monotherapy until the second tests were performed. Topiramate therapy dose was 1mg/kg/day for the first one or two week; increased to follow by slow increase of dose every 2week intervals until a maintenance dose of 5mg/kg/day or 200mg/day reached. Language tests were included language problem solving ability test (TOPS), Peabody Picture Vocabulary Test, Velopharyngeal articulation screening test.
Results: TOPS showed that the abilities of problem solving were worsen after initiation of topiramate in patients. All parameters (Determine Causes, Problem Solving, Predicting) were significantly reduced after initiation of topiramate ($P < 0.05$). Patients with topiramate showed shorten the mean length of utterance in words ($P < 0.05$), answers were ambiguous during the test, and they also showed that the difficulty to select the appropriate word, took more time to answers, and used the wrong grammar. However, there was no statistically significant change in receptive language and precise articulation of patients even after taking topiramate.
Conclusion: Our data suggests that topiramate could be negative effects on the abilities of problem solving and language abilities. We strongly recommend that language tests should be performed during the treatment with topiramate in children.

POSTER ABSTRACTS

AED issues

p189

A comparison of buccal midazolam and intravenous diazepam for the acute treatment of seizures in children and adolescents

MAHVELATI SHAMSABADI F, TONEKABONI SH, ANVARI SS
Pediatric Neurology Research Center, Tehran, Iran, Islamic Republic of

Purpose: The purpose of the present study is to compare efficacy and safety of buccal midazolam with intravenous diazepam in control of seizures in Iranian children.
Method: This is a randomized clinical trial. 92 patients with acute seizures, ranging from 6 months to 14 years were randomly assigned to receive either buccal midazolam (32 cases) or intravenous diazepam (60 cases) at the emergency department of a children hospital. Atonic seizures, generalized tonic, and tonic- clonic convulsion cases were included irrespective of duration or etiology. The primary outcome of this study was cessation of visible seizure activity within 5 minutes from administration of the first dosage. The second dosage was used in case the seizure remained uncontrolled 5 minutes after the first one. In case the seizure remained uncontrolled after 10 minutes, either phenytoin or Phenobarbital was administered.
Results: In the midazolam group, 22 (68.8%) patients were relieved from seizures in 10 minutes. Meanwhile, diazepam controlled the episodes of 42 (70%) patients within 10 minutes. The difference was, however, not statistically significant (p=0.901%). The mean time required to control the convulsive episodes after administration of medications was not statistically significant (p< 0.095). Considering the fact that intravenous line access for diazepam infusion takes longer than administration of buccal midazolam, the second seems to be a more beneficial choice. No significant side effects were observed in either group.
Conclusion: Buccal midazolam is as effective as and safer than intravenous diazepam in control of seizures.

p190

Pharmacokinetics of carbamazepine in Mongolia for people with partial epilepsy

UURIINTUYA M[1], ULZIIBAYAR D[1], BATHUYAG P[1], NARAN G[1], BAYARMAA D[2]
[1]Health Science University, Ulaanbaatar, Mongolia, [2]Shastin Clinical Hospital, Ulaanbaatar, Mongolia

Purpose: The aim of the present study was to determine pharmacokinetic parameters (PP) of CBZ: volume of distribution (Vd), clearance (Cl) and half life ($T_{1/2}$).
Methods: 30 patients with symptomatic focal epilepsy: 14 males and 16 females included in the study. The average age was 29.9 ± 8.7 (range 16-45 years). The mean duration of partial seizure was 11.4 ± 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 ± 2.2 mg/kg. Pharmacokinetic analysis was performed by ABBOTT TDXFLX immunofluorescence autoanalyser. The blood samples were collected from patients in the morning and after 2 hours. CBZ pharmacokinetic parameters were measured by the equations
(VD, Cl and $t_{1/2}$).
Results: Therapeutic effective serum level of CMZ in 18 seizure free patients (60%) was 8.7±1.2 µg/ml (range 6.89-10.77). There was poor correlation between CBZ daily dose and serum concentration C_{max} (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, $T_{1/2}$ was 23.78 hours. No relation between CBZ daily dose (780 mg/day) and CL (r=-0.01).
Conclusion: PP 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 2009.

POSTER ABSTRACTS

p191

What happens to seizures in patients with MIE when the number of AEDs is reduced?

TRIPATHI M, AGRAWAL V, GOEL M, VASANTHA P, BHATIA R, SINGH M, PRASAD K
All India Institute of Medical Sciences, New Delhi, India

Purpose: We aimed to determine the change of seizures and recurrence and its clinical predictors in patients meeting a strict definition of drug resistant epilepsy (DRE) in the Epilepsy monitoring unit (EMU).
Method: A total of 942 patients of DRE who were being evaluated in tEMU from 1998-2008 were identified and prospectively evaluated. After their evaluation in the epilepsy monitoring unit, Anti epileptic drug (AED) adjustments to a minimum of 2 and sometimes 3 was done from the exsisting more than 3. Only 1 was reduced at the time of discharge & further drugs were reduced on the OPD basis over 1-2 months period.
Results: Out of 942 patients evaluated, 575(61%) patients were male & 367 (39%) were female. All patients were receiving 3-6 AEDs with mean of 4.24 AEDs per patient. After reduction patients received 2 or 3 AEDs with mean of 2.15 AED per patient. The estimated 6-month reduction or non-increase was 82.70%. Mean reduction of seizure per patient was 0.50 which was highly significant (p value < 0.001, 95% CI 0.39-0.61). Total 17 patients had the history of status epilepticus out of them 15(88.23%) had worsening of seizures after reduction of AEDs. Side effects were found to be significantly decreased 6 months after reduction of the AEDs (by about 50%).
Conclusion: Our results signify that it is not worthwhile to give more than 3 AEDs in patients with DRE, seizures can be controlled better & adverse reactions can be reduced with less number of drugs.

p192

In vitro transport profile of carbamazepine and its analogs by human P-glycoprotein

ZHANG C[1], ZUO Z[1], BAUM L[1], KWAN P[2]
[1]School of Pharmacy, The Chinese University of Hong Kong, Hong Kong, Hong Kong, [2]Division of Neurology, Department of Medicine & Therapeutics, The Chinese University of Hong Kong, Hong Kong, Hong Kong

Purpose: Efflux transport of antiepileptic drugs (AEDs) by P-glycoprotein (Pgp; encoded by *ABCB1* or *MDR1* gene), overexpressed in the blood-brain barrier in refractory epilepsy, may contribute to AED resistance. We used cell monolayer models to evaluate whether carbamazepine and its analogs and metabolites are substrates of human Pgp.
Method: Polarized cell lines MDCKII and LLC that were transfected with human *MDR1* were used in the concentration equilibrium transport assay (CETA). For each compound, equal concentrations were added to each side of the monolayer at the following concentrations: 2 and 10 μg/ml for carbamazepine-10,11-epoxide (CBZ-E); 5, 10, and 20 μg/ml for carbamazepine (CBZ) and oxcarbazepine (OXC); and 10 μg/ml for eslicarbazepine acetate (ESL) and (S)-licarbazepine (S-LC). Aliquots from both sides were collected at 30, 60, 90, 120, and 180 min followed by quantification of compounds by HPLC.
For compounds transported by Pgp CETAs were also performed in the presence of the Pgp inhibitor verapamil.
Results: ESL, OXC, and their active metabolite S-LC were directionally transported from basolateral to apical sides by Pgp. Pgp transported the active metabolite CBZ-E but not CBZ. The rates of transport were in the order of ESL > OXC > S-LC > CBZ-E. The transport of these drugs was inhibited by the Pgp inhibitor verapamil.
Conclusion: Carbamazepine analogs and metabolites (ESL, OXC, S-LC, and CBZ-E) are Pgp substrates, except for CBZ, implying that overexpression of Pgp may cause resistance to CBZ, OXC, or ESL.
Financial support: CUHK Direct Grant 2008.1.078.

POSTER ABSTRACTS

p193

Efficacy and safety of carbamazepine, phenobarbital, phenytoin, valproic acid, or topiramate as initial monotherapy in patients with newly diagnosed epilepsy: a long-term, head-to-head comparison in Southwest China

HU Y[1], LV Y[2], YU W[3], WANG X[1]
[1]*Department of Neurology, The First Affiliated Hospital, Chongqing Medical University, Chongqing, China,*
[2]*Department of Geriatrics, The First Affiliated Hospital, Chongqing Medical University, Chongqing, China,*
[3]*Department of Neurobiology, Chongqing Medical University, Chongqing, China*

Purpose: To compare the long-term efficacy and safety of topiramate (TPM) and four other older antiepileptic drugs (AEDs) as initial monotherapy in large-sample Chinese cohort with newly diagnosed epilepsy.
Method: 1080 patients were recruited in this study from 1995 to 2000. Carbamazepine (CBZ), phenobarbital (PB), phenytoin (PHT), valproic acid (VPA) or TPM was chosen as an initial drug to treat patients with newly diagnosed epilepsy according to individualized treatment. The retention rate was estimated by Kaplan-Meier method. Cox proportional hazard models were used to analyze the risk factors for retention rate.
Results: TPM was significantly better than CBZ (hazard ratio [HR] 0.64 [95% CI 0.42-0.99]), PHT (0.28 [0.17-0.46]), PB (0.32 [0.18-0.59]) and VPA (0.44 [0.30-0.67]). The most common cause for treatment failure was intolerability (66.1% of all withdrawals) in the first three years of medication, and inefficacy (98.2% of all withdrawals) in the later three years. TPM or traditional AED monotherapy shows similar efficacy for newly diagnosed epilepsy in the first three years. However, TPM is better for long-term therapy. Frequent seizure onsets before treatment, female gender and age were risk factors for retention rate.
Conclusion: This study suggests that TPM possesses the highest retention rate of long-term treatment comparing with CBZ, PHT, PB and VPA. PHT possesses a relative less efficacy and less safety profile. Frequent seizure before treatment might be a predictor for poor prognosis. Treatment for female patients should be more concerned on reproductive cycle and cosmetic effects. Children are better benefit from rational drug selection.

p194

Idiopathic generalized epilepsy and choice of antiepileptic drugs

AL HAMMADI F, TAHA H, AL NEAIMI O, AL SAADI T
Sheikh Khalifa Medical City, Abu Dhabi, United Arab Emirates

Purpose: To report our experience in the Middle East region of how often patients with Idiopathic generalized epilepsy (IGE) are placed on inadequate AEDs before being evaluated at an epilepsy referral clinic.
Methods: We retrospectively reviewed the EEG reports of patients at our EEG lab from the year 2004-2009. Patients with a confirmed diagnosis of IGE based on EEG criteria were identified. We reviewed their demographic data, work up, seizure frequency, and number and type of baseline AEDs prior to evaluation at an epilepsy clinic. The primary objective of this study was to study the percentage of patients with IGE receiving inappropriate AEDs. The secondary objective was to determine the percentage change in seizure response rate after evaluation at the clinic.
Results: 109 patients were identified, ages 12-56, mean 26, with seizures duration of 1 month to 30 years, mean 10 years. When initially seen, 32.11 % were on adequate AED, 25.68% were on ill-advised AEDs and 15.59 % were on various combinations. Of the patients who were receiving inadequate AEDs, 28.44 % were seizure-free and 39.44 % were doing poorly. These were converted to adequate AEDs; of which, 50% became better controlled. Futhrmore, 24.8% of patients were on no AEDs prior to referral and once placed on adequate AEDs, all became controlled.

POSTER ABSTRACTS

Conclusions: In our region, the inappropriateness of some AEDs for IGE is either not well known or neglected. Furthermore, it emphasizes the importance of establishing specialized epilepsy clinics to evaluate patients with difficult to control epilepsy.

p195

Do short-term clinical trials predict long-term response? The SKATE Australia follow-up study

SOMERVILLE ER[1,2], SKATE AUSTRALIA FOLLOW-UP GROUP
[1]Prince of Wales Hospital, Randwick, Australia, [2]University of New South Wales, Sydney, Australia

Purpose:
1. To explore the durability of benefits of treatment with levetiracetam (LEV) seen in a short-term clinical trial.
2. To measure long-term LEV retention rate in refractory focal epilepsy.

Method: Patients who took part in the Australian arm of the SKATE clinical trial, an open-label study of adjunctive LEV in patients with uncontrolled focal epilepsy1, were reviewed 6-8 years later.

Results: 153 patients took part in the Australian arm of the SKATE trial. Of these, complete long-term follow-up data were obtained for 41 patients. Reasons for nonparticipation included death (2), nonparticipation of the SKATE investigator (84), refusal to participate (5) and loss to follow-up (21). 29 (74.4%) of patients were still taking LEV (8 same, 8 lower and 13 higher dose). 5 of the 7 patients (71%) who were seizure-free during the 16 weeks of the SKATE trial remain seizure-free, while a further 7 patients have subsequently become seizure-free. 18 (67%) of the 27 patients who experienced a 50% or greater reduction in seizures in the SKATE trial maintained their ≥50% responder status, while a further 7 patients have subsequently become ≥50% responders.

Conclusion: The long-term retention rate on LEV was high. Most patients who responded favourably to LEV in a 16 week clinical trial maintained the improvement for more than 5 years. The results of this small study suggest that short term clinical trials predict long term response with reasonable accuracy.

Reference:

1. Somerville ER et al. Epilepsy Behav. 2007; 11:338-342

p196

Side effects and tolerability of IV levetiracetam vs. IV phenytoin and follow-on oral regimens in a neurosurgical patient population: a prospective randomised study

FULLER KL, WANG YY, COOK MJ, MURPHY MA, D'SOUZA WJ
Department of Clinical Neuroscience and Neurological Research, St Vincent's Hospital, Melbourne, Australia

Purpose: Intravenous (IV) levetiracetam (LEV) is an effective antiepileptic drug (AED) with potential to be particularly useful where IV phenytoin (PHT) is contraindicated or not tolerated. We aimed to compare the side effect profile including tolerability of IV LEV and IV PHT and follow-on oral regimens for seizure prophylaxis amongst a neurosurgical patient population.

Method: Study design was single-centre, prospective, block-randomised, and researcher-blinded. Participants received at least one standard dose of IV LEV or IV PHT peri-operatively, then the same AED in oral regimen. Side effect profile, medication continuation, and seizure number, were recorded two days post IV administration, at hospital discharge, and three months following surgery, and interval data compared between the AED regimens.

Results: 76 participants were randomised. IV administration to 2-days following (2-day to discharge, discharge to 90-day) datasets were complete for 36 (34, 25) LEV and 36 (35, 18) PHT participants. No side effects were reported by 81% of participants in the LEV group and 86% of participants in the PHT group from IV administration to 2-days following (p=0.38). No significant quantitative difference was found between the study groups for total side effects, major side effects, local side effects, or seizures, for any of the study time intervals (all p-values > 0.19). Less patients ceased LEV because of side effects (discharge to 90-day p=0.04; total IV plus oral regimen p=0.05).

POSTER ABSTRACTS

Conclusion: IV LEV and IV PHT are both well tolerated peri-operatively. LEV was better tolerated in total IV-plus-oral regimen.

p197

Lacosamide as adjunctive therapy with a broad range of AEDs: a pooled analysis of lacosamide clinical trial data by concomitant AED mechanism of action

DE BACKER M[1], ISOJARVI J[2], HEBERT D[2], DOTY P[2], ZACKHEIM J[3], DAVIES K[3], EGGERT-FORMELLA A3, SAKE J-K1

[1]UCB Pharma S.A., [B]russels, Belgium, [2]Schwarz Biosciences (a Member of the UCB Group), Raleigh, USA, [3]UCB, Inc., Smyrna, USA

Purpose: Lacosamide, an antiepileptic drug (AED), has a unique pharmacological action involving selective enhancement of sodium-channel slow inactivation. To evaluate efficacy and safety of lacosamide added to traditional sodium-channel blocking AEDs, post hoc exploratory analyses were performed on pooled Phase II/III trial data grouped by primary mechanism of action of patients' concomitant AEDs.
Method: Patients were grouped based upon inclusion or non-inclusion of ≥1 traditional sodium-channel blocking AED in the baseline AED regimen. Efficacy measures included ≥50% responder rates and change in seizure frequency. Treatment-emergent adverse events (TEAEs) and discontinuations were also evaluated.
Results: Of 1,308 patients, 82% were using ≥1 traditional sodium-channel blocking AED. In this subgroup, adjunctive lacosamide resulted in significantly higher percentages of ≥50% responders with 400mg/day (39.9%) and 600mg/day doses (42.4%) compared to placebo (22.7%; P< .01); 33.3% were ≥50% responders with 200mg/day lacosamide (P=.06). In the limited-size subgroup not taking sodium-channel blocking AEDs (18%), efficacy appeared more pronounced (41.9%, 62.3%, 79.2% for 200mg/day, 400mg/day, 600mg/day lacosamide vs 25.0% placebo), with significant differences for 400mg/day and 600mg/day, suggesting a potential signal for additional benefit with non-sodium or mixed-target AED combinations. Evaluation of median %seizure reduction showed similar results. Common TEAEs included dizziness, headache and nausea; incidences of TEAEs and discontinuations were lower in the non-sodium channel subgroup.
Conclusion: In this post hoc exploratory analysis, adjunctive lacosamide demonstrated significant seizure reduction over placebo regardless of concomitant AED primary mechanism of action. Further study is warranted to confirm whether certain combinations may produce additional benefit or improved tolerability.

p198

Screening for risk of osteoporosis in patients with refractory epilepsy attending epilepsy camp by the use of portable BMD machine study of 61 patients

NADKARNI V

Gita Bhawan Hospital, Indore, India

Purpose: Pharmacological Studies suggest that osteoporosis and osteopenia are potential side-effects of certain anti-epileptic drug (AED)in patients as a reduction in body's vitamin D. The purpose of this survey was to provide a bedside screening test for detection of osteoporosis in patients with chronic epilepsy by poratable ultrasound BMD machine In resource poor countries it is cost effective sreening test for large number of patients with epilepsy examined in epilepsy camp.
Method: Epilepsy camp conducted at community Hospital enrolled 61 patients with refractory epilepsy, Portable BMD Machine with ultrasound. Quantitative U.S measurement of calcaneum Rt heel was available allowing high frequency sound waves to pass through calcaneum for measuring BMD (high,average,low). Age group of patients varied from 11yrs to 78 yrs, includeded 34 male & 27 females. Antiepileptic drug monotherapy was noted in 13(n) and polytherapy in 48(n) patients.

POSTER ABSTRACTS

Results: BMD test indicated severe osteoporosis 11n(18%),Osteopenia in 29n(48% BMD was normal in 21n (34%)Study revealed 66 % patients were at risk of osteopenia & osteoporosis. Clinically all patients are asymptomatic except 3 patients had fractures. Patients on monotherapy (13n) had no osteoporosis while patient on polytherapy (32n) had abnormal BMD with osteoporosis three patients (16%) developed severe osteoporosis with fractures.
Conclusion: In epilepsy camp where resource poor patients participatie of patients had osteopenia and or osteoporosis Ultrsound BMD test measurement is costeffective in such setting Monotherapy in long term antiepileptic drugcarries lesser risk of osteoporosis than patients taking polytherapy. Preventive measures like vitamin D/or Calcium supplement medicines & diet rich in calcium is advisable in these patients.

p199
Response to lamotrigine is associated with genetic variants of ABCB1 and SCN1A
KIM SE[1], HWANG TG[2], SHIN JG[3]
[1]Department of Neurology, Haeundae Paik Hospital, Inje University, Busan, Korea, Republic of, [2]Department of Pediatrics, Haeundae Paik Hospital, Inje University, Busan, Korea, Republic of, [3]Department of Clinical Pharmacology, Haeundae Paik Hospital, Inje University, Busan, Korea, Republic of

Purpose: We explored the contribution of genetic variations to lamotrigine resistance using a multigenetic approach involving the pharmacokinetic (ABCB1, UGT1A4) and pharmacodynamic (SCN1A) lamotrigine genes.
Method: ABCB1 2677G>A/T and 3435C>T, UGT1A4 142T>G and 292C>T, and a comprehensive set of tagged single nucleotide polymorphisms across SCN1A were genotyped in 253 patients with epilepsy taking lamotrigine. Lamotrigine resistance was defined as the occurrence of any unprovoked seizure for 1 year in those taking at least three antiepileptic drugs including a maximally tolerated dose of lamotrigine. Demographic, clinical, and genetic factors for the risk of lamotrigine resistance were estimated with odds ratios (ORs) and 95% confidence intervals (95% CIs) using unconditional logistic regression models.
Results: Seventy-three patients were lamotrigine-resistant and 180 were lamotrigine-controlled. The clinical factors significantly associated with lamotrigine resistance were age at onset < 30 years, seizure frequency before treatment >6, and etiology. The patients carrying the ABCB1 haplotype containing either H3 or H4 had more than a twofold risk of lamotrigine resistance (OR=2.3, 95% CI=1.2-4.5). The SCN1A B2H5 haplotype also had a borderline significantly increased risk (OR=2.2, 95% CI=0.9-5.2). Furthermore, an epistatic interaction was observed; as the number of ABCB1 or SCN1A risk alleles increased, the risk of lamotrigine resistance increased significantly (OR=4.5, 95% CI=1.0-19.4 for patients carrying at least two risk alleles vs. no risk alleles (p for trend < 0.01).
Conclusion: These findings suggest that the genetic variations involving both lamotrigine metabolism and its target are associated with lamotrigine resistance.

p200
The effect of topiramate monotherapy on bone mineral density and markers of bone and mineral metabolism in premenopausal women with epilepsy
HEO K[1], RHEE Y[2], LEE BI[1], LEE HW[3], LEE SA[4], SHIN DJ[5], KIM W-J[6], SONG H-K[7], SONG K[8]
[1]Department of Neurology, Yonsei University College of Medicine, Seoul, Korea, Republic of, [2]Department of Internal Medicine, Yonsei University College of Medicine, Seoul, Korea, Republic of, [3]Department of Neurology, Ewha Womans University Mokdong Hospital, Seoul, Korea, Republic of, [4]Department of Neurology, Asan Medical Center, Seoul, Korea, Republic of, [5]Department of Neurology, Gachon Medical School Gil Medical Center, Incheon, Korea, Republic of, [6]Department of Neurology, Gangnam Severance Hospital, Seoul, Korea, Republic of, [7]Department of Neurology, Kangdong Sacred Heart Hospital, Seoul, Korea, Republic of, [8]Department of Biostatistics, Yonsei University College of Medicine, Seoul, Korea, Republic of

POSTER ABSTRACTS

Purpose: To assess the relationship of topiramate (TPM) to bone mass and metabolism in 36 young women with epilepsy on long-term (at least 1 year) TPM monotherapy compared with 36 patients taking carbamazepine (CBZ), 32 patients taking valproic acid (VPA), and age-matched 36 healthy control subjects.
Method: Subjects completed exercise and nutrition questionnaires and bone mineral density (BMD) studies. Serum was analyzed for indices of bone metabolism.
Results: BMD Z-scores and serum vitamin D (25-hydroxyvitamin D and 1alpha,25-dihydroxyvitamin D_3) concentrations did not differ among the groups. Serum calcium concentrations were significantly less in patients receiving TPM than in those receiving VPA, and in patients receiving CBZ than in those receiving VPA, and control subjects. Patients receiving TPM had less levels of parathyroid hormone (PTH) compared with the other groups. Patients receiving TPM or CBZ had greater levels of bone-specific alkaline phosphatase compared with control subjects. Patients receiving TPM had greater levels of osteocalcin compared with those receiving CBZ and control subjects. C-terminal telopeptides of type I collagen was increased in patients receiving TPM compared with in those receiving CBZ or VPA.
Conclusion: Our results demonstrate that TPM is associated with increased bone turnover. The lower calcium concentrations in patients taking CBZ or TPM compared with those taking VPA, or control subjects suggest that these antiepileptic drugs may have long-term effects. TPM may lead to decreased levels of PTH (and calcium) via effects on protein kinase A and protein kinase C signaling which might affect the calcium-sensing receptor.

p201
Lack of association of lamotrigine-induced cutaneous adverse reactions with HLA-B*1502 in a Han Chinese population
AN D[1], WU X[1], HU F[1], YAN B[1], STEFAN H[2], ZHOU D[1], AEDS GENETICS GROUP
[1]Department of Neurology, West China Hospital, Sichuan University, Chengdu, China, [2]Epilepsy Center, Department of Neurology, University Hospital of Erlangen-Nuernberg, Erlangen, Germany

Purpose: Antiepileptic drugs including lamotrigine (LTG) and carbamazepine (CBZ) are among the most common causes of cutaneous adverse reactions (cADRs). Human leukocyte antigen (HLA)-B*1502 has been strongly associated with CBZ-induced Stevens-Johnson syndrome/toxic epidermal necrolysis (SJS/TEN). This study aimed to investigate the association of LTG-induced cADRs with the HLA-B*1502 allele in a Han Chinese population.
Method: We performed high-resolution HLA genotyping on LTG-tolerant controls, healthy volunteers, and patients affected by LTG-induced cADRs, ranging from maculopapular exanthema (MPE) to SJS/TEN.
Results: Patients with LTG-induced cADRs (n=25, including three with SJS/TEN and 22 with MPE), 21 LTG-tolerant controls, and 71 healthy volunteers were enrolled. The differences in the starting dosage of LTG among the SJS/TEN, MPE, and LTG-tolerant control groups were not statistically significant. HLA-B*1502 frequency was 33.3% (1/3; LTG-induced SJS/TEN group), 9.1% (2/22; LTG-induced MPE group), 4.8% (1/21; LTG-tolerant group), and 8.5% (6/71; healthy volunteers). There was no significant difference in the frequency of subjects with the HLA-B*1502 allele between the SJS/TEN group and LTG-tolerant group (p=0.239, OR=10.0, 95% CI 0.44-228.7), and healthy volunteers (p=0.26, OR=5.42, 95% CI 0.43-68.8), MPE and LTG-tolerant groups (p=1.0, OR=1.08, 95% CI 0.20-5.8), and healthy volunteers (p=1.0, OR=2.0, 95% CI 0.17-23.9). None of the HLA alleles detected were associated with LTG-induced cADRs.
Conclusion: HLA-B*1502 and other HLA alleles are not directly associated with LTG-induced SJS/TEN or LTG-induced MPE. Other HLA biomarkers may be responsible for LTG-induced SJS/TEN.

