International Symposium on Neonatal Seizures and Related Disorders (ISNS)

– Cutting Edge in Seizure Detection, Management and Neuroprotection –

ISNS

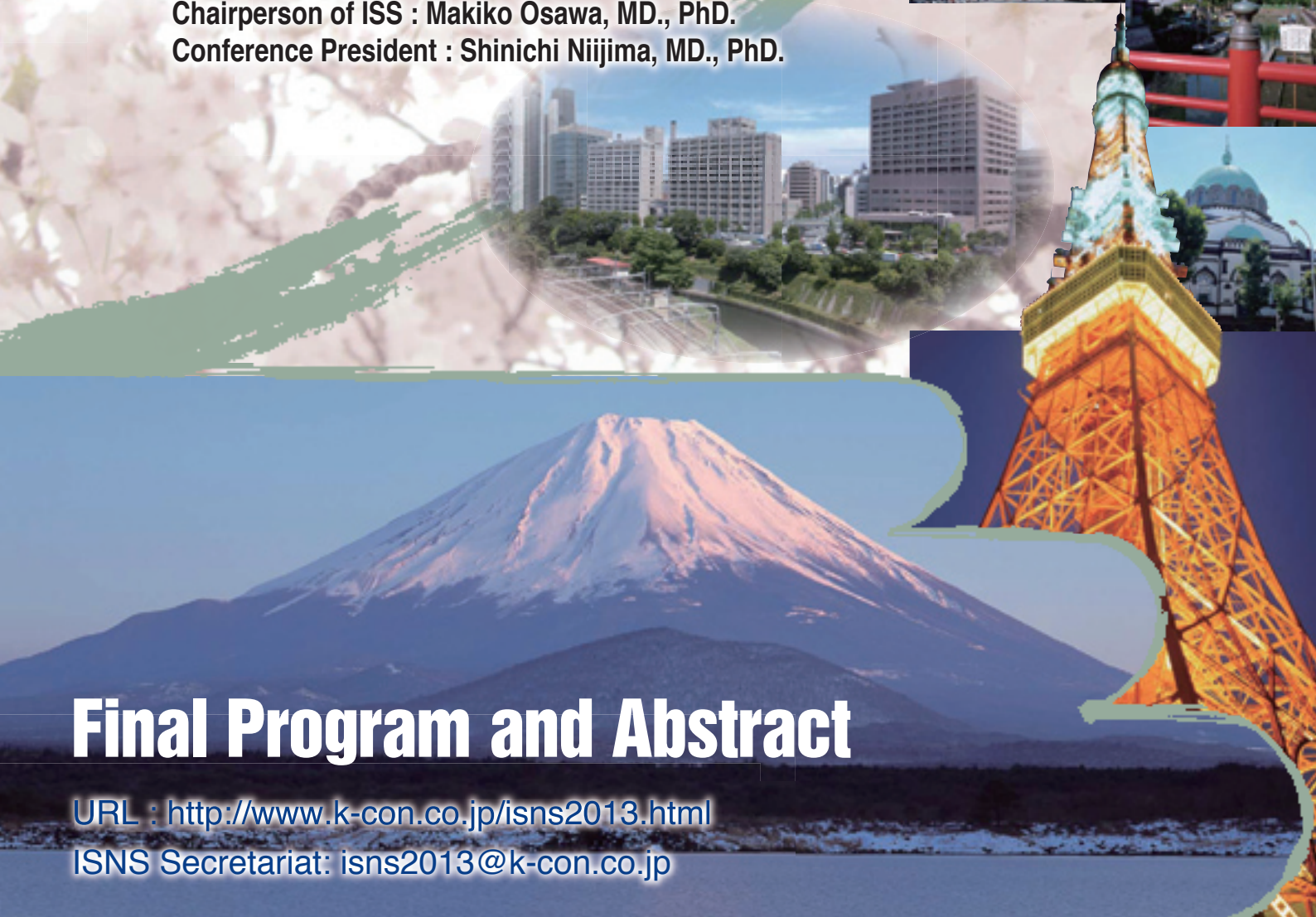
The 15th Annual Meeting of Infantile Seizure Society (ISS)

Under the Endorsement of ILAE, Pediatrics Commission & CAOA

April 12-14, 2013, Tokyo, Japan

Venue : Juntendo University Campus,
Tokyo, Japan

Host : Infantile Seizure Society (ISS)
Emeritus Chairperson of ISS : Yukio Fukuyama, MD., PhD.
Chairperson of ISS : Makiko Osawa, MD., PhD.
Conference President : Shinichi Niijima, MD., PhD.