POSTER ABSTRACTS

p202

Prospective HLA-B*1502 genotyping in preventing carbamazepine-induced Stevens-Johnson syndrome: one medical center experience

HUNG K·L[1,2], LIAO H·T[1], CHIANG T·R[1,2], CHEN Y·T[3]
[1]Cathay General Hospital, Taipei, Taiwan, Republic of China, [2]Fu-Jen Catholic University, Sinjhuang, Taiwan, Republic of China, [3]Institute of Biomedical Sciences, Academia Sinica, Taipei, Taiwan, Republic of China

Purpose: Adverse drug reactions (ADRs) are major clinical problems for patients receiving antiepileptic drug (AED) therapy. The recent discovery of HLA-B*1502 responsible for the development of carbamazepine (CBZ)-induced Stevens-Johnson syndrome (SJS) and toxic epidermal necrolysis (TEN) has made possible the prevention of severe ADR through the pharmacogenomic survey. This study was designed to screen the sensitive gene before the administration of CBZ to prevent the occurrence of severe ADRs.
Method: A multi-centered, prospective study of HLA-B*1502 genotyping in preventing CBZ-induced SJS was conducted in Taiwan during the period from January 2008 to March 2010. Here we present the preliminary data in one medical center in Taipei.
Results: Totally 25 patients, males 13 and females 12, were collected. Their ages ranged from 2 to 17 (mean: 8.2). The seizure types were partial in 23 patients and generalized in 2. Among the 25 patients, 4 were HLA-B*1502 positive and their therapeutic regimen were successfully shifted to other AED before the intake of CBZ. All the 25 patients did not develop the severe SJS/TEN reactions. Only mild sleepiness and gastrointestinal upset were noted in 2 cases, respectively. One additional patient suffering from of SJS was found in this period, who turned out to be B*1502 positive without genotype screening beforehand and prescribed from outside hospital.
Conclusion: The strategy of prospective HLA-B*1502 genotyping to prevent CBZ-induced severe cutaneous ADRs seems to be efficient and cost effective. This study demonstrates the importance of genomic screening in prevention of severe CBZ-induced ADRs for particular population.

p203

The effect of levetiracetam monotherapy on subjective sleep quality and objective sleep parameters in patients with epilepsy: compared with the effect of carbamazepine-CR monotherapy

CHO YW[1], YI SD[1], KIM DH[1], MOTAMEDI GK[2]
[1]Department of Neurology, Keimyung University Dongsan Medical Center, Daegu, Korea, Republic of, [2]Department of Neurology, Georgetown University Hospital, Washington, DC, USA

Purpose: This study was done to evaluate the effect of levetiracetam on subjective sleep quality and sleep architecture in patients with epilepsy, and the results were compared with the effect of carbamazepine-CR.
Method: The study was of a case control design with random allocation of two different treatments, levetiracetam (1000mg/day) or carbamazepine CR (400mg/day). All subjects were partial epilepsy patients and were tested with an overnight polysomnography (PSG) with full electrodes. Sleep questionnaires and the National Hospital Seizure severity Scale (NHS3) also were evaluated. PSG and the questionnaires were repeated after 4 -6 weeks of treatment. Differences between baseline and treatment in the levetiracetam and carbamazepine were compared using the General Linear Model.
Results: Thirty-one completed the study (16 levetiracetam, 31 ±15.31 years and 15 carbamazepine CR, 29±9.31 years). When the baseline PSG was compared with the treatment PSG, there was a significant increase in sleep efficiency and decrease in the number of awakenings after sleep onset in the levetiracetam group. After treatment with each medication, the PSG findings in the levetiracetam and carbamazepine groups revealed similar results, there was no significant difference between the two.

POSTER ABSTRACTS

Conclusion: These results suggest that levetiracetam does not have major effects on sleep structure. However, levetiracetam gives rise to stabilization of sleep, including improvement in sleep continuity, a decrease in sleep fragmentation, the REM latency and the percentage of REM sleep were unchanged, though there were no changes in subjective sleep parameters.

p204
UGT2B7 polymorphisms on carbamazeine pharmacokinetics in epileptic patients developing adverse drug reactions and toxicity
VIVEKANANDHAN S[1], MURALI M[1], TRIPATI M[1], BEHARI M[1], JAIN DC[2]
[1]All India Institute of Medical Sciences, New Dehli, India, [2]Safdarjung Hospital, New Delhi, India

Purpose: Carbamazepine, a widely used anti-epileptic drug, exhibits marked inter-individual variation in Pharmacokinetics and attributed to genetic factors such as UGT2B7 polymorphisms. The present study was undertaken to investigate the influence of UGT2B7 variant genotypes on Carbamazepine Pharmacokinetics in the epileptic patients showing toxicity.
Methods: 30 epileptic individuals who had developed toxicity to carbamazepine and 30 control epileptic subjects who had not developed ADR and toxicity to carbamazepine were genotyped for UGT2B7 polymorphisms by Polymerase chain reaction-restriction fragment length polymorphisms (PCR-RFLP Method). Allele frequencies were derived from genotypic data. Case-control comparisons were made using Chi-square tests. Deviations from the Hardy-Weinberg equilibrium were also tested. Carbamazepine plasma levels were analyzed by reverse phase HPLC method and Pharmacokinetic parameters such as area under the concentration curve (AUC), maximum concentration (C max), time to C max (t max), drug clearance and half-life (t1/2) were estimated by non compartmental analysis using PK solutions software.
Results: The UGT2B7 polymorphisms was seen to be in Hardy-Weinberg equilibrium and showed significant allelic association but did not show significant genotypic association (P = 0.301; OR =0.51 (0.16-1.61).The variant allele showed significant association with the carbamazepine toxicity phenotype (P = 0.01; OR =2.35 (1.19-4.67).
The pharmacokinetics parameters in heterozygosity group showed longer half life (t1/2 =17 hrs) and less clearance rate (CL = 1.5 L/hr) when compared to wild type group (t ½ = 12.8 hrs, CL = 2.9 L/hr).
Conclusion: Our findings suggest that the UGT2B7 genetic polymorphisms may be associated with the individual difference in carbamazepine pharmacokinetics. An individualized dosage regimen design incorporating such genetic information would help to increase the clinical efficacy of carbamazepine and reducing the toxicity in epileptic patients.

Clinical neurophysiology

p205
Cortical hyper-excitability in migraine and epilepsy: is this the link?
BADAWY RAB[1,2,3], JACKSON GD[1,2,3]
[1]Brain Research Institute, Heidelberg West, Australia, [2]Department of Neurology, Austin Health, Heidelberg, Australia, [3]Department of Medicine, The University of Melbourne, Melbourne, Australia

Purpose: There is strong evidence for the co-morbidity between migraine and epilepsy. Previous studies have shown increased cortical excitability in epilepsy compared to controls. We used transcranial magnetic stimulation (TMS) to assess cortical excitability in subjects with migraine compared to controls and epilepsy.

POSTER ABSTRACTS

Methods: 26 patients with recent onset migraine (14 with aura) from a surveyed population of 404 community recruited subjects were studied while drug naïve. These were compared to 19 age and sex matched healthy controls and 50 age and sex matched previously studied drug naïve patients with newly diagnosed epilepsy (22 focal epilepsy and 28 idiopathic generalized epilepsy [IGE]). Motor threshold (MT) at rest and responses to paired pulse stimulation at a number of short (2,5, 10 and 15 ms) and long (50 · 400 ms) interstimulus intervals (ISI) were measured.
Results: When patient groups were compared to controls, cortical excitability was increased in migraine only at the long ISI 250 ms ($p < 0.05$), while in epilepsy it was increased at the short ISIs 2 and 5 ms ($p < 0.05$) and the long ISIs 250 and 300 ms ($p< 0.01$). The effect at the 250 ms ISI ($p < 0.05$) was significantly greater in epilepsy when compared to migraine.
Conclusion: Cortical excitability increases in migraine at the 250 ms ISI suggesting involvement of the GABAB system. The cortical hyper-excitability we show for migraine may help explain the co-morbidity observed between migraine and epilepsy.

p206
Suppressive effect of low-frequency electric cortical stimulation on the seizure onset zone relative to the cortico-cortical connectivity
KINOSHITA M[1,2], MATSUMOTO R[2], MATSUHASHI M[3], MITSUEDA-ONO T[2], HITOMI T[2], TAKI J[4], MIKUNI N[4], FUKUYAMA H[3], TAKAHASHI R[2], IKEDA A[2]
[1]Department of Neurology, Utano National Hospital, Kyoto, Japan, [2]Department of Neurology, Graduate School of Medicine, Kyoto University, Kyoto, Japan, [3]Human Brain Research Center, Graduate School of Medicine, Kyoto University, Kyoto, Japan, [4]Department of Neurosergery, Graduate School of Medicine, Kyoto University, Kyoto, Japan

Purpose: We previously reported the suppressive effect of low-frequency electric cortical stimulation on the seizure onset zone by comparing spike frequency relative to the cortico-cortical connectivity. We further analyzed the change in electrocorticogram (ECoG) power spectra.
Method: A 26-year-old patient with intractable partial epilepsy who underwent invasive presurgical evaluations and had a pathological diagnosis of FCD type IIa was analyzed. His seizures started with left arm convulsion followed by asymmetric tonic posturing and secondarily generalization. By ictal recording, the seizure onset zone resided mainly in right parietal lobe. Low-frequency electric cortical stimulation was delivered to a pair of electrodes in seizure onset zone after obtaining written informed consent based on the approval of the institutional ethical committee (#235). We compared spike frequency and ECoG power spectra 5 min before and after stimulation in all recorded electrodes. Cortico-cortical evoked potentials (CCEPs) were obtained by averaging ECoG time-locked to low-frequency stimuli.
Results: In areas where clear negative deflection of CCEPs was elicited, spike frequency decreased by 18% and power spectra significantly increased in 10-12 and 80-100 Hz bands, whereas in areas without CCEPs elicitation, spike frequency decreased by 41% and power spectra significantly decreased in 12-30 and 40-50 Hz bands.
Conclusion: In this subject, the suppressive effect on epileptogenicity by low-frequency electric cortical stimulation was stronger in areas without elicitation of CCEPs.

p207
Effects of chronic epilepsy on heart rate variability upon the seizure frequency
HARNOD T
Buddhist Tzu Chi General Hospital and Tzu Chi University, Hualien, Taiwan, Republic of China

POSTER ABSTRACTS

Purpose: Dysfunction of autonomic cardiac regulation which representing by the heart rate variability (HRV) has been thought to be related to higher mortality in epilepsy patients. The correlation between disease severity and change of HRV still remains unclear. We tried to study changes of sympathetic and parasympathetic activities in patients with chronic epilepsy and having different seizure frequencies.
Method: The study included 99 patients with primary epilepsy (44 males and 55 females). By their seizure frequencies, 41 patients had less than one seizure per month (group A), 30 patients had seizure frequency of between 1 and 10 per month (group B), and 28 patients had more than 10 seizures per month (group C). Lead I electrocardiograms were taken in 5 minutes during an interictal period in the daytime among these patients. Digital data was converted to frequency-domain analysis of heart rate variability with fast Fourier transformation. Heart rate R-R interval (RR), high frequency power (HF; 0.15-0.45 Hz, represent parasympathetic regulation), low frequency power (LF; 0.04-0.15 Hz, from mixed sympathetic and parasympathetic regulation), and LF/(HF+LF) expressed in normalized units (LF%, represent sympathetic regulation) were analyzed and interpreted. The differences of variables between groups were examined using One-way ANOVA.
Results: The RR had a significant difference between the group B and group C and a borderline significant difference between the group A and group B. There were no between-group differences in LF, LF% and HF.
Conclusion: Epilepsy patients with different seizure frequencies do not have different changes in the HRV.

p208
Surgery of focal and multifocal temporal epilepsy and neurophysiology of early and late epileptogenesis
BERSNEV V, STEPANOVA T, KASUMOV V, KASUMOV R, KRAVTSOVA S, SEBELEV K
Russian A.L. Polenov Neurosurgical Institute, St. Petersburg, Russian Federation

Purpose: Optimization surgical treatment of drug-resistant temporal epilepsy basing on neurophysiologic criteria of epileptogenesis.
Method: Electroclinical examination and surgical treatment over 300 drug-resistant patients aged 18-53 suffering temporal epilepsy. According to ILAE recommendations monitoring of EEG with topographic brain mapping, ECoG, ESCG, SEEG via deep electrodes was used.
Results: Based on focal EEG-SEEG trait-markers peculiarities of initial (pre-clinical), temporal (early) and extratemporal (late) epileptogenesis were depicted clinical-neurophysiologic forms of focal and multifocal temporal epilepsy on different stages of the disease depending on pathways of epileptization. It was shown that temporal epileptogenesis is peculiar for localization of temporal epileptic focus involving limbic structures (amygdala, hippocampus) and temporal cortex with forming monotemporal and "intermediate" (with initial mirror focus) epilepsy. According to ECoG-ESCG-monitoring of the Institute personal material the majority of patients demonstrated a vast epileptic zone with combined damage of temporal neocortex and limbic structures; isolated foci in temporal neocortex (4%) or limbic formations (17%) were rare finding. This optimizes technology of open surgical treatment (anterior temporal lobectomy) in cases requiring additional use of multiple subpial transection in eloquent cortex. Limbical-brain-stem pathways of epileptogenesis were followed-up: hippocampus and amygdala via thalamic nuclei are involved in cyclic Papez and Livingston-Escobar systems, forming multifocal forms, which is of importance in neuromodulating interventions.
Conclusion: Neurophysiologic indicators of epileptogenesis demonstrate that temporal epilepsy is an intricate form having personal electroclinical topical-diagnostic variants, important of surgical treatment. For monotemporal forms open intervention under ECoG-ESCG-control is optimal (78-80% positive effect). Neuromodulating stereotaxic operations are less effective (55-60%).

POSTER ABSTRACTS

p209

The effect of hyperventilation on enhancing epileptic discharge using a simultaneous measurement by electroencephalography and near infra-red spectroscopy

NISHIBAYASHI H, ITAKURA T
Department of Neurological Surgery, School of Medicine, Wakayama Medical University, Wakayama, Japan

Purpose: To investigate the effect of self-paced hyperventilation(HV) on activating paroxysmal discharges using a simultaneous measurement by electroencephalography(EEG) and near infra-red spectroscopy(NIRS) in epileptic patients.
Method: We investigated 12 patients with partial epilepsy after obtaining informed consents. Scalp EEG electrodes were placed at several regions in which epileptic discharges were frequently recorded. A multi-channel NIRS system was applied covering bilateral fronto-temporal areas. A simultaneous EEG-NIRS recording was performed in each patient during pre-HV period (5 minutes), HV period (4 minutes), and post-HV period (6 minutes) in supine position with eyes closed. Frequency of epileptic discharges and fluctuation of oxy-hemoglobin (oxy-Hb), deoxy-hemoglobin (deoxy-Hb) were analyzed.
Results: Oxy-Hb from the baseline decreased at mean 78 seconds after the start of HV until mean 227 seconds after the end of HV, then gradually increased. Deoxy-Hb fluctuated reversely to oxy-Hb. Frequency of epileptic discharges increased after inducing HV in five patients, and also after the end of HV in three of them. All the five patients had intractable epilepsy. Two of them with intractable frontal lobe epilepsy revealed habitual seizures after HV.
Conclusion: Epileptic discharges were activated during HV and post-HV period in intractable epileptic patients. Widespread decrease of oxy-Hb in the periods may be related to the activation of epileptic discharges.

p210

Localization and extent of the intracranial irritative zone in patients who have no detectable interictal epileptiform activity on scalp EEG: a case-control study

UNNWONGSE K[1,2], WEHNER T[3], ALEXOPOULOS A[1]
[1]Cleveland Clinic Epilepsy Center, Cleveland, Ohio, USA, [2]Prasat Neurological Institute, Bangkok, Thailand, [3]Phillips-University Marburg, Marburg, Germany

Purpose: Factors determining the appearance of IEDs on scalp EEG are location, extent, and orientation of the cortical sources. In this intracranial EEG (ICEEG) study, we determine the localization and extent of the irritative zone, the area generating IEDs in patients with scalp invisible IEDs compared to patients with scalp detectable IEDs.
Method: The records of 426 patients who underwent ICEEG for epilepsy surgery from 1997-2008 were reviewed. Of these, 36 patients never had any IEDs detected on scalp EEG (cases). Controls were 36 age-matched patients, who had scalp detectable IEDs. All populations of ICEEG recorded IEDs were reviewed. The extent of the irritative zone was estimated by the most restricted field (number of electrodes involved) for each documented IEDs population. The location of the irritative zone was identified on co-registered positions of electrodes on the MRI using Talairach sublobar classification.
Results: Among groups no difference in terms of epilepsy syndromes, MRI findings, pathology results, and surgery outcomes. The number of IED populations per patient was significantly lower in cases compared to controls (p= 0.027). The irritative zone was similarly identified in the lateral convexity, mesial aspect, basal surface, and insula/deep sulci in both groups (p=0.855). Subdural grid recorded IEDs involved significantly fewer electrodes in cases compared to controls (p=0.004).
Conclusion: In this study, the extent of the irritative zone appeared to be more important than location in determining the presence or absence of IEDs on scalp EEG. Patients with scalp negative IEDs had fewer ICEEG recorded IED populations.

POSTER ABSTRACTS

p211
Gamma EEG in epilepsy and other CNS disorders
WHITHAM E, POPE KJ, LEWIS TW, FITZGIBBON SP, WILLOUGHBY JO
Flinders University, Adelaide, Australia

Purpose: High frequency (above 25Hz) gamma EEG has been linked to epilepsy in animal models and human studies, possibly reflecting enhanced activity in neural circuits. Fast EEG rhythms (above 20Hz) in scalp recordings are usually confounded by electromyographic (EMG) activity, however, recordings from the central scalp electrodes are least likely to be contaminated, especially below 35Hz. Therefore, using central electrodes and a 35Hz limit, we re-evaluated whether increases in gamma EEG are associated with epilepsies and whether they are specific to epilepsy.
Method: Scalp electrical activity was recorded via 128 electrodes with 2KHz sampling frequency using a Neuroscan system. Controls and subjects with epilepsy and other neuro-psychiatric disorders were studied at rest with eyes-closed. Fourier analysis was used to generate power spectra, which were compared with spectra from control groups pair-matched for age, gender and intelligence. EEG power to 25Hz was calculated for all subjects, while heavily EMG-edited records were used above 25Hz. Power is expressed as Mean+/-SD E-14 Volts-squared.
Results: Significant increases in EEG power were detected in localization-related (LRE) and primary generalized epilepsies (PGE) in various frequency bands. Increases in 25-35Hz power were also evident in PGE (7.4+/-8.0 vs 3.8+/-2.1, p< 0.013, n=26), in LRE (5.7+/-5.2 vs 4.0+/-2.8, p< 0.046, n=35) and in bipolar disorder (7.1+/-4.2 vs 3.2+/-1.9, p< 0.024, n=6).
Conclusion: Increased gamma EEG power is a feature of PGE and LRE, but is not exclusive to epilepsy. Bipolar disorder may have a common patho-physiology with epilepsy.
Supported by The Wellcome Trust, NHMRC, Pfizer, and Flinders Foundation

p212
Frequency analysis of ictal neuromagnetic signals in presurgical evaluation of patients with intractable epilepsy
IIDA K[1], HASHIZUME A[1], KAGAWA K[1], NISHIMOTO T[1], KIURA Y[1], HANAYA R[2], ARITA K[2], KURISU K[1]
[1]Department of Neurosurgery, Hiroshima University, Hiroshima, Japan, [2]Department of Neurosurgery, Kagoshima University, Kagoshima, Japan

Purpose: Intracranial EEG shows ictal high-frequency oscillations (HFOs) in the epileptogenic zone. We analyzed the frequency of ictal magnetoencephalographic (MEG) signals to detect HFOs in patients with intractable epilepsy.
Methods: Twelve patients underwent MEG studies using the Neuromag system (sampling rate: 600 Hz), where 204 channels of planar gradiometers were incorporated. We charted the time-frequency map (frequency-band: 0-110 Hz) of each MEG-sensor and superimposed all maps onto individual brain-surface MR images (deployed using Mercator projection) to visualize dynamic changes and distributions of HFO. We compared MEG-derived HFO distributions to those with equivalent current dipole (ECD) and intracranial EEG (sampling rate: 400Hz).
Results: Clinical/subclinical seizures were recorded during MEG in 2 patients who had single-clustered ECDs. Patient #1 indicated complex partial seizures with low-amplitude fast MEG activities and simultaneous EEG in the temporal region during motionless episode in early-stage seizure. Patient #2 with simple partial and secondary generalized seizures showed rhythmic discharges without clinical symptoms in the frontal region during MEG-/EEG-monitoring. Ictal HFOs with wide-range frequency (>100 Hz) were detected and the distributions partially overlapped with clustered ECDs and the ictal onset zone on intracranial EEG in both cases. MEG-derived HFO distributions were concordant to intracranial EEG HFO distributions in both cases. Patients #1 (pathology: gliosis) and #2 (pathology: cortical dysplasia) became seizure-free after cortical resection, including the clustered ECDs and ictal HFO area.

POSTER ABSTRACTS

Conclusion: The distribution of ictal HFOs on MEG was correlated with the epileptogenic zone, and may contribute to zone-localization in a fashion equivalent to intracranial EEG.

p213
The signal detection ability of the scalp dense array electroencephalogram: spike comparison with the simultaneous subdural electrodes
FUJIMOTO A, YAMAZOE T, ENOKI H, OKANISHI T, YOKOTA T, TOYODA G, YAMAZAKI M, YAMAMOTO T
Comprehensive Epilepsy Center, Seirei Hamamatsu General Hospital, Hamamatsu, Japan

Purpose: To evaluate the spike detecting ability of the dense array electroencephalogram (dEEG).
Method: A 21 years old right handed female patient with right neocortical temporal lobe epilepsy underwent subdural electrodes (SD) insertion, the inter-contacts distances were 1cm, followed by the focus resection over the anterior part of the right superior, middle and inferior temporal lobe. This patient has been seizure free after the surgery. During the subdural electrodes monitoring (SDM) we simultaneously monitored dEEG.We compared the thirty SDM spikes and the same spikes on the dEEG.
Results: 83% of the SDM spikes were detected by the dEEG. The SDM spikes were divided into A. less than 2 contacts, 3 contacts, 4 contacts, 5 contacts, 6contacts, 7contacts, 8contacts, 9contacts and more than 10 contacts.55% of the less than 2 contacts of SDM were detected by dEEG. Most of them were from mesial temporal spikes.
Conclusion: The dEEG could detect most of the spikes with the order of 2cm. The spike detection rate of 83% is high. However the spikes from mesial structure could not be detected by the dEEG.

p214
Clinical, neuroimaging and electrophysiological characteristics of patients with refractory eating epilepsy (EE)
CHERIAN A, BAHETI NN, MENON RN, RADHAKRISHNAN A, RADHAKRISHNAN K
Sree Chitra Tirunal Institute for Medical Sciences and Technology, Trivandrum, India

Purpose: To evaluate the clinical, EEG and imaging attributes of patients with EE who underwent video-EEG monitoring for refractory seizures.
Method: Consecutive patients with refractory EE subjected to video-EEG from 2003-2009 were studied. A patient was diagnosed as EE if >75% of the seizures occurred while eating. It was sub-classified as pre-prandial (occurring within five minutes before eating), prandial (any time during eating) and post-prandial (within ten minutes of cessation of eating).
Results: Of the 2652 patients who underwent Video-EEG for refractory epilepsy during this period, 25 (0.9%, M:F 22:3) satisfied the criteria for EE. Mean age of onset was 22.6 ± 9.7 years. Mean follow-up was 24.6 months. EE was prandial &/or pre-prandial in 24 and post-prandial in one. Three also had seizures triggered by gargling and brushing. Eleven patients (42%) had MRI lesions, commonest in the posterior head region in seven (gliosis-4; pachgyria-1; peritrigonal hyperintensities-2). Single CPS was most common (75%) while multiple head drops occurred in five. Temporal (n=6), extratemporal (n=4) and multifocal interictal discharges (n=15) were noted. Ictal onset was diffuse in 10, localized in 8 and lateralized in 7 patients. Eating blind-folded, assisted feeding and reducing bulk of meals ameliorated EE in three. Two with temporal lobe epilepsy underwent surgery, are seizure-free at last follow-up. Of remaining, 5 remained seizure-free and 8 other had >50% seizure reduction on drugs.
Conclusion: Formulating a strict definition and characterization of events by Video-EEG is essential for proper management of this rare reflex epilepsy syndrome which can otherwise impair the quality of life.

POSTER ABSTRACTS

p215

Changes of cortical excitability after epileptic seizure and non-epileptic seizure

SHIN WC[1], CHO YW[2], SUNG MJ[1], RHEE HY[1]

[1]Department of Neurology, East West Neo Medical Center, College of Medicine, Kyung Hee University, Seoul, Korea, Republic of, [2]Department of Neurology, Dongsan Medical Center, Keimyung University, Daegu, Korea, Republic of

Purpose: The transcranial magnetic stimulation (TMS) provides a well-established noninvasive means of investigating human motor cortex excitability and may serve as a marker of excitatory and inhibitory imbalance in the cortical neuron of epilepsy patients because of is sensitively detects relatively subtle alterations in the physiological state of the brain. This study performed to know the difference of cortical excitability in epileptic seizure and non-epileptic seizure with loss of consciousness by using TMS.
Method: 10 patients with drug-naïve epileptic seizure and 8 patients with non-epileptic seizure and 10 healthy normal control were enrolled. We examined the TMS within 24 hours of in loss of consciousness due to epileptic seizure and non-epileptic seizure. After 7 day of events, we measured again TMS parameters in patients with epileptic and non-epileptic seizure.
Results: Motor evoked potential amplitude, Cortical silent periods in patients with epileptic seizure decreased more significantly than in patients with non-epileptic seizure. After 7 day of events, the decreased parameters in epileptic seizure normalized and the parameter in non-epileptic seizure unchanged.
Conclusion: Our study showed that epileptic seizure transiently decreases cortical excitability. Also, these finding suggested the TMS study within 24 hour of loss of consciousness could be useful tool for differentiation between the epileptic seizure and non-epileptic seizure.

EEG

p216

Simultaneous recording of electroencephalography and electromyography enhances the diagnosis of paroxysmal events during video-electroencephalography monitoring

HILL A, BRIGGS B, SENEVIRATNE U

The Alfred Hospital, Melbourne, Australia

Purpose: To investigate the usefulness of adjunctive electromyographic (EMG) polygraphy in the diagnosis of clinical events captured during video-electroencephalography monitoring (VEM).
Method: A total of 40 patients (21 female, 19 male) aged between 19 and 72 years (mean 43) investigated using VEM between August 2009 and May 2010 were studied. EMG activity was simultaneously recorded with EEG in 4 patients selected on clinical grounds. Surface EMG electrodes were placed over muscles suspected to be activated during a typical clinical event.
Results: The mean duration of monitoring was 4.7 days. Of the 40 patients studied, 24 (60%) were given a diagnosis (epileptic seizures 14, psychogenic nonepileptic seizures 7, other paroxysmal events 3), whilst 16 remained undiagnosed. All four patients who had adjunctive EMG polygraphy received a diagnosis, with 3 of these diagnoses being exclusively reliant on the EMG findings. One patient was diagnosed with axial myoclonus with EMG traces showing an order of muscle activation starting at the biceps spreading caudally. Another patient was diagnosed with facio-mandibular myoclonus, detected by EMG electrodes placed over the obicularis oculi and masseter muscles. The third patient was found to have bruxism and periodic limb movements of sleep. Those four patients had been referred with a suspected diagnosis of epilepsy.
Conclusion: The information obtained from surface EMG recordings aided the diagnosis of clinical events captured during VEM in 7.5% of the total cohort. This study suggests that EEG-EMG polygraphy may be used as a technique of improving the diagnostic yield of VEM in selected cases.

POSTER ABSTRACTS

p217
EEG characteristics and surgical treatment of epileptic spasms
ZHOU W
Yuquan Hospital, Tsinghua University, Beijing, China

Purpose: To investigate the effect of EEG in the presurgical evaluation of patients with epileptic spasms, we analyzed the relationship between surgical outcomes and clinical characteristics of their EEG, especially the ictal scalp EEG and intracranial electrocorticography (ECoG).
Method: We studied a consecutive series of 11 patients with clusters of epileptic spasm accompanied by partial seizure, who had received surgical treatment in our department from July 2007 to June 2009. The detailed history, physical examination and MRI, prolonged VEEG of all patients were collected and assessed. To confirm the epileptic origins, 4 patients received intracranial prolonged ECoG monitoring and the others were monitored for 30~60minutes during the operation.
Results: All patients had been followed up for 10-33 months after neurosurgery. Nine patients were classified as Engel I, one Engel II, one Engel IV. Among the 4 patients who received intracranial prolonged ECoG monitoring thirty-one seizures were monitored. "leading" local low-amplitude fast waves, overlapped quickly by positive slow waves were observed in 26 seizures in 2 patients. "Leading" local high-amplitude spike-slow waves, followed by low-amplitude fast waves was observed in five seizures in another 2 patients, originating from the cortex surrounding sensory-motor gyrus.
Conclusion: In nine of the 11 patients we found local disturbance in the MRI, consisting with the origins of abnormal discharges. In four patients, receiving intracranial EEG monitoring, we found local fast waves at the beginning of the epileptic spasms. In conclusion, presurgical evaluation would benefit greatly from EcoG, although more cases should be demanded in further study.

p218
BESA epilepsy software: fast clinical evaluation of interictal spikes in long-term EEG
SCHERG M[1], ILLE N[1], WECKESSER D[1], EBERT A[1], OSTENDORF A[1], LARSSON PG[2], HENNING O[2], BOPPEL T[3], BAST T[4]
[1]MEGIS Software GmbH, Graefelfing, Germany, [2]University Hospital, Epilepsy Center, Oslo, Norway, [3]University Hospital, Neuroradiology, Heidelberg, Germany, [4]Epilepsiezentrum, Kehl-Kork, Germany

Purpose: BESA Epilepsy uses a new, fast hyperclustering technique to combine similar spike events over 24 hours of EEG. Each day, the physician inspects the hyperclusters and decides whether they are epileptiform or not. Optimized EEG displays, 3D maps, and localization in a head scheme allow for fast decision and assessment of the region of origin.
Method: Using a new spike detection and clustering algorithm based on an EEG transformation into 29 regional brain sources, clusters were calculated in 2 hour epochs and combined into daily hyperclusters by similarity in waveshape and topography. 24 hour EEG data of 50 epilepsy patients (25 children) were evaluated using traditional visual inspection and, independently, fast hypercluster evaluation.
Results: Visual rating yielded 130 epileptiform spike types. Hypercluster rating agreed in 88% (temporal lobe spikes 96%, extratemporal 83%). In a 24 h recording, about 15-25 hyperclusters had to be inspected to decide whether they reflected epileptiform discharges, artifacts, or other EEG patterns. The decision and reporting process was typically completed within 5 minutes by an experienced physician.
Conclusion: The traditional hourly evaluation of 2-5 minute epochs of long-term EEG can be readily supplemented by a computer-based hypercluster evaluation. This adds a fast, comprehensive overview and report, an independent control of the existence of one or multiple spike foci, and an estimation of their origin, and saves time in evaluating LTM-EEG. The involvement of the physician in the decision process allowed to increase sensitivity at the cost of reviewing and rejecting more artifact hyperclusters.

POSTER ABSTRACTS

p219

The utility of outpatient short-term video-EEG monitoring in the diagnosis of paroxysmal disorders

SENEVIRATNE U, RAHMAN Z, DIAMOND A, BRUSCO M
Monash Medical Centre, Melbourne, Australia

Purpose: To study the diagnostic yield of outpatient short-term video-EEG monitoring (STVEM) in the evaluation of epilepsy and psychogenic nonepileptic seizures (PNES).
Method: We retrospectively reviewed STVEM records done from February 2006 to February 2009. The demographic variables, indications for the test, abnormalities captured, diagnosis before and after the test and the findings of routine EEG were entered into a statistical database. The diagnostic yield of STVEM was compared with that of routine EEG.
Results: A total of 109 patients (76 females, 42 males) aged between 10 and 87 (mean 36) were studied. The mean duration of monitoring was 210 minutes (SD 43). Interictal epileptiform abnormalities were seen in 19% (focal sharp waves in 9%, generalized spike and wave discharges in 10%) in comparison to 18% in routine EEG. Non-epileptiform interictal abnormalities included focal slowing in 19% and generalized slowing in 9%. Clinical events were captured in over one third of cases (epileptiform events in 7%, PNES in 27%). After the STVEM the diagnosis was confirmed to be epilepsy in 20%, PNES in 24%, epilepsy coexistent with PNES in 1%, and inconclusive in 55%. In 13%, the STVEM resulted in changing the original diagnosis. In all except one the change was from epilepsy to PNES. There were no clinical events captured in routine EEG recordings.
Conclusion: STVEM helps establishing the diagnosis in paroxysmal disorders. The highest diagnostic yield is seen in PNES.

p220

Comparative study of ictal SPECT and video EEG in localization of epileptogenic foci for intractable epilepsy

YANG M-H[1], HUANG Y[1], YANG H-A[1], LIU S-Y[1], YANG H[1], AN N[1], HUANG T[1], LIU L-H[1], SHI X-J[1], CAI F-C[2], ZHANG Q[1]
[1]*Department of Neurosurgery, Xinqiao Hospital, Third Military Medical University, Chongqing, China,*
[2]*Children's Hospital, Chongqing University of Medical Sciences, Chongqing, China*

Purpose: To investigate the value of ictal single photon emission computed tomography and video EEG in localization of epileptogenic foci for intractable epilepsy.
Method: The data of ictal SPECT and video EEG were reviewed of 48 patients for intractable epilepsy, all the results were analyzed and compared with each other. 48 cases were performed the surgical treatment.
Results: The positivity rate of ictal SPECT and video EEG localization was 93.8% and 95.8%,The rate of complete coincidence of the two methods was 45.8% (22/48),partial coincidence was 29.2% (14/48). Comparing ictal SPECT with video EEG in the ratio of lateral and focal epileptic foci localizing, The value was $P>0.05$, $p< 0.01$. Twenty-seven of 48 patients had an Engel Class I outcome after surgery and an additional ten patients had rare seizure (Engel ClassII), and eight patient had a decrease in seizure frequency (Engel Class III). Engel Class I and ClassIIoutcome had 33/37 cases came from its coincidence of ictal SPECT and video EEG localization.
Conclusion: video EEG combined with ictal SPECT can learn from others a strong points to offset one's weakness, elevate the accuracy in localization of epileptogenic foci for intractable epilepsy, The good outcomes can be obtained to find foci and resecting.

POSTER ABSTRACTS

p221
An imPRESsive EEG: novel EEG features of posterior reversible encephalopathy syndrome
BOISSÉ L, BOYD JG, BRUNET DG
Queen's University, Kingston, Ontario, Canada

Purpose: Posterior reversible encephalopathy syndrome (PRES) includes encephalopathy, visual obscuration and seizures. It is associated with hypertension, although renal failure, chemotherapy and eclampsia can also be causative. Classic MRI findings include posterior cerebral white matter oedema with relative sparing of the gray matter, although recent evidence suggests that anterior white matter, and occasionally even grey matter can be affected. Furthermore, the reversible nature of PRES has come into question, as some patients sustain permanent neurological deficits. As PRES shows clinical heterogeneity, and its EEG has not been well characterised, our aim was to describe EEG features of PRES.
Method: We searched our EEG database over a six-year period (2003-2009) and collated the EEG findings of patients with clinical and radiological evidence of PRES.
Results: We recorded EEG's from twenty two patients with PRES. The majority of EEG's were characterised by generalised slow wave activity, although a small minority displayed focal epileptiform discharges, which are classic findings described in the literature. We also noted three previously undescribed EEG findings: one patient with refractory subclinical status epilepticus resulting in death, one patient with FIRDA, and one patient with needle spikes.
Conclusion: Our study documents electrophysiological heterogeneity in the EEG of patients with PRES. Further studies with larger numbers of patients should be conducted in the future to truly clarify EEG findings associated with PRES. In addition, clinical and radiological correlation of EEG findings could be conducted, as there may be a link between EEG findings and long-term prognosis.

p222
Automatic abnormal waves detection from the electroencephalograms of epilepsy cases to sort out the spikes, the sharps, the polyphase based on their zerocrossing and wavelet transformation
NOERTJAHJANI S
Undaris, Ungaran, Indonesia

Purpose: Wavelet transformation and zerocrossings 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 339M, 214F, ages 3-58 years. Clinical status: epileptic (131M,78F: EEG with spike;45M, 32Fwithout); clinical status: nonepileptic: (98M, 65F EEG with spike ; 65M, 39F without). Numerical data were acquired with EEG dump diagnostic Biologic system at Sarjito hospital Yogyakarta etc, 2002-2009. 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 +89μV threshold. The zerocrossings detection could automatically distinguished according to the 20ms-70ms time period for the "spikes" (107M,62F), 70ms-120ms for "sharps" (139M, 88F), and the existence of multiple peaks for " polyphase " (93 M, 64 F).
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.

POSTER ABSTRACTS

p223

Electrocorticography (ECoG) patterns of mesial temporal lobe epilepsy and neocortical temporal lobe epilepsy

DONGMING W
Yuquan Hospital, Tsinghua University, Beijing, China

Purpose: We summarized the electrocorticography (ECoG) patterns from the subdural grids implanted in the mesial temporal lobe and the neocortical temporal lobe originated epileptic patients. We further discussed the value of the functional localization.
Method: The ECoG signals of 60 patients who had either the mesial temporal lobe epilepsy or the neocortical temporal lobe epilepsy and were treated in our department from 2007 to 2008 were reviewed. We analyzed the ECoG signal patterns during seizure and after surgery.
Results: Within the signals from the mesial temporal lobe originated epileptic patients, 14 (23.3%) showed DC shifts, 8 (13.3%) showed low voltage fast activity, 3 (5%) showed theta oscillation, and 1 (1.7%) showed hypersynchronous pattern. Within the signals from the neocortical temporal lobe originated epileptic patients, 6 (10%) showed DC shifts, 10 (16.7%) had low voltage fast activity, 8 (13.3%) showed theta oscillation, and 1 (1.7%) showed hypersynchronous pattern. All the surgeries for these patients were the sections of the anteriotemporal lobe, hippocampus and amygdala. Twenty months after the surgeries, we revisited the patients and defined the recovery level according to the Engel coefficients.
Fifty-nine patients were revisited and 55 (86.7%) patients got level I, 3 (5%) got level II, 1 got level III.
Conclusion: DC drifts, low voltage fast activity, and hypersynchronous pattern were the usual signal patterns of the mesial temporal lobe and the neocortical temporal lobe originated epilepsy. The surgery would be successful if the ECoG pattern during seizure showed DC drifts or low voltage fast activity.

p224

Bemegride-induced test in EEG recording for preoperative localization of temporal lobe epilepsy

LI A[1], LIANG S[1], LIANG S[2], SUN Y[2]
[1]Neurosurgery Department, First Affiliated Hospital of General Hospital of PLA, Beijing, China, [2]Epilepsy Center, First Affiliated Hospital of General Hospital of PLA, Beijing, China

Purpose: To study effect of bemegride-induced test on seizure attack and epileptoform discharge on EEG in patients with temporal lobe epilepsy.
Methods: To retrospectively analysis data of preoperative EEG recording 60 patients' with anterior temporal lobectomy, including 30 cases in experience group with bemegride-induced test and 30 cases in control group without bemegride-induced test. The differences of EEG recording times, seizure times and types, percentage of postoperative seizure freedom were analyses between two groups, and the difference of EEG before and after induced test in experience group was also compared.
Results: Significant difference was found in EEG recording time: 35h in experience group and 56h in control group. There were 99 bemegride induced seizure in experience group, including 71(71.7%) in 5 minutes after induced test, 18(18.2%) in 6-60min, and 10 non-habitual seizures. Also, there were 102 seizures in control group during EEG recording, including 4 (3.9%) non-habitual seizures, and significant difference wasn't found in seizure times and types between groups. Interictal epileptiform discharge frequency increased, but the localization didn't obvious change after bemegride induced test, and bemegride induced ictal discharges was mainly localized on unilateral temporal lobe which was almost same with habitual seizure.
Conclusion: Bemegride induced test didn't affect localization of EEG recording for temporal lobe epilepsy, and can be used in preoperative localization and reduce recording time.

POSTER ABSTRACTS

p225

Electroencephalographic findings among patients with complex febrile seizure: a 5-year review

ADAJAR JA
University of the Philippines-Philippine General Hospital, Manila, Philippines

Purpose: This study aimed to describe electroencephalographic findings in patients diagnosed with complex febrile seizures (CFS) from 2004-2008 in a Philippine tertiary hospital.
Method: A retrospective chart review was done on all neurologically normal children with complex febrile seizure who underwent electroencephalography (EEG) anytime after the index seizure. Frequency distribution and means were the descriptive statistics used in the study.
Results: Eighty-eight EEGs were included in the final analysis. 63 EEGs were normal and the remaining 25 had abnormalities. Of those with abnormal EEG, 48% had recurrent seizures, 28% focal seizures, 16% prolonged seizures and 16% had more than 2 complex features. Majority of EEG abnormalities were focal interictal epileptiform activity followed by diffuse slowing of background activity. These were seen in patients less than 2years old. Majority of abnormalities were noted when the EEG was performed within the first 10 days of the seizure episode (44%). Among the subjects with recurrence or multiple seizures as the complex feature, 58.6 percent of EEG abnormalities were noted when the seizure recurred three times or more.
Conclusion: The results of this study suggest that the leading correlates of paroxysmal EEG abnormality identified were younger age, multiple seizures of three or more, and those with EEGs done within first 10days from the index seizure. Focal interictal epileptiform activities more prominent over the frontal regions were also an important finding.

p226

Use of EEG in cerebrovascular disorder (Moya-Moya disease)

COLEBATCH JG[1,2], MALHOTRA RC[3,4]
[1]Head, Department of Neurology, Prince of Wales Hospital, Sydney, Australia, [2]UNSW, Sydney, Australia, [3]Prince of Wales Hospital, Randwick,Sydney, Australia, [4]UNSW, Randwick, Sydney, Australia

Purpose: To describe the usefulness of EEG in suggesting the diagnosis of Moya moya disease in a patient thought to have seizures.
Method: Case report.
Case: A 9 year old boy presented with episode of unsteady gait and decreased level of consciousness .He was noted to be blankly staring, dropped a plate from his left hand, complained of left arm feeling fizzy and weak .His gait was very ataxic and his mother noted a left sided facial droop. These symptoms resolved after a few minutes. Over the next six months, he had at least another fifteen events of abnormal sensation and movements .During most of these episodes he is noted to have right sided facial droop associated with marked dysphasia and language abnormality. There was no clear history linking these episodes to activity, with events often occurring at rest. They could occur at any time during the day.
A CT scan of the brain showed a focal area of well defined hypodensity in the superior aspect of the head of the caudate nucleus, lateral aspect of the right corona radiata and in the right centrum semiovale.
An EEG captured an episode of altered responsiveness following a hyperventilation manoeuvre and there was prominent post-hyperventilation slowing after a delay.
An MRI scan with MRA showed that both internal carotid arteries are occluded in their supraclenoid segment with multiple leptomeningeal collateral vessels suggestive of Moya-Moya disease.
Conclusion: In children with moyamoya disease,slow waves reappear(“ re-build up”) after hyperventilation(represents a focal reduction of the cerebral perfusion reserve).This post hyperventilation slowing is characteristic of moya moya disease and of potential useful screening test for children presenting with episodic cerebrovascular events.

POSTER ABSTRACTS

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p227
EEG findings in children with anti-N-methyl-D-aspartate receptor (NMDAR) encephalitis
PILLAI SC[1], DALE RC[2], GILL D[1]
[1]TY Nelson Department of Neurology and Neurosurgery, Westmead, Australia, [2]University of Sydney, Discipline of Paediatrics and Child Health, Sydney, Australia

Purpose: NMDAR encephalitis is a newly defined autoimmune limbic encephalitis characterized by the presence of antibodies against the glutamate (NMDA) receptor. The clinical phenotype consists of an encephalopathy with psychiatric disturbances, a movement disorder, seizures, dysautonomia and sleep dysfunction. We detail the EEG characteristics of five children who were retrospectively diagnosed with NMDAR encephalitis.
Methods: Twenty-four EEG's (median 3 per patient; range 1-11) were reviewed. The recordings were reviewed for background rhythms, regional slowing and epileptiform activity. The anterior-posterior montage was used in viewing the EEG recordings. Video recordings were often available used to distinguish epileptiform activity from movement related artefacts.
Results: Generalised slowing was seen in all five patients throughout the disease course. Three patients had bifrontal fast 12 Hz activity. Two patients demonstrated epileptiform activity in the form of spike and wave. In addition, prolonged spindles were seen in two patients. Two of the five patients had clinical seizures which were complex partial seizures and secondary generalized tonic-clonic seizures.
Conclusion: The generalized slowing seen in all patients is consistent with a diffuse encephalopathy. The bifrontal fast activity may be related to medications while the prolonged spindles seen in three patients may be an endophenotype of NMDAR encephalitis. The video EEG is helpful in distinguishing seizures from various movement disorders such as dystonia, dyskinesia and oculogyric crisis which are seen in this condition.

p228
Electroencephalographic patterns in intensive care unit patients
CINAR N, SAHIN S, OKLUOGLU T, TELLIOGLU N, KARSIDAG S
Department of Neurology, Faculty of Medicine, Maltepe University, İstanbul, Turkey

Purpose: We review the patterns of electroencephalography (EEG) in the intensive care unit.
Method: Thirty-eight patients (19 women and 19 men) underwent EEG recordings. Mean age was 72±14 (25-90) years, mean Glasgow coma scale (GKS) score was 7±2 (3-13). The primary cerebral insult (n:11, 27%), respiratory-cardiac (n:14, 37%) and methabolic (n:13, 34%) pathologies were responsible of these encephalopathy cases. The EEGs were evaluated, using visual inspection by an expert who have not aware of the patient´s informations. The EEG patterns were classified as frontal and occipital intermittent rhythmic delta activity (FIRDA, OIRDA), periodic lateralized epileptiform discharges (PLEDs), burst-suppression (BS) and continuous high-voltage delta activity (CHVDA). Patients were divided into two groups according to the GKS. Group I: GKS< 8 (n:25) and Group II: GKS between 9-13 (n:13). There was no observed any clinical seizure activity in patients.
Results: The distribution ratio of EEG patterns were FIRDA-34%, OIRDA-11%, PLED- 29%, BS-11% and CHVDA-16%. Similar wave patterns were observed in all types of encephalopathies and according to the GKS there was no significant differences between the two groups.

POSTER ABSTRACTS

Conclusion: EEG monitoring is important, especially in patients who are stuporous or in coma. EEG monitoring permits clinical and subclinical changes to be followed over time, and may capture transient events such as seizures when they are not manifest clinically. In our study FIRDA and PLEDs waveforms are most commonly observed and we found no statistically significant difference between the etiologies and GKS scores. Further studies are needed included more patients.

p229

Serial electroencephalographic findings in patients diagnosed with subacute sclerosing panencephalitis given intraventricular ribavirin

VICTA EAT, SANCHEZ-GAN BC, LUKBAN MB
University of the Philippines - Philippine General Hospital, Manila, Philippines

Purpose: To describe the background activity, presence of periodic discharges and epileptiform activity with respect to the duration of illness and clinical stage of the disease of 12 SSPE patients. Another objective is to look for any differences before and after ribavirin administration.
Method: Twelve patients with SSPE included in a therapeutic trial on the use of intraventricular ribavirin for SSPE (Lukban et al, unpublished) were enrolled. EEG recordings of the patients which were done prior to the administration of ribavirin were retrieved. Repeat EEGs were then scheduled 3-6 months and 9-12 months after ribavirin administration. Thirty six (36) electroencephalographic recordings from 12 patients with SSPE were studied.
Results: The background activity mostly showed mild to moderate diffuse slowing with 4 severe slowing seen within 2 years duration of illness. There was only one recording with normal background activity which was done with duration of illness of 15 months.
Periodic complexes (17%) and burst suppression pattern (41%) were noted mostly with duration of illness of less than 2 years and a third of the recordings did not show epileptiform activity. Focal discharges were generally more frequent than generalized epileptiform activity.
Conclusion: Subacute sclerosing panencephalitis presents with a wide array of electroencephalographic findings. The typical changes seen in SSPE including periodic complexes and burst suppression pattern were mostly seen in the first 2 years of illness.
Ribavirin therapy had varying effects on the EEG. Some had marked improvement, some deteriorated while a third did not show any change.

p230

Minimally-invasive ECoG recording using the subdural microelectrode array guided by the shape memory alloy wire

YAMAKAWA T[1,2], YAMAKAWA T[2,3], AOU S[3], ISHIZUKA S[3], FUJII M[4], SUZUKI M[4], AOKI T[1]
[1]Shizuoka University, Hamamatsu, Japan, [2]Fuzzy Logic Systems Institute, Iizuka, Japan, [3]Kyushu Institute of Technology, Kitakyushu, Japan, [4]Yamaguchi University School of Medicine, Ube, Japan

Purpose: Due to the electrical characteristics and to the physical layout of the subdural strip/grid electrodes, the recoding area is limited by the surgical area of craniotomy. In order to reduce the invasiveness, we propose the novel subdural microelectrode array guided by a 0.3mm-diameter shape memory alloy (SMA) guidewire.
Method: The Platinum microelectrodes were mounted on the SMA guidewire whose shape were memorized in advance. Since the SMA guidewire is thin and flexible enough in the room/body temperature, the microelectrodes is able to be slipped into the subdural space without injury. After insertion, the electric current is applied and the SMA guidewire is heated by Joule heat. Then the microelectrodes are deployed to the desired positions. Here, the main part of the SMA guidewire was programmed to recover a hexagonal shape, and the microelectrode was mounted on the each vertex of the hexagon. The SMA guidewire is electrically and thermally isolated from the body by two layers of PTFE insulator.

POSTER ABSTRACTS

Results: In a rhesus monkey under general anesthesia, the microelectrodes were slipped into the subdural cavity around the primary somatosensory cortex through a 7mm-diameter hole made on the skull, and were deployed by the DC current. The somatosensory evoked potential was successfully measured with electrical stimulation on the contralateral upper limb.
Conclusion: The SEP of a rhesus monkey was measured by the proposed minimally-invasive ECoG recording method. The results suggest that the proposed method would improve the ECoG recording for the focus detection of intractable epilepsy.

p231
Developing electro-encephalopgram (EEG) service in the management of childhood epilepsy in Bangladesh: an overview
BANU SH[1], HOSSAIN M[2], ISLAM F[3], RANI S[4], KHAN NZ[3]
[1]ICH & SSF Hospital, Dhaka, Bangladesh, [2]ICMH, Matuail, Dhaka, Bangladesh, [3]BICH, Dhaka Shishu Hospital, Dhaka, Bangladesh, [4]Comfort Diagnostic Center, Dhaka, Bangladesh

Background: Treatment gap for childhood epilepsy and comorbidities is huge in developing countries. Attitude of the stake holders was found to be the most important cause in Bangladesh (SH Banu, PhD thesis, 2003). EEG probably plays an important role in reducing this gap.
Purpose: To report changing pattern of presenting complaints and their electro-clinical correlation to answer the question why EEG service is essential to treat neurodevelopmental disorders in children.
Methods: We analyzed electro-clinical findings of 25000 children referred to the first established EEG service for children in Dhaka. 1. 1st 1000 EEGs clinical profiles were reported before. We chronologically reported the changing pattern of their clinical profile through the years. 2. A sub-population with diagnosed epilepsy was studied prospectively to analyze their seizure outcome after regular treatment and correlated with their EEG features. 3. An inter-observer reliability test was performed with their EEGs, reported by two neurophysiologists, one remained blind to the child's clinical information.
Result: 1. Increasing number of children was referred with non-convulsive disorders including mental health, speech and communication disorders during the recent years. Their EEG findings were also remarkable. 2. 1st EEG feature had significant correlation with the seizure outcome, revealing the prognostic value of EEG. 3. The inter-observer reliability study revealed very good kappa score.
Conclusion: This study suggested that EEG has important role in evaluating the convulsive and non-convulsive neurodevelopment disorders in children. A multidisciplinary approach should include EEG to treat them in developing countries like Bangladesh.

p232
Measuring cortical excitability using iEEG responses to an electrical probing stimulus
FREESTONE DR[1,2,3], GRAYDEN DB[1,3], NELSON TS[1], LAI A[1], BURKITT AN[1,3], VOGRIN S[2], KUHLMANN L[3], COOK MJ[1,2,3]
[1]The Bionic Ear Institute, East Melbourne, Australia, [2]St. Vincent's Hospital, Fitzroy, Australia, [3]The University of Melbourne, Parkville, Australia

Purpose: To relate cortical excitability to the pre-seizure state, with the aim to develop a method for seizure anticipation.