Final Program and Abstract

URL : http://www.k-con.co.jp/isns2013.html
ISNS Secretariat: isns2013@k-con.co.jp

Program at the Glance

	Day 1, April 12 Friday	Day 2, April 13 Saturday	Day 3, April 14 Sunday
8:00		Morning Seminar 1	Morning Seminar 2
9:00	Satellite Educational Course: Interpretation of Neonatal EEG (Room 105, Bldg.10)	Session Ⅲ Clinico-Electrical Diagnosis	Session Ⅶ Basic Neurosciences 1
10:00			
11:00		Session Ⅳ Clinical Diagnosis & Outcome	Session Ⅷ Basic Neurosciences 2
12:00	Registration	Luncheon Seminar 1	Luncheon Seminar 2
13:00	Opening Ceremony	Session Ⅴ Management	
	Keynote Lecture		
14:00	Session Ⅰ EEG and Seizure Recognition		Session Ⅸ Genetics
15:00		Coffee and Posters 1 / AOCNA ND Meeting	Coffee and Posters 2 / ISS Business Meeting
16:00	Session Ⅱ Neuroimaging		Session Ⅹ New Syndromes & Perspectives
17:00		Session Ⅵ Further Deliberations	
	Evening Seminar		Closing Ceremony & Awards
18:00		Tea Ceremony	Farewell Party Century Tower
19:00	Welcome Reception Century Tower	Grand Social Party Tokyo Garden Palace Hotel	
20:00			
21:00			

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Message from Emeritus Chairperson of ISS

Cordial Welcome !

It is my special privilege and great honor to address my warmest welcome greetings to all colleagues in the globe who are willing to participate in this International Symposium on Neonatal Seizures and Related Disorders (ISNS), Tokyo, Japan, April 12-14, 2013.

My heart is full with emotion when I extended this invitation to you. On March 11, 2011, an overwhelming Megaquake and tsunami attacked northeastern Japan claiming the confirmed death of about 16,000 people. Subsequent nuclear plant crackdown in Fukushima threatened to fatally destroy overall health and peace of the residents. Under these circumstances, we were forced to cancel the ISNS which had been scheduled to be held on April 9-10, 2011.

A year later, however, Japan achieved a miraculous recovery. We are going to reborn as a new humane this society. And, it is our great pride and pleasure to have organized the ISNS in a replenished form again in April 2013. As you would acknowledge, neonatal seizures are one of the utmost important subjects in child neurology.

We have prepared an attractive program consisting of a comprehensive review of recent advances in research and CME dealing with essential basics on neonatal seizures.

We sincerely hope that by attending this ISNS 2013 you were able to enrich your professional knowledge significantly.

Yukio Fukuyama

Yukio Fukuyama, MD, PhD
Emeritus Chairperson, Infantile Seizure Society
Honorary President, Asian & Oceanian
Child Neurology Association

Message from Chairperson of ISS

Dear friends,

It is my great honor and pleasure to deliver this heartfelt welcome to all colleagues worldwide wishing to participate in the forthcoming International Symposium on Neonatal Seizures and Related Conditions (ISNS), in Tokyo, Japan, April 12-14, 2013. Unfortunately, we had to postpone the ISNS, which was originally to have been held on April 9-10, 2011 due to the extremely large earthquake and tsunami which devastated the eastern part of Japan last year. 2 years have passed since that extraordinary event and we are now undergoing a rebirth as a new and more humane society, showing a response like the plasticity of infancy.

It is thus our great pride and pleasure to again organize the ISNS in a renewed form in April 2013. Thanks to the participation of the international experts contributing to discussions of the advances on a number of important issues, covering topics ranging from a comprehensive review of recent advances in research and CME to the essential basics of neonatal seizures As you know, neonatal seizures are one of the most important subjects in child neurology. We sincerely hope that your participation here have significantly enriched your professional knowledge. Your attendance itself, like a growth factor, will aid in our recover from the disaster.

Makiko Osawa, MD, PhD

Chairperson, Infantile Seizure Society
The Previous President of the Japan Society of Child Neurology,
Board Member of the Japan Epilepsy Society
Vice President of Tokyo Women's Medical University
Professor and Chairperson of the Department of Pediatrics
School of Medicine, Tokyo Women's Medical University

Message from President

Dear colleagues,

On behalf of the Organizing Committee, I am very pleased to welcome you to the 15th Annual Meeting of the Infantile Seizure Society and The International Symposium on Neonatal Seizures and Related Conditions (ISNS) which will be held in Tokyo, Japan, April 12-14, 2013.

I was supposed to chair the 14th ISNS in April 2011, but we postponed the meeting due to the Great East Japan Earthquake on March 11 of that year and the subsequent nuclear accident after conducting an internet survey as to whether or not to hold the ISNS in Japan.

The Great East Japan Earthquake claimed the lives of 16,000 people and 3,000 people are still missing. About 6,000 people were injured and a total of 25,000 people suffered from the disaster. I sincerely pray for the all victims of the Great East Japan Earthquake.

It is well known that NS has number of causes and that we often come across subclinical seizures that do not necessarily involve clinical symptoms. Therefore, it is critical for us to make a quick and precise diagnosis as well as to start early treatment of the primary diseases.

In this Symposium, we focused on electrophysiological diagnosis (EEG, aEEG) which is crucial for accurately diagnosing NS. In addition, we also touched on Genetics, Diagnostic neuro-imaging (MRI), Treatment, Prognosis, Early Infantile Epileptic Encephalopathy (EIEE), Hypoxia ischemia and seizures as well as NS-related diseases.

Tokyo, as the capital of Japan, has a history of four hundred years with a variety of traditions and customs that have endured since the Edo era. It has now been widely recognized as one of the most exciting and modern cities, as a result of its remarkable economic growth and cultural progress. You might imagine Tokyo as a kind of ‘concrete jungle’ with office buildings and skyscrapers everywhere. On the contrary, we have two hundred and seventy-seven splendid parks and gardens where you can fully enjoy nature. Temples and shrines abound. We encourage you all to visit Tokyo, again, to experience all three elements of this city simultaneously, the ancient traditions, the modern scene, and nature.

Thank you very much for being a part of this symposium.

Shin-ichi Niijima

Shinichi Niijima, MD, PhD

Chairperson, President, International Symposium on Neonatal Seizures and Related Disorders

Department of Pediatrics, Juntendo University Nerima Hospital

General Information

Dates April 12 (Fri) - 14 (Sun), 2013

Venue
Ariyama Memorial Hall & Century Tower, Juntendo University Medical School, Tokyo, Japan

President
Professor Shinichi NIIJIMA, Department of Pediatrics, Juntendo University Nerima Hospital, Tokyo, Japan

Main Topics
Neonatal seizures - Early detection, identification, long-term monitoring, differential diagnosis, management, outcome, etiology, pathogenesis, animal models; Neonatal encephalopathies (HIE, PVL, etc), early onset epilepsies (EIEE, West, etc)

Target Attendees
Physicians, researchers, nurses, assistants and students who are involved or interested in child neurology, epileptology, EEG, perinatal medicine, NICU care, basic developmental neuroscience.

Official Language English only

Host Organizations
Infantile Seizure Society (ISS)
Fully endorsed by International League Against Epilepsy (ILAE) Pediatrics Commission

ISNS Headquarter
Yoshiyuki OHTOMO (Secretary General)
Department of Pediatrics, Juntendo University Nerima Hospital, Tokyo, Japan

ISNS Secretariat
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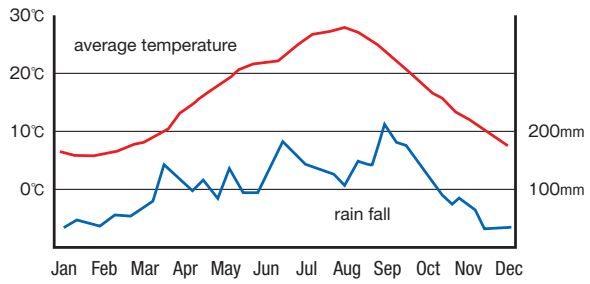
Tel: +81-3-5367-2382 Fax: +81-3-5367-2187

ISS Secretariat
E-mail: iss-contact@iss-jpn.info
Child Neurology Institute, Tokyo, Japan

Climate
Tokyo has four distinct seasons. The summer months (June, July and August) are hot and sticky while winter can be freezing. Tokyo is best visited in spring or autumn. On the conference term, it is in spring season and it is still little bit chilly but getting warm day by day in April.
Tokyo Average temparature in April: 15 °C (60°F)



Currency Exchange
Only Japanese yen is acceptable at regular stores and restaurants. Certain foreign currencies may be accepted at a limited number of hotels, restaurants, and souvenir shops. You can buy yen at foreign exchange banks and other authorized money exchangers on presentation of your passport.