POSTER ABSTRACTS

Method: Patients with intracranial EEG (iEEG) electrodes undergoing evaluation for epilepsy surgery at St. Vincent´s Hospital, Melbourne, were recruited for this study (under human research ethics approval). Electrical probing stimuli were delivered in groups of 100 pulses, with the pulses in each group spaced 3 seconds apart. Each group of 100 pulses was triggered every 10 minutes. This provided 5 minutes of stimulations, followed by 5 minutes of rest. The protocol continued over days of recording for each patient. Following this protocol, a measure of cortical excitability was obtained every 10 minutes (averaged over the 100 stimulations in each group). Phase synchronisation of iEEG was used as a surrogate for excitability.
Results: We show that this method shows promise with successful seizure anticipation in our patient group, motivating further research following this approach.
Conclusion: A probing stimulation provides more information than is available from passive iEEG. Further research into this method may lead to breakthroughs in the field of epileptic seizure prediction.

p233
Pre-tapering of antiepileptic drugs before video EEG monitoring to reduce the monitoring time
KHARBANDA P, PRABHAKAR S, KAUR S
Dept. of Neurology. Postgraduate Institute of Medical Education & Research (PGIMER), Chandigarh, India

Purpose: Tapering of antiepileptic drugs (AEDs) is often required for recording seizures during long term video EEG monitoring (LTVEM). This is usually carried out after initiation of the recording, which leads to increased monitoring time and the consequent costs. This prospective study evaluated the benefits and risks of tapering AEDs prior to starting of LTVEM.
Method: Fifteen patients requiring LTVEM were included in the study. All patients had suspected temporal lobe epilepsy (11 Mesial Temporal Sclerosis, 2 Dysembryoplastic Neuroepithelial Tumor). Only patients, who had seizure frequency of less than one per week, had their AEDs pre-tapered at the rate of one third per day, starting three days prior (Day1-Friday) to the initiation of VEM (Day4-Monday).
Results: Fourteen patients had 69 partial and 11 generalized seizures in all. Fifty one (74%) of the partial and all 11 generalized seizures occurred between Day4 and Day7 of tapering. 12 (80%) patients had seizures between Day4 and Day7. Eight (53%) had seizures including GTCS between Day6 & Day7. No patient had status epilepticus.
Conclusion: This study reveals that pre-tapering of antiepileptic drugs 3 days prior to initiation of LTVEM was a safe and effective method to cut down the monitoring time. Majority of the patients had seizures recorded during first four days of monitoring. Very few partial and no generalized seizures occurred before the initiation of monitoring. If the tapering had been started along with monitoring on a Monday, many patients would end up having seizures during the next weekend (Day6 and 7)

p234
A simple change to photic stimulation markedly improves EEG diagnosis of first seizures
MULLEN S, BERKOVIC S
University of Melbourne, Department of Medicine AH/NH, Melbourne, Australia

Purpose: To evaluate a simply implemented change in EEG protocol for the diagnosis of idiopathic generalised epilepsy (IGE) in a first seizure cohort.

POSTER ABSTRACTS

Method: In June 2006 we implemented an Intensified IPS protocol, replacing a plain glass protocol based on the gold-standard, European Consensus statement. This involved re-ordering frequencies to repeat the critical 10-20Hz band and adding a fine, metal mesh. A small LED strobe was used. Results of EEGs done for first seizure 30 months prior to and 30 months following implementation were audited. Spontaneous focal and generalised epileptiform changes were noted as well as the presence or absence of a PPR.
Results: There were 466 studies studied prior to the change in IPS and 526 after. A PPR was present in 3.2% of EEGs prior to intensified IPS and 6.2% post (P< 0.05). EEGs in which a PPR was the only abnormality went from 5% of all IGE related abnormalities to 20% after introduction of the intensified IPS (P< 0.05). Plain glass IPS thus detects 6% more IGE cases than resting and hyperventilated EEG alone while intensified IPS detects 25% more.
Conclusion: A simple change to IPS can improve detection of IGE related EEG changes by around 20% as compared to the current gold-standard protocol.

p235
Correlation between contrast-enhanced cranial MRI and long-term ictal EEG in determining focus for surgery in patients with temporal lobe epilepsy
BINTORO AC, KUSTIOWATI E
Neurology Departement, Dr Kariadi Hospital / Diponegoro University, Semarang, Indonesia

Purpose: To determine correlation between contrast-enhanced cranial Magnetic Resonance Imaging (CE-MRI)) and long-term ictal EEG (Electroencephalography) in determining focus for surgery in patients with temporal lobe epilepsy
Method: We analyzed all patients data with clinical diagnosis of temporal lobe epilepsy that underwent CE-MRI and longterm EEG ictal from January 2006 until March 2009 in neurophysiology labroratory dr. Kariadi General Hospital, Semarang, Indonesia. Two neurologist reviewed their CE-MRI and long-term EEG based on presence of sclerosis on the temporal lobe and epileptiform on the temporal lobe. Inter-reliability agreement between two reviewer was completed. Then we analyze their correlation among both procedures in determining focus of epilepsy.
Results: There are 51 patients underwent CR-MRI, but only 45 (88%) patients have focus during ictal long-term EEG recording; 13 patients had an epileptic focus on the right anterior temporal area, 27 patients had an epileptic focus on the left anterior temporal area, 5 patients had an epileptiform focus from both anterior temporal area. On CE-MRI, we found 24 patients had a right hippocampal sclerosis, 25 patients have a left hippocampal sclerosis,
2 patients had both hippocampal sclerosis. Inter-rater agreements between two examiners are good.
Conclusion: We found that CE-MRI maybe more sensitive determining anatomical abnormality as an etiology of temporal lobe epilepsy but long term ictal EEG are more specific to determine the focus of temporal lobe epilepsy

p236
EEG features of idiopathic and symptomatic epilepsies
BAYASGALAN B[1], SUVD R[2]
[1]Second General Hospital, Ulan Bator, Mongolia, [2]2EEGMON Clinic, Ulaanbaatar, Mongolia

Purpose: The aim of our study was to clarify EEG characteristics of idiopathic and symptomatic epilepsies.
Method: 157 out-patients who were diagnosed with epilepsy by anamnesis data and had undergone EEG recording were involved in the study. Patients age was between 4 to 78 years (mean age 26.09). There were 92 males and 65 females; sex ratio was 1.5: 1.0. Patients were grouped into three groups as those with symptomatic epilepsy (108 or 68.8%) and idiopathic epilepsy (13 or 8.3%) and other than these two (36 or 22.9%). The ratio of symptomatic, idiopathic and other epilepsies was 7:2:1.

POSTER ABSTRACTS

Results: The EEG characteristics of symptomatic epilepsies was followings: 73.2% had epileptiform discharges, 18.5% had non-specific epileptiform discharges and in 8.3% there were not detected epileptiform discharges. In a group of patients with idiopathic epilepsy all patients EEG showed epileptiform discharges.
Conclusion: Our study shows once again that EEG recording is one of the most informative tools for diagnosis of epilepsy.
Acknowledgement: Authors wish to thank all staff of Shizuoka Epilepsy Center Japan for their kind donation of an EEG machine.

Seizure semiology

p237
A gut feeling about insula seizures
DIONISIO S[1], SOMERVILLE ER[1,2], KOENIG A[1]
[1]Prince of Wales Hospital, Comprehensive Epilepsy Service, Sydney, NSW, Australia, [2]University of New South Wales, Sydney, NSW, Australia

Purpose: To describe an unusual presentation of insular seizures.
Method: Case report - A 43 year old man experienced a single generalized tonic-clonic seizure. For the previous two years, he had undergone several gastroenterological assessments and investigations for sudden episodes of gagging, hypersalivation and lacrimation, occurring three to four times per week. One of four endoscopies had revealed H pylori and oesophagitis. However treatment with antibiotics and a proton pump inhibitor had no effect on the episodes. A typical episode immediately preceded the tonic-clonic seizure.
Results: Two seizures were recorded with video-EEG, comprising 10-20 second episodes of choking sensation, lacrimation, hypersalivation and dysarthria. EEG was obscured by artifact. Interictal F8 spikes were seen. ECG monitoring did not reveal any ictal arrhythmia.
Brain MRI revealed a cavernous haemangioma in the right insula and FDG PET showed this area to be hypometabolic.
No further episodes have occurred during 5 months of therapy with carbamazepine.
Conclusion: The insula has both visceral and autonomic functions. Insular seizures have a wide semiology, including laryngeal discomfort, dyspnoea, peri-oral and somatic paraesthesia and abdominal pain. SUDEP due to cardiac dysrhythmias may occur. Various studies have categorized the right insula as having a sympathetic predominance and the left a parasympathetic role. However, in our patient, the primarily parasympathetic symptoms were due to a right sided lesion.
Insular epilepsy should be considered in the differential diagnosis of episodic brief atypical gastrointestinal symptoms not responding to therapy.

p238
To determine localization value of hypersalivation
GUL G, KORKUT E, KUŞCU DY, BEHREM N, KİRBAŞ D
Prof. Dr. Mazhar Osman Education and Research Hospital, 3rd Neurology Clinic, Istanbul, Turkey

Purpose: A wide range of autonomic phenomena have been described to occur in TLE, some of these provide valuable information on seizure origin. Increased salivation only occasionally has been reported as a manifestation of partial epilepsy. We aimed to determine whether hypersalivation helps lateralize/localize seizure onset during complex partial seizures
Method: We reviewed the charts of 463 patients for this seizure element which was seen by a witness or recorded during video-EEG monitoring. We found 17 cases of hypersalivation during complex partial seizures. We reviewed the clinical features, scalp- video-EEG monitoring, magnetic resonance imaging (MRI) of these patients.

POSTER ABSTRACTS

Results: Taking into account the side of seizure onset, hypersalivation was found in 11/17 of the patients with left sided seizures and in 6/17 of the patients with right sided seizures. Seven of these 11 patients had left MTS, 3 of them had extra-mesial lesion and 1 of them had non lesional MRI. Four of 6 patients had right MTS and 2 of them had extra-mesial lesion. Thus, hypersalivation was rare, but occurred exclusively in seizures of mesial origin.
Conclusion: Increased salivation has been reported rarely as a manifestation of partial seizures, the occurrence of hypersalivation strongly supports mesial seizure onset but does not further differentiate lateralization. However, the present sample is small, so that further studies with larger numbers of patients are needed to clarify this point.

p239
The effects of morphine and naloxon on spontaneuse seizure activity in hippocampal slices
ALIJARAHI A, ESMAEILI MH, AMINI M
Qazvin Azad University, Qazvin, Iran, Islamic Republic of

Purpose: Several studies demonstrate that morphine has a wide variety of effects on neuronal excitation and inhibition involved in seizures. There is however very little information available about the effects of morphine on recurrent spontaneous seizures. Therefore the present study was designed to determine the effects of different dose of morphine and Naloxon on spontaneous seizure activity in epileptogenic hippocampal slices.
Method: Hippocampal slices (~400 µm) were prepared from young Wistar rats (P15- 25). Seizure activity was induced by continuously perfusing the slices with low Mg2+ perfusate in an interface recording chamber. Extracellular recordings were performed in the hippocampal CA1 pyramidal cell layer. Low Mg2+ caused spontaneous epileptic activity in all studied hippocampal slices .Seizure activity was quantified by measuring the amplitude and duration of the ictal events as well as their number before and after the application of the study drugs. Also, the numbers of interictal spikes were determined to complement the analysis of seizure discharges before and after drug application
Results: Low doses of morphine (10 µM) suppressed seizure activity, whereas high doses of morphine (15, 30 & 100 µM) potentiated seizure activity in a dose dependent manner. This effect was completely reversed by the addition of Naloxon (10 ìM).
Conclusion: Differential effects of morphine on seizures, suggests that morphine in different concentrations acts on different receptor subtypes or by different mechanisms. activation of opioid receptors by morphine lead to suppression of GABAergic synaptic transmission thereby disinhibiting pyramidal neurons, resulting in the enhancement seizures.

Psychiatry

p240
Do disturbances in hypothalamic-pituitary-adrenal axis and autonomic function play a role in the comorbidity of depression in patients with epilepsy?
SOH LJH[1], TURNBULL C[2], TAN M[2], LOU N[1], JACKSON B[2,3], ADAMS S[2], VELAKOULIS D[2,4], O'BRIEN T[1], SALZBERG M[1,5]
[1]University of Melbourne, Department of Medicine, Melbourne, Australia, [2]Royal Melbourne Hospital, Melbourne, Australia, [3]Swinburne University of Technology, Melbourne, Australia, [4]Melbourne Neuropsychiatry Centre, Melbourne, Australia, [5]St Vincent's Hospital, Melbourne, Australia

POSTER ABSTRACTS

Purpose: Depression is a common comorbidity in patients with epilepsy, and has been particularly linked with temporal lobe epilepsy (TLE). The mechanistic explanations for the link between epilepsy and depression are still poorly understood, but disturbances in the hypothalamic-pituitary-adrenal (HPA) axis and autonomic function have been implicated. The mesial temporal structures are intimately involved in regulation of HPA axis and autonomic function. The purpose of this study is to identify whether there is an interaction between temporal lobe epilepsy and depression on measures of the HPA axis and autonomic function.
Methods: Patients admitted to the RMH hospital in-patient video-EEG monitoring unit for either uncontrolled epilepsy or psychogenic non-epileptic seizures (PNES) were offered enrolment. Salivary cortisol was measured to assess the diurnal variation and cortisol awakening response in 22 patients (7 TLE, 6 non-TLE epilepsy and 9 PNES). Heart rate variability and the pre-ejection period were used to estimate parasympathetic and sympathetic activity respectively in 24 patients (7 TLE, 6 non-TLE epilepsy and 11 PNES). Patients underwent neuropsychiatric assessment at the time of admission.
Results: For the cortisol levels, a significant interaction effect between epilepsy and depression was identified but was not different between those with TLE and non-TLE epilepsy. No differences between the groups for measures of autonomic function were found.
Conclusion: Disturbances in HPA function may play a role in the pathophysiology of the comorbid depression in patients with epilepsy, but this does not appear to be specific for TLE.

p241
Co-morbidity of autistic spectrum disorder with epilepsy in Chinese children
HAN Y, QIN J, CHEN J, JI X, JIANG Y, LIN Q
Peking University First Hospital, Beijing, China

Purpose: Children with autistic spectrum disorder (ASD) have an increased prevalence of seizures. However, few studies have evaluated the prevalence of ASD in children with epilepsy. The proper intervention to this co-morbidity is of potential role in long-term prognosis of the particular population. The aim of the present study is to estimate the prevalence of ASD in Chinese children with epilepsy, and the factors that may contribute to this co-morbidity.
Method: The sample included 321 epileptic children (205 boys and 116 girls), aged 3-15 years. The ages of onset, the ages at diagnosis, the epileptiform discharges, the types of seizures and syndromes, and the treatment were recorded. The ASDdiagnostic assessment was evaluated by semi-structured clinical interview, Childhood Autism Rating Scale (CARS) and Autism Behavior Checklist (ABC).
Results: Among the 321 children, there were 19 children who had higher scores in CARS detection, and there were 27 children who had higher scores in ABC measurement. Adding clinical interviews in accordance with the diagnostic criteria of DSM-IV, 19 children were diagnosed with ASD. The prevalence of ASD in Chinese children with epilepsy is 5.92%. Symptoms of ASD are more common in some specific types of epilepsies, such as West Syndrome, Lannox-Gastaut syndrome and Landau-Kleffner syndrome.
Conclusion: ASD occurred really frequently in Chinese children with epilepsy. The factors contributed to the increased risk for ASD included the chronic effects of seizures and the epileptiform EEG discharges. The types of seizures or epileptic syndromes were also associated with different risk for ASD co-morbidity.

POSTER ABSTRACTS

Neuropsychology

p242
The relationship between fatigue and C-reactive protein in epilepsy patients
KIM K
Kosin University College of Medicine, Busan, Korea, Republic of

Purpose: To determine the rate of fatigue in epilepsy patients and its relation to C-reactive protein (CRP), fibrinogen and homocysteine levels.
Method: Subjects were 39 epilepsy patients who were receiving antiepileptic drugs, and controls were composed of age- and sex-matched 20 healthy person. Fatigue was evaluated by using Fatigue Severity Scale (FSS). The plasma CRP, fibrinogen, and homocysteine levels were measured by turbidimetric immunoassay, scattered light detection method, and chemiluminescence immunoassay respectively. Univariate regression analysis was performed to assess the relationship between fatigue and CRP, fibrinogen, and homocysteine.
Results: In patients with epilepsy the mean score of FSS was 4.54±1.69, and the rate of fatigue was 66.7%. When compared with normal controls, the mean CRP level of epilepsy patients was significantly elevated ($p < 0.01$). Fatigue was significantly correlated with elevated serum CRP level in epilepsy patients ($p < 0.05$). However, associations between fatigue and fibrinogen and homocysteine were not statistically significant.
Conclusion: These findings suggest that fatigue in epilepsy patients may be correlated with elevated serum CRP level.

p243
The cognitive effects of age at seizure onset in childhood symptomatic focal epilepsy
EMBULDENIYA U[1,2], GONZALEZ LM[1,2], WOOD AG[1,2], HARVEY S[2], WRENNALL J[2], TESTA R[1,3]
[1]Monash University, Melbourne, Victoria, Australia, [2]Royal Children's Hospital, Melbourne, Victoria, Australia, [3]Sunshine Hospital, Melborune, Victoria, Australia

Purpose: The aims of this research were to clarify the effects of epilepsy and developmental variables on cognitive functioning, and in particular, the effects of timing of seizures during early childhood.
Method: Cognitive abilities of children with symptomatic focal epilepsy (SFE) (N=35) were assessed for whole-group analyses. For onset group comparisons, sample consisted of children with onset either between 3-5 years (early-onset;EO; N=18) or 6-8 years (late-onset;LO; N=8). To preclude the confounding effect of duration, only those with both age at onset and age at assessment within these age ranges were included. The cognitive domains of language, attention, memory and visuospatial skills were studied.
Results: The findings of this research indicate that children with SFE demonstrate statistically significant widespread deficits relative to normative standards, in the areas of language (vocabulary, phonological processing, and verbal reasoning), visuomotor coordination, attention span, and memory. Children with secondarily generalised seizures, on polytherapy and from families with lower socioeconomic status were more likely to have cognitive deficits. In regard to onset effects, the EO group performed more poorly relative to both the LO group and normative standards, particularly for skills that were being acquired at time of seizure onset. Visuospatial skills appear especially vulnerable to the effects of seizures during
early childhood.
Conclusion: There is strong case for the role of development in mitigating outcome in children with SFE, with evidence that earlier age at seizure onset is associated with greater cognitive burden than later onset and typically-developing children.

POSTER ABSTRACTS

p244

Evaluation of the material-specific amnesia model in mesial temporal lobe epilepsy

GIUMMARRA J[1], BOWDEN SC[1], MURPHY M[2], COOK M[2]
[1]The University of Melbourne, Melbourne, Australia, [2]St Vincent's Hospital, Melbourne, Australia

Purpose: Minimising post-operative memory deficits is of key concern when offering surgery to individuals with mesial temporal lobe epilepsy (MTLE). Review of the literature suggests much uncertainty regarding material-specific amnesia hypotheses. However, this uncertainty may arise in part from heterogeneity in methods of memory assessment, as well as heterogeneity of patients studied. In this study, we report on a sample of patients with MTLE offered surgery on the basis of volumetric MRI identified hippocampal sclerosis.
Method: Wechsler Memory Scale (WMS-III) indices are reported for 46 patients with left TLE and 44 patients with right TLE seen before and after anterior-temporal lobectomy.
Results: There were no statistically significant differences in verbal or visual memory indices between patients with left- versus right-lateralised seizure foci prior to surgery (all p's > .1). Left TLE patients showed no change in their verbal memory abilities and significant improvement in their visual memory abilities. In contrast, right TLE patients demonstrated the reverse pattern. These effects were highlighted in the significant 3-way interaction between memory type (verbal vs. visual), assessment time (pre- vs. post-operative) and side of seizure focus (left TLE vs. right TLE) $F(1, 88) = 12.29$, $p < .01$.
Conclusion: Results of this study show that, when selected for surgery on the basis of hippocampal volumetrics, mean verbal memory performance in left TLE patients does not deteriorate. Similarly mean visual memory performance does not deteriorate in patients with right TLE. Both patient groups showed modest improvements in contralateral memory function.

p245

Cognitive function in the newly diagnosed juvenile myoclonic epilepsy

WANG T
The Second Hospital of Lanzhou University, Lanzhou, China

Purpose: To study cognitive function in the newly diagnosed juvenile myoclonic epilepsy and estimate the related factors to cognitive function in patients with juvenile myoclonic epilepsy.
Method: 22 newly diagnosed adult patients with juvenile myoclonic epilepsy and 22 healthy volunteers were tested by 5 neuropsychological tests including Auditory verbal memory test ,Verbal fluency ,Trail making test (TMT), the Stroop test, Digit span as well as Mimi mental state (MMSE) and mood (Self-Rating Anxiety Scale SAS, Self- Rating Depression Scale SDS), To examined The relation between the scores of neuropsychological tests and the risk factors using Pearson's correlation test.
Results: compared with healthy volunteers, the newly diagnosed patients with juvenile myoclonic epilepsy exhibit worse performances in the immediately and delayed recall ($p< 0.01$), recognition task ($P<0.01$), verbal fluency ($p< 0.01$), the reading words and color time of the Stroop - test ($p< 0.01$), forward and backward digit spans ($p< 0.01$). The scores of the MMSE test, SAS,and SDS did not differ between the group. There were significant correlations between verbal memory and onset age.when compared with the newly diagnosed patients ($r=0.450$, $p< 0.05$), the patients with intreictal epileptiform discharge showed lower score in the immediately and delayed recall of auditory verbal learning test ($p< 0.05$),there were no significant differences in the attention and executive function
Conclusion: the newly diagnosed patients with juvenile myoclonic epilepsy may exhibit impaired cognitive function in terms of memory and attention and execution, while having normal intelligence and mood.

POSTER ABSTRACTS

p246

Subjective well-being (SWB) in men with adult onset epileptic seizures (AOES) during the pre-ictal stage of a complex partial seizure (CPS)

RAFFAELE M
University of Sydney, University of Sydney, Australia

Purpose: This ethnographic research paper focuses on the subjective well-being (SWB) in men with adult onset epileptic seizures (AOES) during the pre-ictal period. How the lack of self-awareness and social judgement can result in a lowering of SWB. How providing knowledge to those closest to the male with complex partial seizure (CPS), on pre-ictal symptoms not only assists with the recognition of an oncoming ictal period, but also provides understanding for difficulties in personal relationships.
Method: The qualitative research method gathered data via a semi-structured interviewing process. Both men with AOES and their partner have been individually interviewed. A support group for men with epilepsy and those closest to them have been observed. A constructivist interpretive paradigm was used when observing and analysing data. Both major constructivist approaches; Individual, and social were adopted to gain understanding from the internal and external points of view.
Results: Many participants were unaware of the five characteristic changes in personality that often arise in the pre-ictal periods: Irritability; Increased sense perception (smell, taste); Poor frustration tolerance; many symptoms of depression; and Impulsivity/Obsessive, being pre-ictal symptoms; often resulting in many of the relationships difficulties. The more problems occurring, the lower will be the SWB in men with AOES experiencing CPS.
Conclusion: Education and counselling for both the male with AOES and those closest to him on recognising the symptoms of pre-ictal period is necessary. This will ensure that misunderstand does not occur, reducing the impact on SWB in males while in the family environment.

p247

Evaluation of cognitive function in untreated patients with generalized tonic-clonic seizure in rural China

DING D[1], HONG Z[1], WANG W[2], WU J[2], DE BOER HM[3], SANDER JW[4]
[1]Institute of Neurology, Fudan University, Shanghai, China, [2]Beijing Institute of Neurosurgery, Beijing, China, [3]Stichting Epilepsie Instellingen Nederland (SEIN), Heemstede, Netherlands, [4]Stichting Epilepsie Instellingen Nederland (SEIN), UCL Institute of Neurology, Heemstede, London, Netherlands

Purpose: To evaluate the cognitive function in untreated patients with generalized tonic-clonic seizure (GTCS) in rural China.
Method: Study subjects were enrolled from the rural area of Anhui, Jilin, Hebei, Sichuan and Ningxia provinces in China. 144 patients with GTCS and 144 normal controls (matched by age, sex, education, and residence) were evaluated with a battery of neuropsychologic tests, which comprises Mini-Mental State Examination(MMSE), Hamilton Depression Rating Scale for Depression (HAMD), Digit Span Test (DST), Verbal Fluency Test (VFT), Auditory Verbal Learning Test (AVLT) and Digit Cancellation Test (DCT).
Results: The mean score of MMSE, AVLT, DST, VFT, and DCT in case group was significantly lower than that in control group ($P < 0.05$). The mean score of HAMD in case group was significantly higher than that in control group ($P < 0.05$). Multivariate analysis indicated that, age and seizure frequency were associated with DST score; unstandardized treatment with anti-epileptic drug was associated with AVLT, DST, and VFT score; statues epilepticus was associated with DCT score ($P < 0.05$).

POSTER ABSTRACTS

Conclusion: The cognitive function of untreated patients with GTCS in China are widely impaired. Patients with GTCS performed worse of general cognitive deficits, such as verbal memory, episodic memory, verbal learning capacity, visual spatial memory, attention, and calculative ability. Unstandardized treatment with anti-epileptic drug is the main impact factor of cognitive function impairment.

p248
Memory functions of epilepsy patients with temporal lesions before and after epilepsy surgery
KWOK FY, CHAK WK
Tuen Mun Hospital, New Territories, Hong Kong

Purpose: To compare the memory functions of patients with temporal lesions before and after epilepsy surgery.
Method: A total of 12 patients age ranging from 16 to 54, six with left-sided temporal lesion, and six with right-sided lesion, were assessed on their intellectual functioning, verbal and visual memories. Patients were assessed on standardized neuropsychological tests before and one-year after surgery.
Results: Patients with left temporal lesions had better visual memory in pre-surgical assessment. No significant decline was found in both their verbal and visual memories after surgery. Five patients with right-sided temporal lesions had better or similar verbal memory when comparing with their visual memory, while one had worse verbal memory in pre-surgical assessment. No significant decline was found in both their verbal and visual memories after surgery. Among the 12 patients, six became seizure free and six had seizure reduction. One of them became medication free and six had reduction in medication. Five patients had to maintain same medication dosage.
Conclusion: After epilepsy surgery, no significant decline was found in memory functions of patients with either left-sided or right-sided temporal lesions.