Electricity
Electric current is uniformly 100 volts, AC, throughout Japan, but with two different cycles: 50 in eastern Japan including Chiba and Tokyo, and 60 in western Japan including Kyoto and Osaka. Leading hotels in major cities have two outlets of 100 and 220 volts but their sockets usually accept a two-leg plug only.

Access to Venue

Juntendo University (Hongo Campus)

Address:

Head Office, University & Post Graduate University of Juntendo
Hongo 2-1-1, Bunkyo-ku,Tokyo 113-8421, Japan
Telephone : +81-3-3813-3111 Fax : +81-3-3814-9100

Access from Narita International Airport:

Keisei Skyliner:
From Narita Airport to Nippori Station: Approx. 40 min. by Keisei Skyliner.
From Nippori Station to Akihabara Station: Approx. 8 min.by JR Yamanote Line.
From Akihabara Station to Ochanomizu Station: Approx. 2 min. by JR Sobu Line.

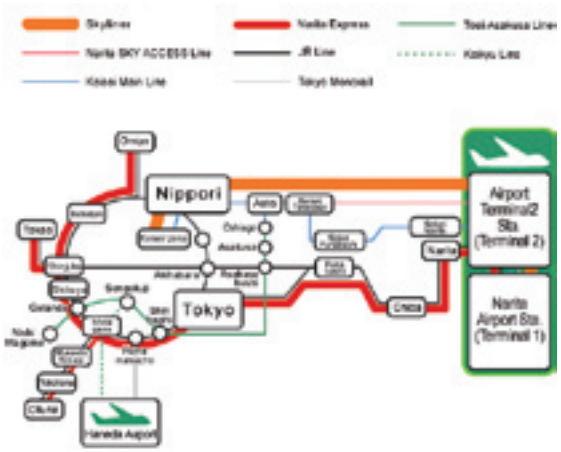
Narita Express Train:
From Narita Airport to Tokyo Station: Approx. 1 hour by JR (Japan Railways) Narita Express Train.
From Tokyo Station to Ochanomizu Station: Approx. 5 min. by JR Chuo Line.

Narita Airport Access Information URL:
http://www.narita-airport.jp/en/access/train/index.html

Access from Haneda Airport (Tokyo International Airport):

From Haneda Airport to Hamamatsucho Station: Approx. 20 min. by Monorail.
From Hamamatsucho to Tokyo Station: Approx. 5 min. by JR Yamanote Line.
From Tokyo Station to Ochanomizu Station: Approx. 5 min. by JR Chuo Line.

Haneda Airport Access Information URL:
http://www.haneda-airport.jp/inter/en/access/

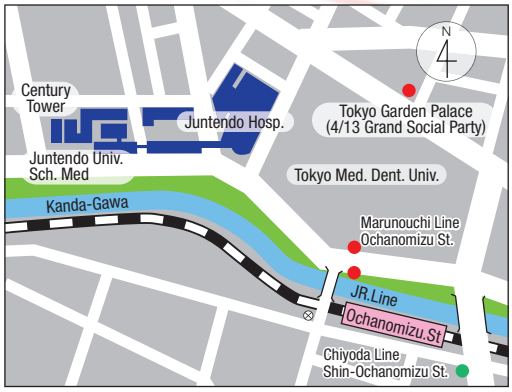


Local Access:

JR : Chuo-line & Sobu-line “Ochanomizu Station”
Subway : Marunouchi-line “Ochanomizu Station”
Subway : Chiyoda-line “Shin-Ochanomizu Station”
Approx. 8 min on foot from the stations