Social issues

p249
Knowledge, attitude and practices on epilepsy among care-takers of Myanmar children with epilepsy
MIN AYE AM, LINN K
Yangon Children Hospital, Yangon, Myanmar

Purpose: This study aims to know knowledge, attitude and practices on epilepsy among care-takers of Myanmar children with epilepsy. This is the first study done in Myanmar care-takers.
Method: A cross sectional study design was used. Face-to-face questionnaire-based interview of 103 care-takers were done in epilepsy out-patient clinic of Yangon Children hospital from March to December, 2008.
Results: Sixty percent believed epilepsy as a brain disease and 26.2% thought due to evil spirits. Regarding treatment options, though 97% believed the western medicine, about one third of those believed folk medicine and spiritual healing. Epilepsy was considered: familial (42.7%), curable (69.9%) and contagious (only 1.9%). Positive attitudes were seen in the areas of disclosure of having epilepsy (70.9%), safety during play (82.5%), and schooling (65%). Negative attitudes were noticed in the areas of marriage and pregnancy (55.3%), and employment opportunities (59.2%). Although 62.2% took treatment with doctors before coming to hospital, folk medicine (24.3%) and spiritual healing (17.5%) were also tried. The majority had regular follow up (85.4%) and gave anti-epileptic drugs as instructed (74.6%). About half of the children were attending either mainstream or special school. Some wrong beliefs like evil spirits, folk medicine and spiritual healing were significantly related to negative attitudes and wrong practices.

POSTER ABSTRACTS

Conclusion: Care-takers of people with epilepsy generally possessed high levels of knowledge and practice about many aspects of epilepsy. However, some wrong beliefs, negative attitudes and practices were still prevalent and it demonstrates the need for educational programmes aimed at demystifying epilepsy.

p250

Knowledge, attitudes and practice toward epilepsy among epilepsy patients in Ladakh, a rural, mountaineous region of Himalayas, India

SRIVASTAVA PV[1], MOHD G[2], THINLEY T[2], NORBOO T[2]
[1]All India Institute of Medical Sciences, New Delhi, India, [2]District Hospital, Leh, India

Purpose: We conducted a survey to assess knowledge, attitudes and practice toward epilepsy in a hospital based study in Ladakh, a rural, mountaineous region of Himalayas, Kashmir, India.
Methods: We utilized an established knowledge-attitudes-practice questionnaire. This was administered by trained medical social workers and nurses to patients diagnosed with epilepsy over a period between 2003 to 2009, attending the district hospital of Leh, Ladakh. Ladakh is the highest plateau of Kashmir, 9800 ft. above sea level. It spans the Himalayan and Karakoram mountain ranges and upper Indus River Valley.
Results: Out of 550 patients, only 35% had heard about epilepsy; 26.7% had known someone with seizures. 35% believed epilepsy to be a brain disease while 55% thought it is due to "visitation from evil spirits"; the rest could not comment. 80% felt it is a stigma and would like to conceal it from others. 79% had taken treatment from "Amchi", an indigenous alternative system of medication, before allopathy. A simultaneous survey amongst attendees for other diseases to the outpatient showed objection to their children interacting with known epileptics (45%); to their family members marrying an epileptic (75%); to providing employment (46%). The educated few (26.9%) had more familiarity with epilepsy, compliance to treatment, and less negative attitude toward epilepsy.
Conclusions: Knowledge amongst rural population of Ladakh, is poor even compared to rest of India. Illiteracy, poverty and lack of medical facilities contributed to ignorance and rampant negative attitude towards epileptic patients hampering their treatment avenues.

p251

Parental cognitive appraisals and coping behaviours following child´s epilepsy diagnosis: a qualitative study

NGUYEN S[1], KETTLER L[1], PERTINI M[2], FRANCIS M[3]
[1]University of Adelaide, Adelaide, Australia, [2]Women's and Children's Hospital, Adelaide, Australia, [3]Epilepsy Centre, Adelaide, Australia

Purpose: In accordance with the Risk-Resistance Model (Wallander & Varni, 1992), stress-processing as manifested in cognitive appraisals and coping behaviours has a fundamental role in defining parental adjustment to paediatric illness. Using qualitative methods, this study aimed to investigate the cognitions and coping behaviours used by parents following an epilepsy diagnosis.
Method: Twenty-two parents participated and interview data was analysed using theory-driven thematic analysis (qualitative method).
Results: Ten main themes emerged that encompassed parents' overall experience of the epilepsy diagnosis but the findings presented in this article focus on the cognitive and behavioural strategies that regulated psychological symptomatology. Effective cognitive appraisals included maintaining a positive outlook (positive comparisons, hope), re-structuring expectations (normalizing, living one day at a time, ceding control) and finding meaning from their experiences (personal growth, assisting others). Meanwhile problem-solving, emotional ventilation, time to self and speaking with parents in similar situations were behaviours that buffered against carer strain.

POSTER ABSTRACTS

Conclusion: These coping strategies have been identified as sources of resilience and therefore provide a guide for improving parent outcomes in the context of paediatric illness. Implications for clinical services and suggestions for future research are discussed in this paper.

p252

Adventure therapy: 'a life changing experience'

CLARKE L[1], TODD L[1], MARCHINTON T[2]
[1]Epilepsy Action Australia, Sydney, Australia, [2]Purple Soup Pty Ltd, Victoria, Australia

Purpose: Epilepsy Action Australia (EAA) is committed to providing programs that respond to the emotional and psychosocial needs of people with epilepsy. Clients indicate that living with epilepsy can be isolating and the diagnosis can be associated with anxiety and depression. In partnering with Adventure Therapy Camp specialists 'Purple Soup', EAA provides camps which are designed to build confidence, self esteem, trust and courage as well as teach life skills and provide opportunities for meeting others with epilepsy.

Method:

- Using a standard evaluation form, baseline measures are collected prior to the Adventure Therapy experience and compared with post experience measures.
- Each camp is evaluated on whether the experience has assisted to reduce feelings of isolation; the participants' confidence in managing challenging situations; knowledge of seizures and epilepsy; ongoing peer support from people with epilepsy.
- The shift in rating scale pre and post camp is analysed.
- Free text comments are also collected and themed for analysis.

Results:

- In the past year, 128 people with epilepsy have participated in the program.
- Evaluations show a positive shift on all measures.
- Free text comments indicate that some participants consider Adventure Therapy a life changing experience.
- This presentation will demonstrate how the camp is based on Therapeutic Recreation models; detail the activities and their benefits and provide outcomes of evaluations.

Conclusion: The evaluation of Adventure Therapy indicates it is an effective strategy for decreasing isolation, enhancing self esteem and building confidence.

p253

North indian women with epilepsy: a survey of medical and social concerns

KAUSHAL S, TANDON S, TANEJA A, SINGH G
Dayanand Medical College and Hospital, Ludhiana, India

Purpose: Women with epilepsy (WWE) are a subgroup of patient population who are predisposed to special situations due to the disease and its treatment. These situations vary depending on the age, education, social background and even marital status of WWE. The study was designed to determine the medical and social concerns among WWE of North Indian origin

Method: The survey was conducted among the WWE attending the Epilepsy Clinic at a Medical School Hospital over a period of one year. Structured questionnaires were developed separately for married and unmarried WWE to identify their concerns and were administered to respective groups.

POSTER ABSTRACTS

Results: Fifty married (median age: 30 years) and 64 unmarried (median age: 19 years) consenting women were enrolled. Among these only 3% were illiterate. Employment rate was 14 % for unmarried and 4% for married women. Marriage negotiations took place in 11 (17.2%) unmarried women. The disclosure of epilepsy was done in 6, resulting in 100 % (6/6) breakdown of negotiations. Marital relations were disturbed in 60% (6/10) women, who had concealed diagnosis before marriage. 38 % (19/50) married women had reported use of some form of contraception (including 10% with history of use of COCs). Preconceptual folate supplementation was taken by 59.6% of those WWE who were on AEDs during pregnancy.
Conclusion: A major concern for unmarried was impact of disclosure or concealment of epilepsy during marriage negotiations on marital relations. While married were more concerned about effect of epilepsy and antiepileptic drugs on fertility, pregnancy and children.

p254
Evaluating a specialist epilepsy bereavement service
PRESTON J, HANNA J
Epilepsy Bereaved, Oxford, United Kingdom

Purpose: To evaluate an epilepsy bereavement service.
Method: Analysis of intake data from people bereaved through epilepsy in the UK during 2009 (Access DataBase); internal surveys (2006) and two external evaluations (2006 and 2010).
Results: In 2009 Epilepsy Bereaved responded to 93 newly bereaved in a year (80% from the internet); and 1010 calls from a total of 1047 bereaved families. 12% of new contacts had very complex information needs. The service is run by two bereavement counsellors supported by a panel of SUDEP experts and offers information resources on epilepsy mortality; weekend and day events; a memorial service; a magazine; a website in addition to a telephone support line. A bereavement counselling service is being piloted during 2010.
During 2009 15% of the families choose to be supported to be active in the work of the charity through awareness campaigns and fundraising. Internal surveys and external evaluation (2006/2007) found 97% reporting participation as helping to channel their grief in a positive way as well as developing strong peer group support. Surveys of health professionals engaging with the bereaved have positively endorsed the participation of the bereaved as increasing their knowledge. During 2010 the service was honoured by the award by the Queen of an OBE to the Director of Epilepsy Bereaved.
Conclusion: Research on the impact of epilepsy mortality is urgently needed, but data from the UK supports the demand for and value of a specialist epilepsy bereavement service.

Surgery

p255
Psychiatric symptoms changes after corticoamygdalohippocampectomy in medial temporal lobe epilepsy patients through Symptom Checklist-90-Revised
GUANGMING Z, WENJING Z, HUANCONG Z, FUQUAN Z, YAN Z, ZHAOHUI S
Tsinghua University, Beijing, China

Purpose: Corticoamygdalohippocampectomy (CAH, anterior temporal lobe resection plus amygdalohippocampectomy) is common in epilepsy surgery. Pre- and postoperative psychiatric disorders occurred sometimes in refractory medial TLE (temporal lobe epilepsy) patients. We want to know if CAH has an affirmative effect on medial TLE patient's psychiatric symptom through a quantitative method.

POSTER ABSTRACTS

Method: Sixty-two medial TLE patients who had CAH accomplished SCL-90-R (Symptom Check-list 90 Revised) questionnaires thrice (presurgical, postsurgical one and two years). Average GSI (global severity index) scores in SCL-90-R were calculated and statistically analyzed.
Results: There was no statistical difference in the presurgical average GSI scores between Engel I and Engel II-IV subgroup. Postoperative 1 and 2 years average GSI scores of Engel II-IV subgroup were both statistically higher than those of Engel I subgroup. There were no statistical differences between other subgroups in different time. Postsurgical 1 and 2 years average GSI scores of the whole group and Engel I subgroup were statistically lower than those of presurgery. Postoperative 2 years average GSI scores of the whole group and Engel I subgroup were statistically lower than those of postsurgical 1 year. For Engel II-IV subgroup there were no statistical differences among the average GSI scores in different time.
Conclusion: CAH could improve the psychiatric symptoms of TLE patients as assessed by the SCL-90-R. This improvement was related to the therapeutic effect and was not related to gender, lateralization, and MRI abnormality.

p256
Pre-surgical evaluation and surgical treatment of epilepsies in adults
SITNIKOV AR, GRIGORYAN Y, MISHNYAKOVA L
Federal Centre of Neurosurgery, Moscow, Russian Federation

Purpose: to analyse the results of surgical treatment of cortical dysgenesia - associated epilepsies.
Method: 340 adult patients with epilepsy underwent the standard evaluation including MRI, video-EEG and fMRI. 12 patients with various cortical dysgenesias were operated. The topectomy was done in all cases after pre-surgical evaluation which obligatory included an MRI - scan (T1, T2, FLAIR axial, sagittal and coronal series), fMRI with navigation, EEG - video and invasive corticography for every patient.
Results: The mean age of patients was 26 years (between 17 and 33 years). There were 7 females and 5 males. The MRI scan showed FCD in 7 patients, pachygyria in one patient and gray matter heterotopia in one patient. All patients underwent an invasive corticography before the surgical resection to identify the epileptogenic region which was resected afterwards using the intra-operative navigation and corticography before and after topectomy. In five cases awake-craniotomy was performed for patient with dysgenesia located in eloquent brain areas. There was no neurological complication. The morphological examination confirmed FCD I type in 4 cases, FCD IIa in 3 patients and IIb -in 3 patients, focal pachygyria in one case and gray matter heterotopia in one case. The three-years follow - up showed a complete seizure-free outcome in 6 patients (Engel Ia), 5 patients achieved Engel II (rare partial seizures) and one patient has Engel III outcome.
Conclusion: dysgenesia - associated epilepsies in adult patients can be successfully treated with topectomy without neurological complication.

p257
Para-insular hemispherotomy: a new modification of peri-insular technique
CHANDRA S, GARG A, GAIKWAD S, BAL C, SARKAR C, TRIPATHI M
All India Institute of Medical Sciences, New Delhi, India

Purpose: To validate a new modification of hemispherotomy: pari-insular hemispherotomy (PIH) performed in patients with intractable epilepsies

POSTER ABSTRACTS

Methods: A prospective study 2004-March 2010: 22 cases of PIH from a total of 608 cases operated for intractable epilepsy, were selected.All the cases operated for intractable epilepsy underwent a complete epilepsy surgery workup. Including EEG, VEEG, MRI- epilepsy protocol, SPECT, SISCOS, PET. Age range 4-19 years (mean 5.2 years), 16 males. The seizure frequency ranged from 5-200 episodes per day; five were in status; three in epilepsia partialis continua. The pathologies included Rasmussen's, hemimegelencephaly (unilateral hemispheric enlargement with severe cortical and subcortical changes), hemispheric cortical dysplasia, post-stroke, post-traumatic encephalomalacia and encephalopathy of unknown etiology. Neuronavigation was used in seven cases. The technique of PIH was used, where the hemispheric disconnection was performed via fronto-temporal route rather than through the sylvian fissure
Results: Class I outcome [Engel's] was seen in 21 cases and Class II in one assessed at 32-178 weeks of follow-up. Cognitive profile improved in all patients and 11 cases returned back to school. Post op MRI were performed in all cases to ensure complete disconnection.
Conclusion: As compared to other techniques (peri insular and vertical) by the same author, PIH took lesser operative time, was more simpler to perform especially in hemimegalencephalies and required a lesser learning curve with good clinical and seizure outcomes.

p258

Paediatric epilepsy surgery program in New Territories West Cluster, HKSAR: audit of seizure outcome from a tertiary referral center

CHAK WK[1], FONG D[2], YAM KY[2], LAI TT[3], WONG YC[4], KWOK F[5], YIP R[6]
[1]Department of Paediatrics and Adolescent Medicine, Tuen Mun Hospital, Hong Kong, Hong Kong, [2]Department of Neurosurgery, Tuen Mun Hospital, Hong Kong, Hong Kong, [3]Department of Psychiatry, Castle Peak Hospital, Hong Kong, Hong Kong, [4]Department of Radiology, Tuen Mun Hospital, Hong Kong, Hong Kong, [5]Department of Psycho-Behavoural Unit , Tuen Mun Hospital, Hong Kong, Hong Kong, [6]Department of Occupational Therapy, Tuen Mun Hospital Genetcs, Yok, Hong Kong, Hong Kong

Purpose: To report the seizure outcome of Paediatric Epilepsy Surgery.
Method: Patients with medical refractory epilepsies have undergone pre-surgical evaluation including: long-term video-EEG; MRI; neuro-psychological assessment. SPECT; PET scan, Wada test, intra-operative electro-corticography and functional mapping were performed in selective cases. One patient underwent implantation of subdural and depth electrodes, followed by extra-operative intracranial EEG monitoring and functional mapping. Seizure outcome was evaluated according to Engel's classification.
Results: Total 35 children and adolescent underwent surgery from 1998 to 2009. Their age at operation ranged from 9 months - 19 years. Etiology was including: Malformation of cortical development 8; mesial temporal sclerosis 5; post-viral encephalitic epilepsy 2; benign temporal tumour or cyst 5; neurofibroma 1; cavernous hemangioma 1; Arteriovenous Malformation 1; hypothalamic hamartoma 2; tuberous sclerosis with hemimegalencephaly 1; porencephalic cyst 1; 8 children with refractory generalized epilepsy and drop attack underwent corpus callosotomy. 16 patients underwent temporal lobe surgery; 6 patients underwent extra-temporal lesionectomy; 2 patients underwent resection of hypothalamic hamartoma; 1 patient underwent functional hemispherectomy; 1 patient underwent TPO disconnection; 1 patient underwent resection of residual AVM and scarring. There was no mortality, and one patient had transient right eye ptosis post-opertaively. The mean duration of follow up was 40 months (range 6 month - 10 year). 42% of patients were Engel class 1, 29% of the patient were Engel class 2-3, 29% of the patient were Engel class 4.
Conclusion: Epilepsy surgery is a safe and effective treatment for selected paediatric and adolescent patient with medical refractory epilepsy.

POSTER ABSTRACTS

p259

One stage surgery of tuberous sclerosis patients with multiple epileptic foci

LIU S-Y[1], AN N[1], YANG H[1], YANG M-H[1], HOU Z[1], LIAO W[1], ZHANG Q[1], CAI F-C[2]
[1]Department of Neurosurgery, Xinqiao Hospital, Third Military Medical University, Chongqing, China, [2]Children's Hospital, Chongqing Medical University, Chongqing, China

Purpose: To investigate the surgical procedures and outcomes of tuberous sclerosis patients with multiple epileptic foci(MEF).
Method: Surgery of 21 tuberous sclerosis patients with MEF between 2002-2008 were reviewed. The MEF of 15 patients limited in one hemisphere. 6 patients had hemispheric dominant MEF but had contralateral discharge. Resection of epileptogenic lobe or tuber, and(or) multiple subpial transection(MST) was conducted. Anterior corpus callosotomy were chosen to deal with the hemispheric MEF patients with contralateral discharges.
Results: At follow-up, on average 3.2 years after surgery, thirteen of 21 patients (61.9%) had an Engel Class I outcome after surgery and an additional foure patients (19.0%) had rare seizure (Engel ClassIII), and three patient had a decrease in seizure frequency (Engel Class III). Mean intelligence quotient (IQ) improved from 51.7 to 59.6. Temporary complications were observed in five patients. One patient developed hydrocephalus.There was no death.
Conclusion: One stage surgery is suitable for the tuberous sclerosis patients with hemispheric MEF or hemispheric dominant MEF.

p260

Predictors and outcomes following surgery in childhood temporal lobe epilepsy

JOHNSON AM[1,2], CONNOLLY AM[1,2], SUGO E[1], LAWSON JA[1,2], MALONE SA[3], BOYLE RC[1,2], BYE AME[1,2]
[1]Sydney Children's Hospital, Randwick, Sydney, Australia, [2]University of New South Wales, Sydney, Australia, [3]Royal Children's Hospital, Brisbane, Australia

Purpose: Following surgery in childhood Temporal Lobe Epilepsy (TLE), outcome relates to seizure freedom, behaviour and neurodevelopmental impact. Our primary aim was to evaluate postoperative outcome and define predictors, in a series of children with TLE.
Method: We conducted a retrospective analysis of children with medically resistant TLE who underwent surgery (2002-2008) at Sydney Children's Hospital. Patient demography, seizure semiology, neurophysiology, imaging, neuropsychology and pathology were re-examined and current status determined.
Results: The cohort was 28 children (15 boys) aged 2-18 years, median age 10, at surgery. Median age of epilepsy onset was 4.5 years. Hippocampal sclerosis (HS) (n=13) and low grade tumour (n=13) were the most common lesions. Twelve patients had dual pathology with dysplasia or microdysgenesis in addition to their HS or tumour. Complex febrile convulsions were only reported with HS (54%). Nineteen patients (68%) had Engel class I outcome at follow-up (median four years). Four patients had an Engel class III/IV outcome, all of whom had dual pathology. HS and tumour groups were equally represented in patients with class III/IV outcome. Eight patients underwent multiple operations, including the previous four patients with dual pathology who did not improve. The remaining patients had single pathology, and all improved to Engel class I/II. Patients with pre-morbid behavioural/psychiatric difficulties were more likely to have post-operative problems (p=0.003).
Conclusion: Irrespective of primary pathology, 68% had class I outcome. The four patients with class III/IV outcome had dual pathology. Pre-operative behavioural and psychiatric problems were associated with post-operative disturbances.

POSTER ABSTRACTS

p261

Staged total callosotomy for Lennox-Gastaut syndrome by surgical section or gamma-knife: two case reports

KO T·S[1], LEE E·H[1], JEONG M·H[1], YUM M·S[1], HONG S·H[2], LEE J·K[2]

[1]Dept of Pediatrics, Asan Medical Center, University of Ulsan College of Medicine, Seoul, Korea, Republic of, [2]Dept of Neurosurgery, Asan Medical Center, University of Ulsan College of Medicine, Seoul, Korea, Republic of

Purpose: We report two cases with Lennox·Gastaut syndrome(LGS) who underwent staged total callosotomy and showed favorable outcomes.
Case 1: A 6·year old boy having myoclonic seizures from the age of 8 months was diagnosed as LGS when he was 27 months old. Brain MRI was normal and EEG revealed generalized slow spike·and·wave discharges. Various antiepileptic drugs(AEDs) and ketogenic diet failed to control his multiple types of seizures. At the age of 36 months, he underwent corpus callosotomy and achieved an immediate, post·operative seizure free state. After three months, however, various seizures relapsed and were refractory to additional vagus nerve stimulation. We performed a second operation, total callosotomy. Finally, he has obtained seizure·free state with normalization of EEG for 10 months after the procedure.
Case 2: A 10·year old girl was referred to our hospital because of intractable epilepsy for 6 years. Multiple types of seizures including drop attacks and generalized slow spike·and·wave discharges were consistent with LGS. Brain MRI was normal and additional AED therapy was unsuccessful. She underwent anterior 2/3 corpus callosotomy at 12 years of age. For the first few months after surgery, the frequency of her seizures lessened, but then returned to the baseline level. Total callosotomy using gamma·knife was performed and her drop attacks have been away after the procedure.
Conclusion: We suggest that staged total callosotomy could be considered as a second step when the first callosotmy fails to reduce seizures in patient with LGS. In patient with surgical risk, gamma·knife can be another option.

p262

Epilepsy surgery in tuberous sclerosis complex: emphasis on surgical candidate and neuropsychology

LIANG S[1], LI A[1], JIANG H[2], DING C[3]

[1]Department of Neurosurgery, First Affiliated Hospital of General Hospital of PLA, Beijing, China, [2]Department of Psychology, First Affiliated Hospital of General Hospital of PLA, Beijing, China, [3]Department of Neurology, Tiantan Hospital, Beijing, China

Purpose: To discuss neuropsychological outcome and candidate of epilepsy surgery for tuberous sclerosis complex (TSC).
Methods: To retrospectively analyze clinical data of 25 patients with TSC and epilepsy who underwent epilepsy surgery from 2001 to 2007. Seizure reduction was analyzed at 1 year (1FU), 2 years (2FU) and 5 years (5 FU) follow·up visit after surgery, and outcomes of intelligence quotient (IQ) and quality of life (QOL) were evaluated at 2FU.
Results: Resective procedures included 14 tuber resections, 9 lobectomies and 2 tuber resections & lobectomies. Corpus callosotomies (CCTs) were performed as adjunctive approach in 8 cases with low·IQ and behavior problems. The percentages of seizure·free were 72% at 1FU, 60% at 2FU and 54.5% at 5FU, and the factors used for predicting seizure freedom included the course of seizure and patients' age at surgery. Significant improvement was found in performance IQ in patients with preoperative low·IQ or CCT. Significant improvements of mean QOL score were observed in all patients, and also patients with postoperative seizure free, preoperative low·IQ and CCT.

POSTER ABSTRACTS

Conclusion: Surgical candidates for TSC with epilepsy should be patients with indentified epileptogenic tubers, not excluding those with low-IQ and multiple epileptogenic tubers. Satisfactory seizure control was often achieved with early operation while improved QOL was frequently seen in postoperative seizure free patients. CCT could be performed as an adjunctive approach to resective operation for TSC patients with epilepsy and low-IQ, and rendered improvement of performance IQ and QOL.

p263

Supratentorial pilocyitic astrocytoma-associated epileptic intractability related with tumor location and morphology

NISHIMOTO T[1], IIDA K[1], HASHIZUME A[1], KAGAWA K[1], HANAYA R[2], SUGIYAMA K[1], ARITA K[2], KURISU K[1]

[1]Department of Neurosurgery, Hiroshima University, Hiroshima, Japan, [2]Department of Neurosurgery, Kagoshima University, Kagoshima, Japan

Purpose: Supratentorial pilocytic astrocytoma (PA) may cause intractable epilepsy; however, findings on epileptic intractability associated with PA location and morphology have been limited.
Method: The relationships between tumor location, MRI-based morphology, epileptic intractability, and seizure outcome were examined in 12 PA patients (age range: 1-42; mean: 20.9 years) pathologically MRI-diagnosed with either cystic tumor (CT) (group CT; n=7) and or solid tumor (ST) (group ST; n=5).
Results: PA locations of group CT included the frontal lobe (n=2), temporal lobe (n=1), parietal lobe (n=1), and basal ganglia/third ventricle (n=3). Four of 7 patients (group CT) had no history of pre-operative seizures, while the remaining 3 patients encountered symptomatic epilepsy (well managed with pre- and postoperative medications). As for PA locations in the temporal lobe (n=4) and optic tract (n=1) of group ST, the former (n=4) experienced preoperative intractable epilepsy (the latter established well-controlled epilepsy with antiepileptics). All 7 patients in group CT underwent lesionectomy. While 4 patients with temporal lobe PA in group ST underwent anterior temporal lobectomy/lesionectomy, the remaining 1 patient with optic tract PA underwent lesionectomy. Seizure outcome in the 4 patients with intractable epilepsy (group ST) was categorized as Engel class I during the follow-up period of 16-36 months.
Conclusion: Epilepsy surgery involving peritumoral cortical resection of the epileptogenic zone is essential in patients with solid PA in the temporal lobe; a site which is closely associated with epileptic intractability. Lesionectomy is adequate for establishing control of symptomatic epilepsy in patients with CT in the extra-temporal lobe.

p264

Intracranial electroencephalographic findings and histopathological features in epilepsy patients with dysembryoplastic neuroepithelial tumor (DNT) and DNT-like lesions

KAGAWA K[1], IIDA K[1], HASHIZUME A[1], NISHIMOTO T[1], KIURA Y[1], HANAYA R[2], SUGIYAMA K[1], ARITA K[2], KURISU K[1]

[1]Department of Neurosurgery, Hiroshima University, Hiroshima, Japan, [2]Department of Neurosurgery, Kagoshima University, Kagoshima, Japan

Purpose: Based on intracranial video-electroencephalographic (IVEEG) findings, histopathological features, and seizure outcome, we elucidated the epileptogenic zone in patients with DNT and DNT-like lesions (DNT-L).
Methods: Five patients pathologically diagnosed with DNT (n=4) and DNT-L (n=1) underwent IVEEG-monitoring to identify ictal onset zone (IOZ) and irritative zone (IZ). We examined the correlations of localization of IOZ and IZ with the MRI-visible lesion (MR-L) and their histopathological features.