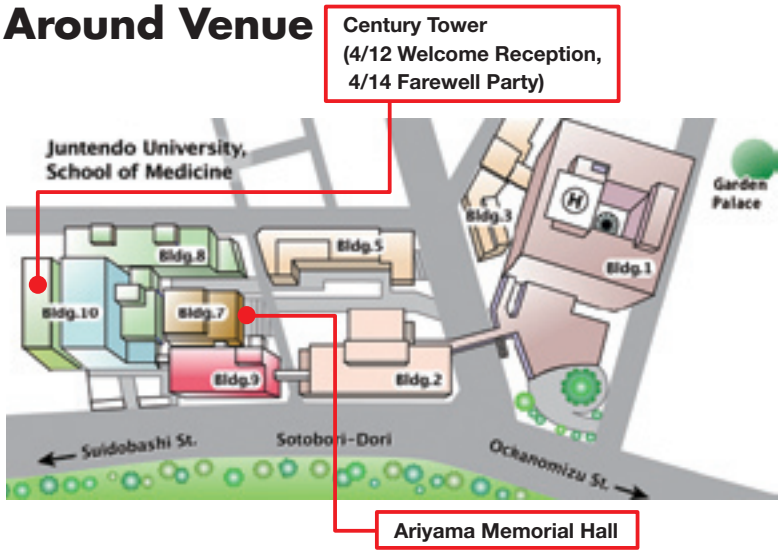
Venue

Hongo Campus

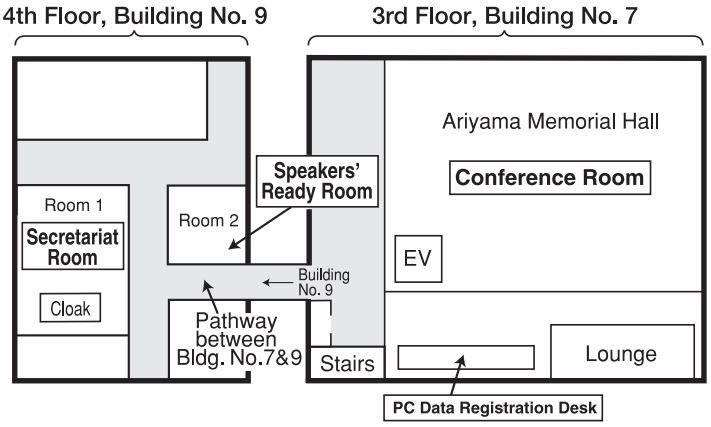


Venue: Bldg.7 "Ariyama Memorial Hall" and Century Tower

Around Venue



Juntendo University Hongo Campus



Registration Desk = 1st Floor Lobby, Building No. 7

Registration Desk: Lobby, 1st Floor, Building No. 7
PC Data Registration Desk: Lobby, 3rd Floor, Building No. 7 (In front of Ariyama Memorial Hall)
Conference Room: Ariyama Memorial Hall, 3rd Floor, Building No. 7
Speakers' Room: Meeting Room 2, 4th Floor, Building No. 9
Secretariat Room: Meeting Room 1, 4th Floor, Building No. 9
Cloak: Meeting Room 1, 4th Floor, Building No. 9
WC: 2nd Floor, Building No. 7 / 4th Floor, Building No. 9
*The 4th Floor of Building No. 9 is on the same level of the 3rd Floor of Building No. 7.

Poster Sessions: Exhibition Hall, Basement Floor, Century Tower
Exhibition and Drink Corner: Exhibition Hall, Basement Floor, Century Tower

Welcome Reception on April 12th: Reception Hall, 19th Floor, Century Tower
Grand Social Party on April 13th: Room "Takachiho", 2nd Floor, Tokyo Garden Palace Hotel
Farewell Party on April 14th: Reception Hall, 19th Floor, Century Tower

Instruction for Oral Presentation

*All Oral Presentation is requested by PC.
*The screen is single projection without sound.
*Please complete your PC registration until 30 minutes ahead of your presentation time at the PC registration desk.

To bring your own PC is requested

*Windows PC will be set at the conference room for your presentation.
*If you bring your own PC, please make sure that your PC has D-Sub 15 pin mini terminal for monitor output. (Some compact PC needs another connector. In case of that, please carry your own connector.)
*Macintosh is acceptable if you will bring your own PC (Please carry your own connector).
*Please bring battery adapter to avoid battery off.
*Because sometimes screen saver or power saving system could be a reason of battery off, please set your PC appropriately.
*Please operate your PPT data by yourself at the podium.

If you bring your data by portable media

*The PC will set at registration desk as follows.
Windows PC (Office 2003, 2007 and 2010)
*To avoid garbled characters, please use standard font which is originally installed by OS.
*Please put your name on your data file.
*Presentation data should be provided by USB memory stick or CDR.
*Backup data by another media should be held by presenter.
*If your presentation includes movies, please carry your own computer.

Instruction for Poster Presentation

*A panel width 90cm×length 180cm will be provided for each poster as following example.

*Abstract number will be prepared by secretariat.

*Title and present author's name are required to prepare by each presenter.

*Pins for display will be provided at each poster panel.