POSTER ABSTRACTS

Results: IVEEG located the IOZ at the margin of MR-L (all 4 DNT patients) and on the MR-L (1 DNT-L patient) with the IZ extended to the surrounding cortex in a fashion more extensive than the IOZ. Lesionectomy of MR-L performed in 2 DNT patients (group L) and additional cortical resection (including the IOZ and IZ) in the remaining 2 DNT and 1 DNT-L patients (group L+CR) yielded postoperative seizure-free outcome (Engel' class I) in group L+CR, and classes II and III in one each of group L. Histopathologically, specific glioneuronal elements (SGNE) were distributed inside the MR-L of 4 DNT patients with oligodendroglia-like cells (OLC) extended continuously beyond the MR-L with/without proliferation of astrocytes or cortical dysplasia (CD) in 2 DNT patients. The CD component intermixed with OLC was predominantly manifested in the DNT-L patient.
Conclusions: The extent of coexisting pathological features with DNT/DNT-L varied among the patients. These features may be associated with the epileptogenesis and IVEEG-based cortical resection for seizure control.

p265
Epilepsy surgery In Indonesia: how to start the program in limited situation
MUTTAQIN Z
Faculty of Medicine, Diponegoro University, Semarang, Indonesia

Purpose: With 0.5% prevalence, Indonesia has 1.5 million epileptic and 250.000 potential surgical candidates (ES). Excellent results of ES, and absence of proper insurance system makes ES important and should be developed, especially for TLE.
Method: To evaluate presurgical evaluation, cases were divided into first 5 years (56 cases) and recent 5 years (182 cases). To evaluate surgical results, those over 36 months follow-up were included (106 cases) and grouped into those operated before or after 25 Y-old, and into those operated when the length of epilepsy is less or more than10 years.
Results: For the first five years MRI is decisive in 54 of 56 TLE cases. For the recent five years, MRI is decisive in 91 out of 156 TLE cases. Ictal EEG were performed in 46, subdural EEG in 10, PET in 7, 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: Role of MRI is decreasing as it was 96.4% (the beginning) to 58.34% for the recent five years. This means that we worked on more difficult cases. SF rate was significantly higher for those operated earlier. This means that surgery should be offered earlier for intractable TLE patients.

p266
3 cases of disconnection syndrome after staged total callosotomy for intractable epilepsy
TODA K, BABA H
Department of Neurosurgery, Nagasaki Medical Center, Omura, Japan

Purpose: Disconnection syndrome may complicate the clinical outcome of some patients over 10 years old undergoing total corpus callosotomy. Although the more extensive callosal section appeared to yield the better surgical outcomes, the extent of callosal section has long been the subject of debate among epilepsy surgeons. We demonstrate 3 cases of two-staged total callosotomy over 15 years old for the treatment of severe drop attacks.
Method: All 3 patients showed intractable generalized epilepsy including drop attack. The age of seizure onset was between 1 and 8 years old. Mean age at anterior two thirds corpus callosotomy was 20.7 years old, but the procedure failed to obtain satisfactory seizure control. Additional posterior callosal section was performed for drop attack (DA) between 15 and 33 years old.

POSTER ABSTRACTS

Results: After two-staged callosotomy, complete cessation of DA was obtained in all patients. However all patients showed various types of disconnection syndrome; tactile dysnomia in 3 patients, non-dominant hand agraphia in 3, and alien hand syndrome in 1, those were not appeared after anterior corpus callosotomy. Fortunately, these symptoms gradually improved and did not affect activities of their daily life.
Conclusion: Total callosotomy performed before puberty is not followed by permanent deficits of disconnection syndromes. Two-staged total callosotomy for patients over 15 years old may yield transient deficits of disconnection syndrome. But patients will sometimes not be aware of those symptoms until it is brought to their attention and be satisfied by good seizure control.

p267
Sectioning of the corpus callosum for refractory epilepsy
ZHOU J, LUAN G
Capital Medical University Sanbo Brain Institute, Beijing, China

Purpose: To evaluate the efficacy and safety of the anterior corpus callosotomy for the generalized seizures who cannot underwent a section procedure.
Method: Analyze date of the refractory seizures who underwent an anterior corpus callosotomy (n=131) from 2004 to 2009.
Results: It is difficult to compare the outcome of different seizure types. For all seizures taken together, the rate of seizure free is 68% and 87% achieved satisfactory (90% or greater reduction of seizures). The quality of life of 92% patients was gains associated with reduced seizure frequency. The rate of complication is 2.7%.
Conclusion: Corpus callosotomy is a safe and effective palliative procedure for medically refractory epilepsy when no respectable lesion is indentified. This is especially true when drop attacks or generalized tonic seizures are the main types. Most neurological complications after callosotomy are temporary or clinically unapparent.

p268
Dense array EEG for the tuberous sclerosis complex with epileptic seizure and intraventricle tumor
TOYODA G, FUJIMOTO A, YAMAZOE T, ENOKI H, OKANISHI T, YOKOTA T, YAMAZAKI M, YAMAMOTO T
Comprehensive Epilepsy Center, Seirei Hamamatsu General Hospital, Hamamatsu, Japan

Purpose: Tuberous Sclerosis complex (TSC) is classically defined with the trias of epilepsy seizure, mental retardation and angiofibloma. There is also sub-category of systemic hamartoma in addition to the trias. The death is mostly caused by the renal failure, central nervous system lesion including brain tumor and followed by heart failure. The epilepsy seizure also highly affects to the high mortality rate and has started being considered surgical treatment for brain tumor and epilepsy seizure.
Method: The patient is 9-year-old right handed boy with TSC. He exhibited gelastic seizure, restlessness seizure and choking spell. We captured the gelastic seizure and restlessness seizure during conventional long term video EEG monitoring. However the seizure onsets of both types of seizure were unclear. Enhanced MRI showed multiple cortical tubers and heterogeneously enhanced intraventricle tumor, considered to be giant cell astrocytoma. We used 256ch-dense array net type EEG(d-EEG) for this patient.
Results: We captured the gelastic and restlessness seizures during the d-EEG. The d-EEG date showed right frontal and temporal onsets independently under the visual analysis. The analysis of Local autoregressive analysis (LAURA) inverse solution for the interictal epileptiform discharges also estimated the focus over the right frontal and temporal regions on the MNI Typical Brain

POSTER ABSTRACTS

Conclusion: The d-EEG could detect the focus which the conventional EEG could not. The d-EEG has better spacial resolution. Even patients with mental retardation and uncooperative patients are able to be evaluated by d-EEG for the focus diagnosis.

p269
Epilepsy surgery for extratemporal epilepsy, reasons for surgical failure
WONG TH[1], WONG CH[1], BIRKETT J[1], MCANALLY L[1], DEXTER M[1], SOMERVILLE E[2], BLEASEL A[1]
[1]Westmead Hospital, Westmead, Australia, [2]Prince of Wales Hospital, Randwick, Australia

Purpose: To determine the factors predictive of outcome in extratemporal epilepsy surgery and identify the reasons for surgical failure.
Method: A retrospective analysis of extratemporal epilepsy surgery cases between 1998 and 2009 found 71 extratemporal surgical patients; 43/71 (60.6%) lesional and 28/71 (39.4%) non-lesional. We examined concordance between interictal and ictal EEG, SPECT and PET, and the epileptogenic zone.
Results: Seizure freedom (Engel 1) was achieved in 28/43 (65.1%) lesional cases and 13/28 (46.4%) non-lesional cases. In seizure free lesional cases, concordance with the epileptogenic zone was high; interictal EEG (75%), ictal EEG (57.7%) SPECT (78.9%) PET (77.3%), with ≥ 3 investigations concordant in 46.4%. In non-lesional cases; concordance was also high, interictal EEG (84.6%), ictal EEG (61.5%), with SPECT (76.9%) and PET (53.8%) with ≥ 3 investigations concordant in 69.2%. In the 15 lesional cases with recurrent seizures SPECT was concordant in 41.7% and PET in 50.0%. In 15 non-lesional cases without remission, SPECT was concordant 66.7% but PET in only 28.6%. In the surgical failures fewer patients had concordance of ≥ 3 investigations; lesional 40%, non-lesional 33.3%.
The most common reasons for surgical failure were widespread or residual disease; 17/30 cases. Failure of localisation occurred in 7/30 (6/7 non-lesional) and in 6/30 failure was unexplained. With failure to localise, only 1/7 patients achieved concordance ≥ 3 investigations.
Conclusion: Non-lesional extratemporal epilepsy surgery remains challenging and concordance between multiple non-invasive investigations is associated with a better outcome. In patients with lesions lower rates of concordance between investigations is possible.

Neuroimaging

p270
Methionine positron emission tomography (Met-PET) in symptomatic epilepsy
OTSUBO T , FUJITA S , HANAYA R , SUGATA S , ARITA K , YAMASAKI I , YATSUSHIRO K , NAKAMURA K , FUJIMOTO T
Fujimoto Hayasuzu Hospital, Miyakonojo, Japan, Kagoshima University Graduate School of Medical and Dental Sciences, Kagoshima, Japan

Purpose: Epilepsy patients often have intracranial lesions. Although fluorodeoxyglucose positron emission tomography (FDG-PET) is widely used to detect malignancies of several organs, brain tumors are well known to be difficult to detect by this method. However, Met-PET can detect even these tumors, because methionine is not taken up by the normal brain but is concentrated in malignant brain tissue. We report herein the findings of Met- and FDG-PET in symptomatic epilepsy patients with magnetic resonance imaging lesions.

POSTER ABSTRACTS

Method: Subjects were 9 consecutive preoperative patients with intractable symptomatic epilepsy who had undergone non-invasive studies including Met- and FDG-PET since 2005. They were categorized into 3 groups: brain tumor (BT), cortical dysplasia (CD), and encephalomalacia (EM). Standardized uptake value (SUV) was used as a semi-quantitative index of malignancy. A three-dimensional region of interest was constructed in the lesion and in the contralateral brain as a control. The usefulness of Met- and FDG-PET was determined from SUV ratio of the lesion to the control site (L/C).
Results: L/C was 1 or higher in 2/3 of the BT group, 2/4 of the CD group, and 0/2 of the EM group. In all cases of FDG-PET, L/C was < 1. For Met-PET, SUV was 1.00 to 1.70 and L/C was 0.73 to 1.43.
Conclusion: SUV of Met-PET varied according to pathological findings while that of FDG-PET was low, reflecting the epileptogenesis of lesions. However, SUV of Met-PET was never greater than that seen for malignant brain tumors such as astrocytomas.

p271
EEG-fMRI identifies focal cortical BOLD signal change in some individuals with childhood absence epilepsy
CARNEY PW[1,2], FLANAGAN D[2], SCHEFFER IE[1], BERKOVIC SF[1], JACKSON GD[2,3]
[1]Epilepsy Research Centre, The University of Melbourne, Melbourne, Australia, [2]Brain Research Institute, Melbourne, Australia, [3]University of Melbourne, Melbourne, Australia

Purpose: We have performed EEG with functional MRI (fMRI) on over 40 subjects with an electro-clinical history of absence seizures (AS). Here we describe four subjects with asymmetric or unilateral focal cortical Blood Oxygen Level Dependent (BOLD) signal change.
Methods: Patients were recruited from local EEG departments and electro-clinical information was obtained. EEG was recorded during continuous acquisition of gradient-recalled echo planar images at 3 Tesla. In scanner EEG was reviewed off-line and epileptiform activity (generalised spike and wave) was marked as events of interest as part of an event related analysis using spm8.
Results: All patients had a typical electro-clinical picture for childhood absence epilepsy (CAE) at diagnosis. Two subjects were studied prior to treatment, and became seizure free on medication. The other 2 subjects were refractory to therapy at the time of study, one with generalised convulsions. In all subjects negative BOLD change was seen in the parietal cortex, caudate nucleus and brainstem and positive BOLD in the thalamus; findings which are seen consistently in patients with CAE. These subjects also demonstrated increased BOLD in frontal association cortex, particularly in the region of the middle and superior frontal gyrus. In the new onset cases this was seen bilaterally but asymmetrically, while in the chronic refractory cases this was seen unilaterally only. Findings were reproduced in two subjects.
Conclusion: Our data suggests that focal cortical regions, as detected by functional imaging, may be important in the generation of AS in some subjects with an electro-clinical diagnosis of CAE.

p272
3-Tesla ^{1}H MRS in Chinese temporal lobe epilepsy patients
ZHAO Y[1], LU Q[2]
[1]Xinhua Hospital, Jiaotong University, Shanghai, China, [2]Renji Hospital, Jiaotong University, Shanghai, China

Purpose: To estimate the clinical value of short-echo-time ^{1}H-MR-spectroscopy (^{1}H-MRS) at 3.0 Tesla in TLE with hippocampal sclerosis (HS).
Method: Single voxel ^{1}H-MRS measurements, including NAA/Cr, Cho/Cr and NAA/(Cho+Cr) ratios, from bilateral hippocampus were performed in 40 TLE patients and 30 controls. We compared MRS results with those of MRI. Furthermore, we studied correlation between ipsilateral metabolite indices and the duration of epilepsy, seizure frequency.

POSTER ABSTRACTS

Results: 1. The NAA/Cr and NAA/(Cho+Cr) ratios were abnormally lowered in the hippocampus bilaterally (P< 0.01). The comparison of Cho/Cr among groups showed no statistical significance (P>0.05). 2. The decrease of NAA/Cr and NAA/(Cho+Cr) ratios was less significant in MRI negative hippocampi than that in HS (P< 0.01). ^{1}H-MRS had a sensitivity of 53.85% for lateralization in TLE, whereas bilateral involvement was detected in 29.17%, but MRI accounted for only 45% and 16.67%, respectively. The abnormal rate climbed to 72.5% by using ^{1}H-MRS together with MRI. Moreover, MRS detected HS in 92.31% of intractable TLE, while MRI in 78.57%. 3. No significant correlation was observed between duration of epilepsy and ipsilateral ratios (P>0.05). Nor was a relation found between Cho/Cr and seizure frequency (P>0.05). However, the ipsilateral NAA/Cr and NAA/(Cho+Cr) ratios displayed a negative correlation to seizure frequency (P< 0.01).
Conclusion: ^{1}H-MRS could help lateralizing TLE and plays a role in detecting bilateral metabolite dysfunction. ^{1}H-MRS, valuable in MRI negative TLE patients, improves lateralization of affected hippocampus if combined with MRI. Nevertheless our findings suggest that ^{1}H-MRS abnormality does not reflect disease progression.

p273
[^{18}F]-Flumazenil-PET for the localisation of medically refractory focal epilepsy using without need for arterial blood sampling: a pilot study
VIVASH L[1], GREGOIRE M-C[2], LAU EW[3], WARE RE[3], ROSELT P[3], BINNS D[3], BOUILLERET V[1], KATSIFIS A[2], MYERS DE[1], HICKS RJ[3], O'BRIEN TJ[1]
[1]Department of Medicine, The University of Melbourne, Melbourne, Australia, [2]ANSTO LifeSciences, Sydney, Australia, [3]Centre for Molecular Imaging, Peter MacCallum Cancer Centre, Melbourne, Victoria, Australia

Purpose: [^{11}C]-flumazenil-PET (FMZ-PET) has proven to have high sensitivity for localisation of the epileptogenic zone (EZ) in patients with medically refractory focal epilepsy, with a more restricted region of abnormality that FDG-PET. However, practical aspects of [^{11}C] and requirement for arterial blood sampling have limited its clinical application. We have developed a method utilising a new radioliogand, [^{18}F]-FMZ, that does not require arterial blood sampling. In the current study we have assessed [^{18}F]-FMZ-PET for localisation of the EZ in patients with medically refractory focal epilepsy.
Method: Four subject groups were studied; healthy controls (n=20), patients with well-localised temporal lobe epilepsy (TLE) with hippocampal sclerosis on MRI (n=12), patients with well-localised TLE and normal MRI (n=14), and patients with other focal epilepsies (n=4). A 60min dynamic [^{18}F]-FMZ-PET scan and an FDG-PET scan were acquired. Blinded visual assessment of static images was undertaken. Parametric images of binding potential (BP) were generated and region of interest analysis and statistical parametric mapping (SPM) used to localise the EZ.
Results: Visual assessment of static images has shown [^{18}F]-FMZ-PET to have high specificity (94%), sensitivity (60.9%) and positive predictive value (87.5%) for the EZ, with a more restricted EZ compared to FDG-PET. Initial SPM results also depict a more restricted area of abnormality on FMZ BP images than FDG; regional analysis is ongoing.
Conclusion: Preliminary analyses show that [^{18}F]-FMZ may have improved localisation of the EZ compared with FDG, indicating its potential as a new clinical tool for the evaluation of patients for epilepsy surgery.

p274
GABA-A receptor density abnormalities associated with seizure outcomes in epileptogenic cavernous angioma
KONDO A[1], MATSUDA K[1], BABA K[1], TOTTORI T[1], UMEOKA S[1], NAKAMURA F[1], TERADA K[1], USUI K[1], MIHARA T[1], AGARI T[2], DATE I[2], INOUE Y[1]
[1]National Epilepsy Center, Shizuoka Institute of Epilepsy and Neurological Disorders, Shizuoka, Japan, [2]Department of Neurological Surgery, Okayama University Graduate School of Medicine, Dentistry, and Pharmaceutical Sciences, Okayama, Japan

POSTER ABSTRACTS

Purpose: Single-photon emission computerized tomography (SPECT) analysis of Gamma-aminobutyric acid-A (GABA-A) receptors binding by (123)I-labelled Iomazenil ((123)I-IMZ) has been applied in some neuropsychiatric disorders. The deficit in GABA-A receptors indicated that abnormal synchronization was mediated by the lack of inhibitory postsynaptic mechanism. In this study we investigate IMZ SPECT in a small series of patients who
harbor supratentorial cavernous angiomas presenting with seizures and surgically treated in our institute.
Method: Ten patients underwent microsurgical resection of supratentorial cavernomas which were pathologically confirmed. We performed lesionectomy, extended lesionectomy, standard temporal lobectomy respectively. The preoperative interictal IMZ SPECT findings in these patients were reviewed retrospectively. The data were statistically analyzed using three-dimensional stereotactic surface projection (3D-SSP).
Results: Consequently, 80% (8/10) of patients with intractable epilepsy achieved Engel Class I outcomes. Decreases of IMZ uptake were observed in the lesion (which means epileptogenic area) in 60% (6/10) of patients and they were statistically significant. In addition, increases of IMZ uptake neighboring the lesion were recognized in 75% (6/8) of Engel Class I patients. On the other hand, about 70% (2/3) of patients without increases of IMZ uptake around the lesion have class III (worthwhile improvement).
Conclusion: This is, to our knowledge, the first report on GABA-A receptor density abnormalities associated with seizure outcomes in epileptogenic cavernous angioma patients. We hypothesize that in our series the increase in GABA-A receptor density surrounding the lesion might be related to the intrinsic antiepileptic mechanisms. Further studies are needed to clarify the possible mechanisms.

p275
The influence of the morphological brain changes on electrogenesis and pharmacoresistance in epileptic patients
KHORSHEV SK[1], KORSAKOVA EA[1], POLYAKOV YI[2]
[1]The V.M. Bekhterev Psychoneurological Research Institute, St.Petersburg, Russian Federation, [2]Institute of the Human Brain, Russian Academy of Sciences, St.Petersburg, Russian Federation

Purpose: The purpose of the present study consisted in investigation of the influence of the character of morphological brain changes on the level of cerebral pathoelectrogenesis in epileptic patients and the frequence of occurrence of pharmacoresistance.
Method: 93 adult patients with epilepsy and 30 healthy subjects were examined. The objective estimation of the pathological process was carried out with the aid of magnetic resonance tomography, phase-contrast angiography when necessary and electroencephalography. There were determined the fractal dimensionality of the alpha rhythm power fluctuations (D) in the O1 as well as alpha rhythm cross-correlation coefficient (R) between the F3 and O1.
Results: According to the degree of possibility of triggering of the epileptic process and its receptivity to the treatment with AEP and depending on their character organic brain changes can possess: 1) high epileptogenic influence (hippocampal sclerosis, etc), characterized by maximum manifestations of pathoelectrogenesis in the form of the highest values of D and R, determining pharmacoresistance in 33% of cases; 2) medium epileptogenous influences (enlargement of the ventricular system, intracerebral and arachnoidal cysts, vascular malformations) reflected by a something less pronounced disturbance of electrogenesis with pharmacoresistance in 20% of observations; 3) low epileptogenous influence (cortical atrophy, cerebral calcinates) determining minimum pathological changes of electrical cerebral processes and pharmacoresistance in 10.5% of cases
Conclusion: Thus there has been established the effect of the character of structural-morphologic cerebral disorders on the intensity degree of epileptogenesis and the level of intactness of the latter to the medicamentous influence.

POSTER ABSTRACTS

p276

FDG PET and Iomazenil SPECT study of late onset medial temporal lobe epilepsy (mTLE) patients with subtle amygdalar enlargement and slight increased signal on MRI

MATSUDA K, BABA K, USUI N, UMEOKA S, KONDO A, TOTTORI T, MIHARA T, INOUE Y
National Epilepsy Center, Shizuoka Institute of Epilepsy and Neurological Disorders, Shizuoka, Japan

Purpose: Among mTLE patients who has been diagnosed no obvious abnormality on MRI, we found a group of MRI findings with subtle amygdalar enlargement accompanied with slight increased signal. The clinical profile showed late seizure onset and relatively medically controllable. Surgical specimens showed minor neuronal morphological changes with moderate glial proliferation. We investigated functional neuroradiological findings in this peculiar group.
Method: : Nine mTLE patients (age at examination: 30-59y; age at seizure onset: 21-56y) with unilateral subtle amygdalar enlargement and increased signal on MRI were studied. Epileptic focus was identified by seizure manifestation and both interictal and ictal EEG recording. The side of subtle MRI abnormality corresponded with the side of epileptic focus. Seizures of 7 patients are medically well controlled and 2 patients were operated for intractable seizures. In this study, neuroradiological findings of FDG PET for glucose metabolism and iomazenil SPECT for central type benzodiazepine receptor binding were examined.
Results: (1)FDG PET: hypometabolic area of focus side was detected in all patients, but hypometabolic change was dominantly recognized in lateral and anterobasal temporal cortex compared to medial structure.(2)Iomazenil SPECT: hypoaccumulation of both medial and lateral temporal cortex in focus side were detected in 6 out of 9 patients.
Conclusion: Functional imagings of FDG PET and iomazenil SPECT are useful to detect and confirm epileptic focus in late onset mTLE with only subtle amygdalar abnormality on MRI.

p277

Imaging mouse models of brain network disorders

KOLBE S[1], GAVRILESCU M[2], WRIGHT D[1], JOHNSTON L[1,3], O'BRIEN TJ[4], EGAN G[1], HILL EL[4]
[1]*Howard Florey Institute, Parkville, Australia,* [2]*Air Operations Division, Defence Science and Technology Organisation, Fishermans Bend, Australia,* [3]*Department of Electrical & Electronic Engineering, University of Melbourne, Parkville, Australia,* [4]*Department of Medicine (RMH), University of Melbourne, Parkville, Australia*

Purpose: Numerous brain disorders including epilepsy and autism potentially result from abnormal neuronal connections and network instability. Altered neural connectivity as observed in autism patients could also be evident in genetic mouse models. Investigations of neural structure and function in mice are limited by technical challenges of imaging small animals. We are therefore developing imaging techniques to investigate neuronal function and neuro-axonal microstructure in a mouse model expressing a gene mutation in the synaptic adhesion molecule Neuroligin-3 (NL3) linked to autism. These mice demonstrate increased somatosensory cortical inhibition. Outcomes from this study may lead to improved understanding of neuronal network disorders.
Method: Five adult mice were investigated using functional MRI and diffusion tensor imaging (DTI) under anaesthesia (0.5-1.5% isoflurane). We have developed and assessed analytical procedures for investigating structural and functional connectivity in a single animal.
Results: Functional connectivity within the somatosensory network was enhanced compared to the visual network potentially reflecting the acutely developed tactile sensory system in mice. Rostro-caudal patterns of DTI parameters indicating corpus callosal structural integrity were similar to published results.

POSTER ABSTRACTS

Conclusion: Assessment of structural and functional connectivity in mouse models of brain network disorders is feasible. These techniques will be used to characterise brain structure and function in the NL3 mouse model to potentially reveal altered network behaviour which can be further investigated using invasive electrophysiological techniques. The results may inform the understanding of how such disturbances in cerebral network function contribute to the development of epilepsy, a common co-morbidity in children with autism.

p278

Ictal SPECT in paediatric epilepsy of temporal lobe origin: correlation of perfusion patterns with surgical outcome

PUSHPALATHA SUDHAKAR L[1], SITA JS[1], MANAS P[1], SUNDARAM C[2], PRABHAKAR RAO VVS[2]
[1]Krishna Institute of Medical Sciences, Secunderabad, India, [2]Nizam's Institue of Medical Sciences, Hyderabad, India

Purpose: To identify Ictal perfusion patterns in paediatric Temporal lobe epilepsy (TLE) & to correlate with Surgical outcome.
Method: A retrospective analysis of Ictal & Interictal SPECT, Ictal Video EEG, MRI and surgical outcome was performed in 27children of TLE. SPECT Perfusion patterns were classified as Typical (anteromedial, anterolateral, inferior) and Atypical (extra temporal). Surgical outcome was assessed according to Engel's classification.
Results: Ictal and Interictal SPECT were done in 21, Interictal alone in 6. Sixteen had Hippocampal sclerosis (HS), while nonHS group had neuronal loss in four, Dual pathology in two, FCD in 2, ganglioglioma in 1, gliosis in one and normal in one. Sensitivity of ictal SPECT is 95 %, interictal is 78%. SPECT was diagnostic in 85.7 % of normal MRI patients. Typical pattern with anteromedial and lateral hyperperfusion is commonest in HS (75%). Atypical pattern is more commonly seen in nonHS group than with HS (45.4% vs 25%). 91.6 % of HS group with typical pattern are in Engel's class 1 outcome. Whereas 80% of nonHS group with atypical pattern are in poor surgical outcome ($p < 0.05$)
Conclusion: Ictal SPECT is highly sensitive (95%) in presurgical evaluation of paediatric TLE. Typical pattern is commonest in HS (75%) and shows good surgical outcome. Significant association of atypical pattern with poor surgical outcome is noted in nonHS group (p value< 0.05) SPECT was diagnostic in 85.7% of normal MRI patients.

p279

Abnormal MRI finding in focal cortical dysplasia: correlation with surgical outcome and pathologic finding

KIM S[1], KIM DW[2], CHU K[1], LEE SK[3], CHUNG C[K4]
[1]Comprehensive Epilepsy Center, Department of Neurology, Seoul National University Hospital, Seoul, Korea, Republic of, [2]Department of Neurology, Konkuk University Medical Center, Seoul, Korea, Republic of, [3]Department of Neurology, Seoul National University Hospital, Seoul, Korea, Republic of, [4]Department of Neurosurgery, Seoul National University Hospital, Seoul, Korea, Republic of

Purpose: Although focal cortical dysplasia(FCD) is no MRI abnormality in many case, We had noticed that MRI abnormalities correlate with severe pathologic feature in previous study. But, MRI appearances of FCD have catergorizable abnormality. (high signal intensity in T2 weighted image, blurring of Gray-white matter junction, cortical thickness, adjacent ventricular abnormality, cystic change, cortical atrophy). We investigated correlation of MRI abnormality with pathologic finding and surgical outcome.

POSTER ABSTRACTS

Method: Our study included patients who had undergone surgical treatment for refractory epilepsy and had abnormal MRI with pathologic diagnosis of FCD at the seoul national university hospital between November 1995 and December 2008. MRI finding was classified into high signal intensity in T2 weighted image, blurring of Gray-white matter junction, cortical thickness, adjacent ventricular abnormality, cystic change and cortical atrophy. We performed pearson chi-square test to investigate correlation of pathological finding and surgical outcome with these MRI findings.
Results: A total of 69 consecutive patients were included. Most frequent finding was cortical-subcortical high signal and atrophy (25/69, 36.2%). Cortical thickness (p = 0.001) and blurring of Gray-white matter junction (p = 0.03) were associated with type II palmini group. All of MRI finding were not associated with surgical outcome.
Conclusion: Our study shows that certain MRI abnormality in FCD is associated with severe pathologic finding.

p280
In vivo MRS glutamate/glutamine levels in brain tumour associated epilepsy
LIUBINAS SV[1,2], YUEN T[1], MOFFAT B[2], STEWARD C[2], D'ABACO G[1], PARADISO L[1], KOKKINOS C[2], ROSENTHAL M[2], DESMOND P[1,2], DRUMMOND KJ[1,2], KAYE AH[1,2], O'BRIEN TJ[1,2], MOROKOFF AP[1,2]
[1]The University of Melbourne, Melbourne, Australia, [2]The Royal Melbourne Hospital, Melbourne, Australia

Purpose: Brain tumour associated epilepsy (TAE) is common, difficult to control with current anti-epileptic drug (AED) therapy, and a cause of significant loss of quality of life. Excessive glutamate in the peritumoural region may play a role in the mechanisms of TAE. In a pilot study we analysed the correlation between in-vivo glutamate/glutamine concentrations measured by magnetic resonance spectroscopy (MRS) and TAE in 9 patients with supratentorial gliomas.
Method: 9 patients with supratentorial gliomas underwent routine preoperative MRI and MRS prior to surgery. MR spectra were obtained from three 8cm3 voxels: one placed in central, non-necrotic tumour; one in the peritumoural region, within 2 cm of tumour border but not including macrosopic tumour; and one in the contralateral brain, mirroring the tumour position.
Results: When compared to contralateral brain, glutamate/glutamine levels were observed to be significantly higher for the gross tumour (p=0.024) but not for the peritumoural area (p=0.12). Significantly higher (p< 0.047) glutamate/glutamine levels were observed in gross tumour compared to peritumoural regions in all the glioma patients. Elevated glutamate/glutamine levels (p=0.02) were also observed in the peritumoural region of TAE positive patients compared to TAE negative patients, however there was no significant difference when the comparing the gross tumoural regions (p=0.4).
Conclusion: This pilot study demonstrates proof-of-concept for the feasibility of MRS being used to detect increased peritumoural glutamate levels in-vivo as a biomarker for patients at risk of developing TAE.

p281
Hyperactive putamen in patients with paroxysmal kinesigenic choreoathetosis: a resting-state functional magnetic resonance imaging study
ZHOU B, CHEN Q, ZHANG Q, CHEN L, GONG Q, SHANG H, TANG H, ZHOU D
West China Hospital, Sichuan University, Chengdu, China

Purpose: Paroxysmal kinesigenic choreoathetosis (PKC) is a rare neurologic disorder characterized by sudden attacks of brief involuntary dyskinetic movement which are precipitated by voluntary movement. The purpose of this study is to localize cerebral functional abnormalities in idiopathic PKC with resting-state functional magnetic resonance imaging (fMRI).

POSTER ABSTRACTS

Method: From May 2007 to August 2008, seven patients with idiopathic PKC were included. Seven subjects of an age- and sex-matched control group were recruited. Interictal brain fMRI was performed in the PKC patients and in the normal controls. Voxel-based analysis was used to characterize the alteration of amplitude of low frequency fluctuation (ALFF) in patients with PKC. Difference between the patient patterns and the control was analyzed with student t-test. To our knowledge, there has been no research about the ALEF in idiopathic PKC.
Results: The patients with PKC showed increased ALFF in the bilateral putamen and the left postcentral gyruson the resting-state fMRI. Compared with the control group, the patient group presented significant differences ($T>3.1, P < 0.005$ uncorrected ,voxel size >10). In contrast, other brain regions revealed no significant differences in ALFF between the 2 groups. Furthermore, in the bilateral putamen and the left postcentral gyrus, ALFF values were not significantly correlated with age at onset, duration of disease, frequency of episode, onset side.
Conclusion: fMRI could be useful to evaluate PKC with negative conventional imaging. It was suggested that abnormal cortico-striato-pallido-thalamic loop might be associated with the pathophysiology of idiopathic PKC.

p282
Investigation of ictal NIRS in children with intractable epilepsy
ISHIYAMA A[1], NAKAGAWA E[1], SUKIGARA S[1], OKAZAKI T[1], HIYANE M[1], FUKUMURA S[1], AIZAKI K[1], TAKESHITA E[1], SAKUMA H[1], SAITO Y[1], KOMAKI H[1], SUGAI K[1], SASAKI M[1], KOBAYASHI I[2]
[1]Department of Child Neurology, National Center Hospital, NCNP, Tokyo, Japan, [2]Center for the Research and Support of Educational Practice, Tokyo Gakugei University, Tokyo, Japan

Purpose: The purpose of this study was to confirm the feasibility of near-infrared spectroscopy (NIRS), determining changes in cerebral blood flow (CBF) and cerebral blood oxygenation (CBO) just before and during seizures in children with intractable epilepsy, by comparing findings with those of ictal electroencephalography (ictal EEG) and ictal single photon emission computed tomography (ictal SPECT).
Method: Subjects were fifteen children with localized-related epilepsy, startle epilepsy or epilepsy converted from West syndrome ranging in age from 4 months to 17 years (5 males and 10 females) admitted to our hospital from October 2009 to May 2010. The hemodynamics patterns of oxy- and deoxy-hemoglobin were recorded using the ETG 4000 (Hitachi Medical Corp.) and compared with the findings of ictal EEG and ictal SPECT.
Results: In total, 22 seizures were recorded in eleven children (11/15, 73.3%) with NIRS. All findings of NIRS demonstrated that CBF and CBO increased remarkably on epileptogenic zone during seizures and presented any of the following three patterns just before seizure; monophasic (increase), biphasic (decrease-increase) or triphasic (increase-decrease-increase) pattern. Same tendency was found in 85% of the subjects (6/7) in ictal SPECT, and 72.7% (7/11) in ictal EEG, respectively. Especially, in four subjects, whose findings were similar in both ictal EEG and ictal SPECT, ictal NIRS findings were also the same tendency.
Conclusion: It is possible to measure the change of CBF and CBO using NIRS before and during seizures in children with intractable epilepsy. NIRS is useful to elucidate the pathophysiology of epileptic seizures.

p283
Cortical reorganization and reduced efficiency of visual word recognition in right temporal lobe epilepsy: a functional MRI study
JENSEN E, HARGREAVES I, PEXMAN P, GOODYEAR B, FEDERICO P
University of Calgary, Calgary, Canada

POSTER ABSTRACTS

Purpose: We investigated the efficiency of lexical and semantic processing in participants with right temporal lobe epilepsy (TLE). We also mapped brain activation patterns during this processing using functional magnetic resonance imaging (fMRI).
Methods: Ten participants with right TLE and 12 healthy controls were studied. All participants underwent a 3 tesla fMRI investigation during a lexical decision task (LDT). Stimuli included words (concrete and abstract) and nonwords. Lexical and semantic processing were examined by comparing behavioural (response times and accuracy) and fMRI data associated with words and nonwords (lexicality) and with concrete and abstract words (concreteness), respectively.
Results: Both groups exhibited significant behavioral effects of lexicality and concreteness. However, right TLE participants showed a larger lexicality effect and had longer response times compared to controls. The right TLE group exhibited different patterns of fMRI activation compared to controls. Specifically, increased left hemispheric activation was seen, particularly in the left inferior frontal gyrus (IFG) during nonword processing.
Conclusion: Right TLE negatively affects the efficiency of lexical processing and lexical decision making. Increased involvement of the left IFG suggests that reorganization of the cortical networks involved in lexical processing occurred as a result of pathology in the right hemisphere.

p284
Single-subject voxel-based T2 relaxometry in focal epilepsy of uncertain origin
KOSIOR R, SHARKEY R, FRAYNE R, FEDERICO P
University of Calgary, Calgary, Canada

Purpose: Voxel-based relaxometry (VBR) is a whole-brain statistical analysis of T2 values from magnetic resonance imaging that can identify abnormalities not easily visible on routine scans. VBR may provide important information in patients with focal epilepsy of uncertain origin. Our objective was to use single-subject VBR to corroborate, or refute indeterminate seizure localization.
Methods: We assessed 51 patients and 25 healthy controls. Scanning was performed at 3T with a Carr-Purcell-Meiboom-Gill sequence. Initial diagnoses were based on history, video-EEG, and structural MRI. Patients were classified as having suspected but unconfirmed epilepsy (SE), known epilepsy with unknown focus (KE) or known epilepsy with a suspected focus (SF). The SF group were determined to have a suspected lobe (SF-L), or a suspected lobe and side of origin (SF-LS). VBR was performed with SPM2 (alpha = 0.05, uncorrected). VBR severity scores were based on abnormal findings in 13 predefined regions, classified as high (>6 areas), medium (3-6), low (1-2) or no VBR abnormalities (0).
Results: Seventeen of 27 SF patients (63%) showed VBR abnormalities in the suspected focus, confirming seizure localization. The SF-L group showed the highest proportion of patients with high or moderate VBR scores. Patient groupings exhibited more VBR abnormalities than controls, where for example, the average number of VBR abnormalities was 1.96 for controls versus 5.55 for the SF group.
Conclusions: Single-subject VBR can help identify or confirm seizure localization in patients in whom seizure localization is uncertain based on conventional investigations.

Miscellaneous

p285
Epilepsy education at the "grass root level" using simple tools - a prototype model: an Indian perspective
KULKARNI C
St.John's Medical College, Bangalore, India

POSTER ABSTRACTS

Purpose: Pharmacology deals with use of drugs in diseases, conventionally taught through didactic lectures. Such teaching/learning requires imagination on part of both teacher and learner. PowerPoint presentations have made break through, but are considered 'killing'. Newer methods are therefore necessary to make pharmacology interesting with pharmacotherapy of epilepsy being no exception. Epilepsy afflicts 50 million population. Global Campaign by WHO, ILAE and IBE, recommend strategic approach in educating people about epilepsy to bring 'epilepsy out of shadows'. In this context, an attempt was made to design tools to educate health professionals at 'grass root level' using epilepsy as a prototype model. The objectives were to - i] identify methods of teaching / learning ii] design simple tools to encourage active learning and iii] make learning interesting.
Method: An integrated approach since 1999, has been evolved in teaching pharmacotherapy of epilepsy, in collaboration with neurologists including relevant psychosocial aspects. Teaching involved medical undergraduates, postgraduates and nursing students. In addition to didactic lectures, students' seminars, problem based learning, clinical case discussions, prescription writing, demonstrations using animal experiments, exercises, Computer Assisted Teaching [CAT] module, research projects, participation in community educational campaigns, were employed.
Results: Methods employed were simple, feasible and innovative. Students' qualitative feed back revealed, that these methods have made learning interesting and exciting.
Conclusion: Attempts are being made to employ one or more of these tools in teaching/learning of other topics. In addition, relevant and suitable methods are being evolved, to develop practical skills and to improve patient care.

p286
The effects of opiate receptor agonists and antagonists on spontaneuse seizure activity in hippocampal slices
ALIJARAHI A, AMINI M
Qazvin Azad University, Qazvin, Iran, Islamic Republic of

Purpose: Inthe present study we addressed the question of whether morphine application potentiated spontaneous seizure activity in epileptogenic hippocampal slices.
Method: Hippocampal slices(~400µm) were prepared from young Wistar rats (P15- 25).Seizure activity was induced by continuously perfusing the slices with low Mg2+ perfusate in an interface recording chamber. Extracellular recordings were performed in the hippocampal CA1 pyramidal cell layer.Low Mg2+ caused spontaneous epileptic activity in all studied hippocampal slices (n = 5).Seizure activity was quantified by measuring the amplitude and duration of the ictal events as well as their number before and after the application of the study drugs.Also,the number of interictal spikes was determined to complement the analysis of seizure discharges before and after drug application.
Results: 1)Low doses of morphine(10 µM)suppressed seizure activity,whereas high doses of morphine(15,30 & 100 µM) potentiated seizure activity in a dose dependent manner. This effect was completely reversed by the addition of naloxone(10µM).2) Dyn-A(10 µM), a κ-opioid receptor agonist caused a significant increase in the incidence and amplitude & duration of ictal activity and these effects were completely reversed by the addition of a selective κ - opioid receptor antagonist, nor-BNI(10 µM). 3) DAMGO (10 µM), a µ-opioid receptor agonist similar to Dyn-A increase seizure activity.
Conclusion: morphine in doses > 10 µM potentiates seizure activity in epileptogenic hippocampi in vitro probably through a direct action on the ì and ê-opiate receptors. However, lower concentrations of morphine inhibit seizure discharges in the low Mg2+ hippocampal epilepsy model by an unknown mechanism that needs further investigation.

POSTER ABSTRACTS

p287
Knowledge of epilepsy among paediatric nurses in an acute regional hospital
LEE WC, YIP LH
Tuen Mun Hospital, Hong Kong, Hong Kong

Purpose: Epilepsy is the most common chronic neurological disorder in children, background knowledge of epilepsy among health care providers is essential. This study aims to evaluate epilepsy knowledge among Paediatric nurses working in an acute hospital.
Method: A one-week survey was conducted in March 2008; self-administered epilepsy knowledge questionnaires (EKQ) were distributed to all registered nurses working in the Department of Paediatrics and Adolescent Medicine, Tuen Mun Hospital. EKQ consisted of 34 true-false items. The questionnaires have been standardized and reported. EKQ were scored based on correct answers. Demographic data of nurses was collected.
Results: 118 (99%) nurses responded. All of them were females. 17 (14.4%) had higher nursing qualifications. 64 (54.2%) had >10 years nursing experience. Average EKQ score was 24.8 (range 20-30, median 25, SD 2.6). Of the 34 questions 70% were answer correctly (0-100%). Half of the responders were medium scorers (EKQ scores >25) and 5 (4.2%) were high scorers (EKQ scores >30). Five questions were answered incorrectly in >60% of responders. Incorrect answer was provided by all in one question regarding anticonvulsant prescription. Scores were not associated with nursing qualifications and experience. Rather, experience in dealing children with epilepsy was related to a higher score.
Conclusion: Epilepsy knowledge was acceptable among paediatric nurses (higher when compared with similar study carried in another South East Asia country). Gaps in knowledge were identified, in particular relating to causes of epilepsy and medications.

p288
First pediatric epilepsy service in Myanmar
LINN K, MIN AYE AM
Yangon Children Hospital, Yangon, Myanmar

Purpose: This study aims to describe how the pediatric epilepsy service recently established in Myanmar and the profile of children with epilepsy during early years of epilepsy service.
Method: This is a descriptive study and the hospital records were used to study the patient profile.
Results: Pediatric epilepsy service started as an out-patient clinic in Yangon Children Hospital (YCH) in 2005. Initially, all children with epilepsy were diagnosed and managed clinically without the help of EEG. Altogether 95 children in 2006, 131 children in 2007, 135 children in 2008 were managed. First pediatric neurologist was appointed in 2006 and he got an epilepsy fellowship form the Asian Epilepsy Academy in 2008 for training in epileptology. YCH has got a digital EEG machine in 2008; therefore epilepsy management could be carried out more effectively since 2009. In 2009, 167 children were newly diagnosed as epilepsy, 42% as generalized seizure, 57% as focal seizure and 1% could not be classified. Epileptic syndromes could be classified only in 27 children (16.2%) and idiopathic generalized epilepsy, benign childhood epilepsy with centrotemporal spikes, childhood absence epilepsy and West syndrome were common epileptic syndromes. Regarding the etiology, 21% were symptomatic, 51% cryptogenic and 28% idiopathic. Common causes were intracranial infections and perinatal events.
Conclusion: Presently the country still has only one pediatric neurologist. Although pediatricians, physicians and general practitioners are managing many epileptic children, education, training, treatment, services and research are still required all over the country.

POSTER ABSTRACTS

p289

Effect of valproic acid on carotid artery intima-media thickness in epileptic patients

ZHAO J[1], ZHANG M[1], LENG Y[2], DENG L[1], LUO X[1], GONG L[1]
[1]The Second Affiliated Hospital to Nanchang University, Nanchang, China, [2]The First People's Hospital of Jiujiang, Jiujiang, China

Purpose: To investigate the changes of the carotid artery intima-media thickness(IMT) in epileptic patients treated with valproic acid(VPA), and the factors related to the changes.
Method: 30 patients under VPA therapy and 36 healthy control subjects were included in the study. All the subjects received measurement of IMT at both sides of the common carotid artery (CCA) by B-mode ultrasonography, then mean CCA IMT was calculated. A fasting blood sample for uric acid, glucose, and lipid profile [total cholesterol(Tc), triglycerides(TG), high-density lipoprotein cholesterol(HDL-c), low-density lipoprotein cholesterol(LDL-c) was also collected to assess. Meanwhile, we calculated atherosclerosis index (Tc/HDL-c and LDL-c/HDL-c ratio). Multiple regression analysis was adopted to screen the risk factors for mean CCA IMT (such as duration of epilepsy, administration time, et al).
Results: Compared with control group, CCA IMT was significantly increased in epileptic patients. Other parameters including serum uric acid, fasting blood glucose, and TG were significantly elevated in epileptic patients, while HDL-c was significantly reduced($p<0.05$). Although we didn´t observe any differences in serum Tc and LDL-c, the atherosclerosis index was significantly higher in epileptic patients($p<0.05$). Multiple linear regression analysis further revealed that duration of epilepsy was independently associated with CCA IMT(Standardized coefficients Beta is 0.619; $p<0.001$) in epileptic patients. When they were divided into two groups according to the duration of epilepsy(≤3 years;>3 years), significantly higher CCA IMT was found in>3 years group($p<0.001$) .
Conclusion: Epileptic patients have a tendency to develop higher CCA IMT, the reason for which may be the duration of epilepsy rather than VPA therapy.

p290

Treating epilepsy on board a train in India: can this be a sustainable and successful model for epilepsy care delivery in India?

SINGH MB, PRASAD K, PADMA MV, BHATIA R, BEHARI M
All India Institute of Medical Sciences, Department of Neurology, New Delhi, India

Purpose: A 70-80% treatment gap for epilepsy in India is largely due to a lack of awareness and a predominantly rural population which cannot access the largely urban health facilities. The purpose of this ongoing endeavor is to explore if epilepsy education and treatment can be successfully provided by a 'train-hospital'. India has 6,909 railway stations over a total route length of more than 63,327 kilometers.
Method: A 2-3 day epilepsy education, screening and treatment camp has been conducted at each of Lifeline Express's (train-hospital) destinations since June 2009. Villagers at each stop have been persuaded and incentivized to attend an epilepsy clinic on the train. A structured questionnaire has been developed, validated and pilot-run and is being used to explore epilepsy related knowledge, attitudes and practices across the country.
Results: Till date, 1098 PWE have been seen in 7 villages, with 364 females and 287 children. In this sample, 88% were drug naïve and another 28% were on irregular treatment. Non-availability of medications(76%), financial hardships(42%), a belief that AEDs are ineffective (59%), lack of information about epilepsy(78%), prevalence of faith healing(64%) and ignorance about the importance of compliance (60%) were the main causes of the treatment gap. Significant regional differences between villages exist.
Conclusion: Use of the extensive railway network may be effective for spreading epilepsy awareness and narrowing the treatment gap in India. This is an ongoing project being developed as a 'hub and spokes' program enlisting help from medical/paramedical staff available in the rural community.

POSTER ABSTRACTS

p291

Naturalistic study on the efficacy of topiramate, as monotherapy in high doses: a clinical trial on 18 cases of resistant seizures to common AED

NAGARAJAN V[1,2]

[1]Tamil Nadu Dr Mgr Medical University, Madurai, India, [2]Vn Neuro Care Center, Madurai, India

Purpose: 18 cases of resistant epilepsy to common AED have been selected for Monotherapy of Topiramate in Asian population.
Method: The selected patients were of the age group of 15 - 21 years, both male and female. All these patients presented with structural damage to the brain parenchyma due to perinatal injuries. These cases have been tried with conventional AEDs, with adequate serum concentration of therapeutic range, with poor clinical and EEG seizure control. They were administered with Topiramate as monotherapy in graded doses, to achieve upto 900 mg per day, the maximum tolerated dose.
Result: The follow up period showed total seizure control of 100 % (n = 18) and EEG seizure control up to 88.88 % (n = 16). The cases were followed up over a period of 18 months. The observed side effects were mainly attributable to weight loss, accounted to 16.66% (n = 3). No other significant side effects observed during the study period.
Conclusion: The findings were statistically significant using paired ' t ' test ($p < 0.05$) This naturalistic study observed the efficacy of the drug in the seizure control with structural damage in young Asian adults, showing administration of higher doses is feasible with minimal side effects.

p292

Free testosteron level and sperm assessment in people with epilepsy among Indonesian population in Jakarta

ISTIAWATI SE

Dharmais Cancer Hospital, Jakarta, Indonesia

Objectives: There are various clinical manifestations apart from epileptic seizures including impairment of testosterone and sperm quality. The aim is to asses free testosterone level and the quality of sperm among Indonesian population in Jakarta, Indonesia.
Method: A descriptive analysis study conducted in active epilepsy who had no epileptic surgery and no long medication of other co-morbidities. The diagnose was made by interview with the patients and the witness including my clinical data. I correlated with the patient's age, type of seizures, OAEDs and the efficacy of treatment.
Result: Participant were 61 male subjects. The patient's age was 16 - 60 year-old. Among them, 9.8 % CPS , 67,2% SP evolving to generalized seizures and 23% CP evolving to generalized seizures. Monotherapy in 77.0% and politherapy in 23.0%. About 34.4% were controlled and 65.6% uncontrolled.
Testosteron assessment were fail and inconclusive in 16.4%. Low in 25.8% and 74.2% in controlled and uncontrolled respectively ; p 0.035.
Sperm assessment fail in 36.1%. Normal sperm group (count,morphology,motility and viability) was 49.2% and abnormal (oligozoosperm, teratozoosperm, asthenosperm and moderate viability) was 14.8%. The sperm movement was assess in two group of sperm: 'normal' in 11.1% abnormal group ;' abnormal' in 88.9% abnormal group (p 0.006).
Conclusion: There are significant difference regarding low level of free testosterone between controlled and uncontrolled subjects and abnormal sperm quality between the abnormal and normal sperm movement.

POSTER ABSTRACTS

p293

Characterization of the anti-epileptic properties of a complement factor 5a antagonist in mouse models of epilepsy

THOMAS NK, BENSON M, WOODRUFF T, TAYLOR S, BORGES K
University of Queensland, Brisbane, Australia

Purpose: Epilepsy is a disorder of the central nervous system affecting approximately 1% of the population. Of those affected, 30% are pharmacoresistant; hence the need for novel anti-epileptic treatments. Complement factor 5a (C5a), the most powerful anaphylatoxin produced by the complement cascade, has been implicated in both human epilepsy and animal epilepsy models. PMX53, the C5a receptor antagonist, has demonstrated therapeutic properties in various inflammatory disease models. We have characterized the properties of PMX53 in the 6Hz mouse model which mimics human complex partial seizures.
Method: Briefly, low frequency (6Hz) long duration (3s) rectangular electrical stimulation, pulse width 0.2ms, is applied to anaesthetised cornea (0.5% tetracaine) using an electroconvulsive rodent shocker. The CC50, the critical current at which 50% mice seize, was calculated as described by Kimball et al. (Radiation Research 1957;7:1-12). Mice were treated at various time points (15min, 30min, 60min, 90min, 4hour) prior to treatment with vehicle, 1mg/kg or 3mg/kg PMX53 sub-cutaneously.
Results: At 30 min, the CC50 of mice treated with 3mg/kg PMX53 (18.98±0.88mA, n=13) was significantly higher than those treated with vehicle (14.34±0.21mA, n=13) or 1mg/kg PMX53 (14.7±0.87mA, n=13). Furthermore, pretreatment with 3mg/kg PMX53, 30 min prior to testing, significantly increased the CC50 compared to vehicle or other pretreatment times.
Conclusion: Our results suggest a transient anti-epileptic effect of PMX53, although it is not yet clear how this is mediated. We plan to test the effects of PMX53 in C5a receptor-deficient mice to elucidate whether this effect is due to antagonism at C5a receptors.

p294

Experiences on pharmacist epilepsy counseling service in specialist outpatient clinics

HIE SL, LEE SHD
KK Women's and Children's Hospital, Singapore, Singapore

Purpose: The aim of setting up pharmacist epilepsy counseling service at outpatient setting is to improve parents' and caregivers' knowledge of epilepsy, its management and use of antiepileptic drugs, thereby improving patient's compliance to medication and consequently reducing time spent at consultation with neurologists.
Method: Various counseling tools were developed and employed. Patients were referred by the neurologists to the counseling service. Trained outpatient pharmacists were rotated on weekly basis to provide this payable service. A follow-up call to the parent/caregiver was made two weeks post-counseling to ensure patient's compliance and address concerns that might arise. Patient demographics, antiepileptic drug usage and response to the service were collated and analysed.
Results: Total of 114 patients were referred to the counseling service in two years. At the time of counseling, majority of the patients (92 out of 144, 81%) were prescribed with one antiepileptic drug. The most commonly counseled drugs were sodium valproate and carbamazepine (49 out of 114, 43%). About 51% of cases (58 out of 114) had special issues addressed during counseling. 32 cases received follow-up from December 2009 to April 2010. Three of these were lost to follow-up. None of the follow-up cases had issues which necessitate early follow-up with their respective neurologists.

POSTER ABSTRACTS

Conclusion: Pharmacist epilepsy counseling service at outpatient setting was able to assist neurologists in addressing various issues that epileptic patients and their parents/caregivers may have. Follow-up service provided assurance to the parents/caregivers that subsequent concerns or issues could be addressed in a timely manner.

p295
Working of free epilepsy clinic in the community: 25 years experience
CHAUDHRY HR[1], SHABBIR A[1], SHAFIQ N[2], USMAN S[1]
[1]Department of Psychiatry, Fatima Jinnah Medical College/ Sir Ganga Ram Hospital, Lahore, Pakistan, [2]Fountain House, Lahore, Lahore, Pakistan

Free Epilepsy clinic in the community was started in 1985 with the help of FLAME (First Librated Action Movement against Epilepsy) at Ahbab Hospital, Ravi Road, Lahore, Pakistan. This clinic is situated in the urban slums of Lahore. In the last 25 years, over 250,000 patients have attended this free clinic. A visiting team comprising of neuropsychiatrist, medical officers, psychologists, pharmaceutical representatives, students, and housewives weekly attend the facility. Approximately 300 patients attend this facility every week, and they are provided with free medicines. This number is increasing gradually, possibly due to the large number of referrals from old patients or due to the continuity of the service and availability of free medicines. The educational programs are also conducted for patients and their families quarterly within the premises of this facility and annually at Fountain House (a well-known rehabilitation facility for chronic psychiatric patients). These programs are attended by the patients and their families, teachers, students, writers, journalists, artists, politicians, sportsmen, and mental health professionals. The main aim of these programs is to educate and provide awareness to the general public about epilepsy, with special emphasis on providing information about education, career selection, marriage and pregnancy in patients with epilepsy. During the past 25 years, research studies various aspects of epilepsy have been conducted and published in well reputed national & international journals. Working of this clinic provides an excellent model of community involvement in running a free epilepsy clinic for the poor and deserving patients.

p296
An unusual case of Hashimoto's encephalopathy associated with thyrotoxicosis
TOMITA S, KINOSHITA M, TAHARA M, YAMAKAWA K, OEDA T, SAWADA H, KONISHI T
Utano National Hospital, Kyoto, Japan

Purpose: Hashimoto´s encephalopathy (HE) is poorly recognised encephalopathy generally seen in patients of chronic thyroiditis. To characterize encephalopathy with autoimmune thyroid disease, we report an unusual case of HE associated with Graves' disease (GD).
Method: A 79-year-old Japanese woman admitted with altered mental status, high fever, tachycardia, and generalized tonic clonic seizures. She had had hypothyroidism and been treated with levothyroxine for 6 years and in euthyroid status, however four months ago she presented with fever, palpitation and tremor. She was diagnosed as Graves' disease based on hyperthyroidism, positive TRAb and high uptake in Thyroid-Tc scintigraphy, and was under anti-thyroid treatment. Blood exam showed overt hyperthyroidism and a high titer of anti-thyroperoxidase (anti-TPO) antibody. EEG showed generalized slow waves and intermittent polyspikes. After excluding other cause of encephalopathy, thyrotoxic encephalopathy was suspected and she was initially treated with propylthiouracil, phenytoin and atenolol for 3 days. Fever and tachycardia disappeared but her consciousness level gradually worsened.
Intravenous administration of high dose corticosteroid dramatically improved her consciousness. Epileptiform activities on EEG disappeared.

POSTER ABSTRACTS

Results: Althogh this case was found to fulfil the diagnostic criteria of both GD and HE, considering the clinical course resistant to anti-thyroid treatment, elevated anti-TPO antibody and marked steroid-responsiveness, seizures and consciousness disturbance of this case were very likely caused by HE.
Conclusion: HE should be suspected in the face of acute encephalopathy associated with autoimmune thyroid disease even in the presence of thyrotoxicosis.

p297
Prophylactic phenytoin in cases of cerebral malaria
BHANDARI G, MALOO JC
Manidhari Hospital & Maloo Neuro Center, Jodhpur, India

Purpose: To study seizure prophylaxis in cases of cerebral malaria with concomitant use of phenytoin. Cerebral malaria is unarousable coma caused by acute infection with plasmodium Falciparum. Cerebral malaria is associated with generalized Tonic-Clonic seizures in upto 40% of adults, and these seizures are associated with prolonged coma and increased risk of neurological sequalae and death.
Method: We studied 100 cases of cerebral malaria from april 2003 to march2010 , age range was 15 to75 years, 70% were males and 30% were females. All patients were managed in ICU and treated with antimalarials (combinations of IV Quinine and IV Artesunate), supportive treatment, antipyretics and prophylactic Phenytoin. The loading dose was 15 mg./kg followed by maintenance dose of 100 mg 8 hrly
Results: Majority of patients (90%) recovered completely and seizure was not observed in any of the patient.
Conclusion: Prophylactic Phenytoin in all cases of cerebral malaria can prevent seizures without significant adverse effects and prevent Post Malaria Neurological Syndrome.

p298
Towards consumer directed service delivery
IRELAND C, KAIN S, TODD L, WELSH C
Epilepsy Action Australia, Sydney, Australia

Purpose: To better understand consumer needs, a review of client individual service plans (ISP's) was undertaken. This review provided vital information about the needs experienced by people with epilepsy and the most effective strategies to meet these needs.
Method: ISPs developed by Epilepsy Action Australia (EAA) service providers in response to clients' needs in New South Wales over a 3 month period were collated. Results were segmented by client age to better understand client needs across the lifespan.
Results:

- Overall the most commonly identified needs were 'information (31%)' and 'seizure management (29%)'
- 'information' was identified mostly by parents of pre-school/ school age clients.
- 'Seizure Management' was the most commonly identified need for both school age and adult clients.
- 'Emotional/ social' needs were most commonly identified for school age clients.
- A review of internal processes has been conducted as a result of information gained about the strategies and length of time taken to meet various needs.
- The review indicated the need to develop a new outcome measurement tool.
- Initial results indicated the need to expand the research sample size and geographical spread - the conference presentation will display results of the larger sample.

POSTER ABSTRACTS

Conclusion: To provide consumer directed care, it is vital to understand the needs experienced by people with epilepsy and the most effective strategies to meet these needs.
By collating and analysing client ISPs, EAA has identified information vital for all epilepsy service providers.

p299
Utility of long-term video-EEG monitoring in older adults and elderly
BAHETI NN, RADHAKRISHNAN A, RADHAKRISHNAN K
Sree Chitra Tirunal Institute for Medical Sciences and Technology, Trivandrum, India

Purpose: World-over, the majority of patients undergoing Video-EEG monitoring (VEM) are in second or third decades. Although elderly represent the fastest growing segment of population with epilepsy, only few of them undergo VEM. We evaluated the utility of VEM in diagnosis and long-term management of older-adults and elderly with epilepsy.
Method: 148 consecutive patients aged 45 and above, who underwent long-term (≥8 hours) inpatient VEM from 1996-2009 formed the cohort. Using a structured proforma, we gathered their demographic, clinical, electrophysiological and long-term outcome data.
Results: The mean age was 51.3 ± 6.4 years; mean duration of VEM was 69.3 hours; mean duration of follow-up was 37.7 months. The indications of VEM were presurgical evaluation (58.8%), syndromic classification (20.6%) and suspicion of non-epileptic events (NEE) in 21%. None developed any complications during monitoring. Seizures with NEE occurred in 19 (12.8%), NEE alone in 14 (9.5%) patients. VEM improvised syndromic diagnosis and lead to antiepileptic drug (AEDs) optimization in 79 patients (53.4%), of these 26 (32.9%) were seizure-free, 38 (48.1%) had ≥ 50% reduction in seizures at last follow-up. At presentation, 138 patients were on AEDs, 100 (67.6%) on duo/polytherapy, while at last follow-up only 50 (33.8) were on ≥2 AEDs and 24 were off AEDs (p< 0.0001). 25 (14.9%) underwent surgery, all remaining seizure-free at a mean follow-up of 39.4 months.
Conclusion: VEM is a safe and cost-effective investigation strategy in older-adults and elderly. It aided in improving the diagnosis, offered better treatment including surgery and helped in excluding non-epileptic paroxysmal events in majority.

p300
Antiepileptic effects of triheptanoin in genetic absence epilepsy rats from Strasbourg (GAERS)
SHE D[1], JONES N[2], O'BRIEN T[2], BORGES K[1]
[1]The University of Queensland, Brisbane, Australia, [2]The University of Melbourne, Melbourne, Australia

Purpose: Triheptanoin, the triglyceride of the anaplerotic C7 fatty acid heptanoate, has been successfully used as a dietary treatment for hereditary metabolic disorders in patients. We recently established that feeding triheptanoin is antiepileptic in two chronic focal mouse epilepsy models, (i) the corneal kindling model and (ii) a second hit pentylenetetrazole model in mice after pilocarpine-induced status epilepticus. This indicates that triheptanoin may be effective against absence seizures. To further elucidate the antiepileptic profile of triheptanoin, this study will determine the efficacy of triheptanoin against electrographic genetic absence seizures in Genetic Absence Epilepsy Rats from Strasbourg (GAERS).
Method: GAERS were surgically implanted with 6 electroencephalogram (EEG) recording electrodes and underwent serial 4 hour baseline EEG recordings prior to diet initiation of either 35% calories as triheptanoin (n=5) or standard diet (n=5). They then underwent serial 4 hour recordings for a further 4 weeks on treatment. The number of seizures and the total time in seizure during the recording period were calculated by review of the EEG and compared between the two diet groups.
Results: Preliminary results suggest that, after initiation of triheptanoin diet, no alteration in the number of seizures (192 ± 20 seizures vs 224 ± 10 seizures; p=0.16) or the cumulative time spent in seizure (11.5 ± 0.5% vs 11.3 ± 0.4%; p=0.86) occurs, compared to animals on the control diet.

POSTER ABSTRACTS

Conclusion: Although this needs to be confirmed, it appears as though the triheptanoin diet does not influence the expression of absence seizures in GAERS.

p301
Insights into epilepsy through visual art
CHAMBLISS J[1,2], COOK M[3,4], BOLT B[5], WILLIAMS D[1], COSS E[6], PINIKAHANA J[2], BARDENHAGEN F[7], MEADE C[7], SIMPSON L[7], LASALLE COLLEGE OF THE ARTS · ART THERAPY INTERNS
[1]University of Melbourne, Carlton, Australia, [2]Epilepsy Foundation of Victoria, Camberwell, Australia, [3]University of Melbourne, Department of Medicine, Fitzroy, Australia, [4]St Vincent's Health, Fitzroy, Australia, [5]Victoria College of Arts, Melbourne, Australia, [6]LaSalle College of the Arts, Singapore, Singapore, [7]St. Vincent's Health, Fitzroy, Australia

Purpose: The objective was to evaluate how informed observations of visual art can help to diagnose and to better understand epilepsy.
Method: More than 120 artists with epilepsy and/or migraines provided more than 1,200 images of preexisting artworks for blind studies by an art therapist and by a neurologist. Additionally participants completed two drawing tasks that were evaluated by objective and subjective standards in independent blind studies. Family members of the artists with epilepsy completed the drawing tasks as controls. Participants also provided written background information regarding age, education, medical status, artistic training, artistic preferences, and experiences with visual impairments, illusions and hallucinations. The artworks and drawings were reviewed by Jim Chambliss after reading the written information and conducting brief interviews when appropriate. Dialog with artists with epilepsy provided affirmation of their conscious intentions, creative preferences, and prominent influences of epilepsy in their visual art.
Results: The independent evaluations, surveys, and observations disclosed a significantly higher probability that participants had focal epilepsy when the contents of their artworks contained:(1) erratic and fragmented line quality,(2) distortion of objects and human forms,(3) distortion of spatial awareness,(4) representations of visual illusions and hallucinations,(5) dreamlike or surreal content, and/or(6) heightened detail (particularly in the drawing tasks).
Conclusion: Visual art is a valuable source of information to help diagnose and to understand epilepsy, interictal behavior, comorbid conditions, and the people behind the art. Such insights often cannot be acquired through patient interviews and traditional diagnostic procedures.

p302
Ketogenic diet in refractory epilepsies
THAMMONGKOL S[1], VEARS D[2], BICKNELL-ROYLE J[1], NATION J[1,3], DRAFFIN K[4], STEWART KG[5], CAMERON F[6,7], SCHEFFER I[2,5,6], MACKAY M[1]
[1]Children's Epilepsy Program, Royal Children's Hospital, Melbourne, Australia, [2]Epilepsy Research Centre and Department of Medicine (Neurology), University of Melbourne, Austin Health, West Heidelberg, Australia, [3]Department of Nutrition and Dietetics, Royal Children's Hospital, Melbourne, Australia, [4]Nutrition and Dietetics Department, Austin Health, West Heidelberg, Australia, [5]Paediatrics Department, Austin Health, West Heidelberg, Australia, [6]Department of Paediatrics, The University of Melbourne, Royal Children's Hospital, Melbourne, Australia, [7]Department of Endocrinology, Royal Children's Hospital, Melbourne, Australia

Purpose: To report the efficacy, side effects and tolerability of the ketogenic diet (KD) in refractory epilepsies focussing on outcome with regard to epilepsy syndromes and aetiology.
Method: 64 consecutive children 4 adults were treated with the classical KD between 2002 and 2009. Electroclinical details were obtained and each patient was classified according to their epilepsy syndrome and aetiology. Responses to the KD were defined as >50 % reduction in seizure frequency compared to baseline.

POSTER ABSTRACTS

Results: 52 patients had symptomatic generalised epilepsy, 7 had idiopathic generalised epilepsy, 2 had focal epilepsy. Aetiology of the epilepsy was determined in 35/64 (54%) patients. Aetologies included structural or metabolic (24), genetic causes (18) adn unknown (19). Mean age at initiation was 4.8 years (IQR 2.0-8.6 years). 7/68 patients were excluded from analysis. 29/61 (48%) patients were responders at 3 months; including 2 adults. Two patients became seizure free. Responsive syndromes included migrating partial epilepsies of infancy, childhood absence epilepsy, focal epilepsy, epilepsy with myoclonic-atonic seizures and Dravel syndrome. Aetiology did not appear to influence likelihood of response and children with lissencephaly adn hypoxic ischaemic encephalopathy had surprisingly
good responses. Hypercalciuria was the most common side effects, seen in 22/61 (36%) of patients.
Discussion: The Ketogenic Diet is an effective and generally well tolerated treatment for children adn adults with refractory epilepsy. Response is predicted by epilepsy syndrome but not by aetiology. Accurate characterisation of electroclinical syndrome is an important factor in decisions about timing of initiation of the KD.

p303
Risk of seizure recurrence after anti-epileptic drug withdrawal in neurocyticercosis treated and non-treated group
GORKHALY MP
National Academy of Medical Sciences, Bir Hospital, Kathmandu, Nepal

Purpose: To assess the recurrence rate of seizures in patient with epilepsy following drug withdrawal after 2 or more seizure free years in NCC treated and non-treated group.
Method: This is a prospective study of 100 patients with epilepsy. Patients were divided into 2 groups. Group1 included50 patients whose CT scan showed calcified spot and weren't treated for Neuro-cysticercosis and Group 2 included patient whose CT scan showed active NCC and serology positive and treated with Albendazole and steroids. The patients were evaluated periodically for 2 yrs and another 18 months after stopping drugs. Patient having tubercular granuloma weren't included since recurrence may be due to reactivation of tuberculosis. Similarly progressive neurological deficits, severe mental retardation, cerebral palsy, Juvenile myoclonic epilepsy, acute symptomatic seizure and previous recurrent seizure at AED withdrawal were excluded.
Results: In albendazole treated group, with clearance of ring enhancing shadow at the time of drug withdrawal, there's no episodes of recurrence in 18 months follow-up. However, in patient with imaging showing calcified spot (non- treated group), 6 patients (12%-p-value (t-test):< 0.001) showed recurrence of seizure in 18 months follow-up.
Conclusion: Recurrence rate of seizure is more common in patient with Non-treated group with imaging showing calcified spots than patients with treated and normal imaging group. Risk of recurrence may be related to duration and more no. of seizures at time of diagnosis.

p304
Effect of insulin and melatonin on kainic acid induced learning impairment in rats
NIKBAKHT F, YEGANE F
Department of Physiology and Neuroscience Research Centre, Shiraz University of Medical Science, Shiraz, Iran, Islamic Republic of

Introduction: Kainic acid an excitotoxin which produces seizure and neuronal damage, exhibits deficits in special learning in the Morris water maze. Insulin and melatonin are hormones with beneficial function in learning and memory. This study was to determine if these hormones would block the behavioral effects of kainic acid on learning using the Morris water maze task.

POSTER ABSTRACTS

Materials and methods: 35 male Sprague Dawley rats were randomized into 6 groups:
Group 1: control received 0.9% saline injections i.p.
Group 2: convulsive dose of kainic acid (7mg /kg i.p.) rats received their injections 5 days prior to water maze testing and then received no further injection.
Groups 3 and 4: insulin and melatonin. In these groups rats received either 1 IU/kg insulin 2 hours before testing each day or 20 mg/kg i.p. melatonin 45 min before testing.
Groups 5 and 6: (combination of kainic acid with insulin or melatonin).
Results: The results showed that daily insulin or melatonin treatment didn´t alter acquisition learning in Morris water maze. Kainic acid administration caused significant deficits in learning on days 1-5 ($p < 0.05$).In combination groups , pre-treatment blocked the kainin acid induced deficits in spatial learning. (days 3-5 $p < 0.05$ for KA+Insulin group and days 4-5 $p < 0.05$ for KA+Melatonin. How ever ,pre-treatment with insulin or melatonin didn't reduce the seizure signs in rats.
Conclusion: the results revealed a neuroprotective role for insulin and melatonin against kainic acid induced neurotoxicity with convulsive dose.

AUTHORS INDEX

AUTHORS INDEX

AUTHORS INDEX

AUTHORS INDEX

AUTHORS INDEX

AUTHORS INDEX

AUTHORS INDEX

AUTHORS INDEX

AUTHORS INDEX

AUTHORS INDEX

AUTHORS INDEX

AUTHORS INDEX

VIMPAT® (lacosamide) 50 mg, 100 mg, 150 mg and 200 mg film-coated tablets. **Dosage and administration:** Starting dose of 50 mg twice daily increased to an initial therapeutic dose of 100 mg twice daily after one week. Depending on response and tolerability, the dose may be increased by 50 mg twice daily every week, to a maximum recommended daily dose of 400 mg (200 mg twice daily). If VIMPAT® has to be discontinued, gradual withdrawal is recommended. **Contraindications:** Hypersensitivity to any components. Known second- or third-degree atrioventricular (AV) block. **Precautions:** VIMPAT® may cause dizziness or blurred vision. Driving or operating hazardous machinery. Dose-dependent prolongations in PR interval have been observed. Use with caution in patients with conduction problems or with severe cardiac disease. May predispose patients with diabetic neuropathy and/or cardiovascular disease to atrial arrhythmias. Abnormalities in liver function tests have been observed. AEDs, including lacosamide, increase the risk of suicidal thoughts or behaviour. Patients, caregivers, and families should be advised of the need to be alert for the emergence of worsening of the signs and symptoms of depression, any unusual changes in mood or behaviour, or the emergence of suicidal thoughts, behaviour, or thoughts about self-harm. Elderly. Pregnancy. Do not withdraw abruptly. **Adverse Reactions:** Very common: dizziness, headache, nausea and diplopia. Common: depression, balance disorder, coordination abnormal, memory impairment, cognitive disorder, somnolence, tremor, nystagmus, vision blurred, vertigo, vomiting, constipation, flatulence, pruritus, gait disturbance, asthenia, fatigue, fall, and skin laceration. Based on the full disclosure Product Information approved by the TGA 10 Dec 2009. Amended 26 May 2010.
References: 1. VIMPAT® Approved Product Information. 2. Beydoun A *et al. Expert Rev Neurother* 2009;9:33–42.

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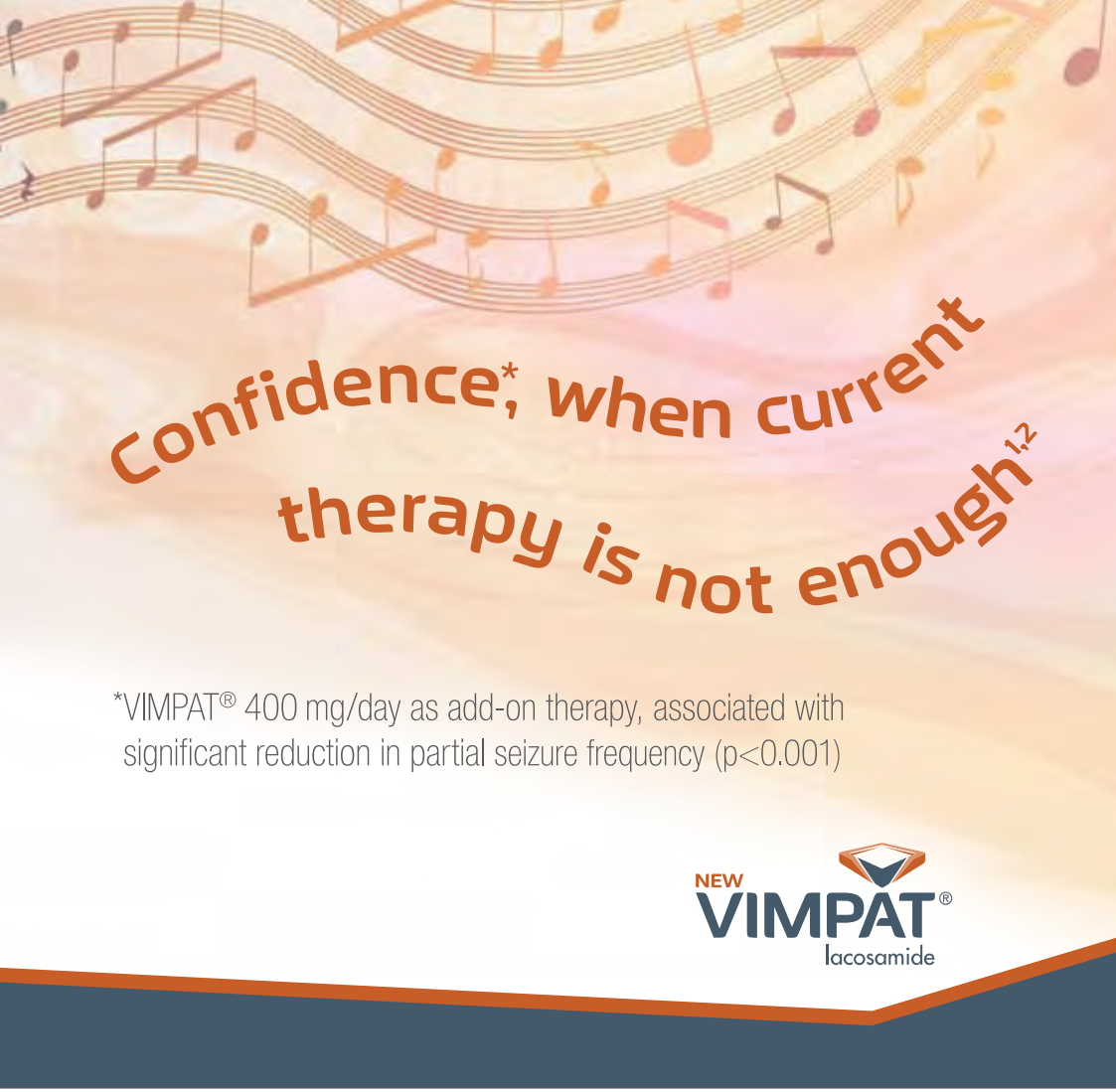

PBS Information: Tablets: Authority required.
Refer to PBS Schedule for full authority information.
Injection: This product is not listed on the PBS.

VIMPAT® is indicated as add-on therapy in the treatment of partial seizures with or without secondary generalisation in patients with epilepsy aged 16 years and older. Please review VIMPAT® Approved Product Information before prescribing. Full Product Information is enclosed in your AOEC satchel.

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Management of inadequately controlled epilepsy – evaluating the evidence

Saturday 23rd October
13:30 – 15:00
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Chair
Prof. Ernest Somerville (Australia)

My journey: from uncontrolled epilepsy to control
Marion Clignet (France)

Uncontrolled epilepsy – evidence-based decision making in clinical practice
Prof. Sam Berkovic (Australia)

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