***Presentation time: 5 min. Discussion 3 min. Total: 8 min.** for each poster.

*Place: Exhibition Hall, Basement Floor, Century Tower

*Schedule:

Poster Attachment: April 12, 9:00-18:00

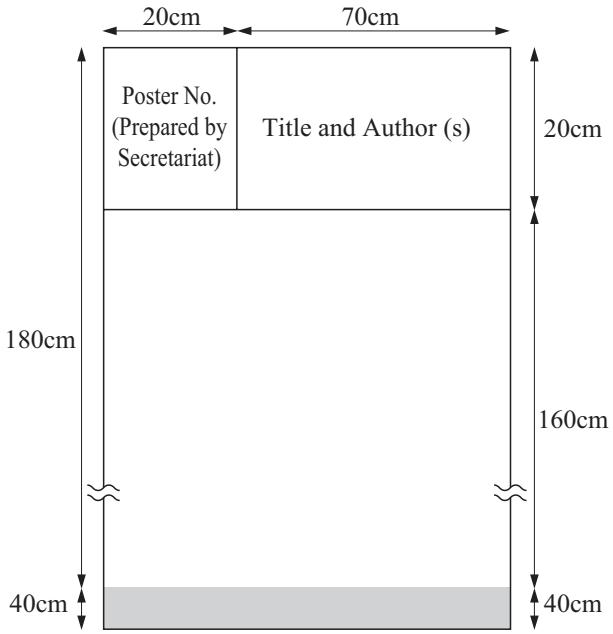
Poster Presentation & Discussion:

Poster Session 1: April 13, 14:40-16:20

Poster Session 2: April 14, 14:50-15:50

Poster Removal: April 14, 16:30-18:00

Poster Panel



Scientific Program

Day 1, April 12 (Friday)

Registration Desk Open **09:00**

Satellite Educational Course

09:00-12:00

Theme : Interpretation of Neonatal EEG
Venue : Conference Room 105, First Floor, Building No.10
Language : Japanese
*This course needs separate Pre-registration to attend.

S1

NORMAL EEG IN TERM AND PREMATURE BABIES (80 min)
Akihisa OKUMURA (Tokyo, Japan)

S2

ABNORMAL EEG IN TERM AND PREMATURE BABIES (80 min)
Tetsuo KUBOTA (Aichi, Japan)

Opening Ceremony

13:00-13:15

Opening Address (1) : **Makiko OSAWA** (Chairperson, ISS)
Opening Address (2) : **Shinichi NIIJIMA** (President, ISNS)
Opening Address (3) : **Hajime ARAI** (Dean, Juntendo University School of Medicine)

Keynote Lecture

13:15-13:55

Chairperson : **Akira OKA** (Tokyo, Japan)

L01 **13:15-13:45 (+10min discussion)**

NEUROPHYSIOLOGICAL ASPECT OF NEONATAL SEIZURES
Kazuyoshi WATANABE (Nagoya, Japan)

Session I EEG and Seizure Recognition

13:55-15:45

Chairpersons : **Lena HELLSTRÖM-WESTAS** (Uppsala, Sweden)
Akihisa OKUMURA (Tokyo, Japan)

L02 **13:55-14:15 (+10min discussion)**

CONVENTIONAL ELECTROENCEPHALOGRAM IN NEONATAL SEIZURES

Toru KATO (Okazaki, Japan)

L03 **14:25-14:55 (+10min discussion)**

AMPLITUDE INTEGRATED EEG FOR SEIZURE DETECTION

Lena HELLSTRÖM-WESTAS (Uppsala, Sweden)

L04 **15:05-15:35 (+10min discussion)**

NON EPILEPTIC MANIFESTATIONS AMONG NEONATES

Federico VIGEVANO (Rome, Italy)

Session II Neuroimaging

15:45-17:05

Chairpersons : **Haluk TOPALOGLU** (Ankara, Turkey)
Toyojiro MATSUISHI (Kurume, Japan)

L05 **15:45-16:15 (+10min discussion)**

DIFFUSION IMAGING OF THE FETAL AND NEONATAL BRAIN

Emi TAKAHASHI (Boston, USA)

L06 **16:25-16:55 (+10min discussion)**

NEUROIMAGING IN NEONATAL SEIZURES

Jeffrey J. NEIL (Saint Louis, USA)

Evening Seminar

17:15-18:15

Chairperson : **Makiko OSAWA** (Tokyo, Japan)

Sponsored by GlaxoSmithKline K.K.

L07 **17:15-18:05 (+10min discussion)**

NEONATAL SEIZURES IN THE PRETERM INFANT

Terrie E. INDER (Saint Louis, USA)

Welcome Reception (at the Century Tower, University Campus) **18:30-21:00**

Day 2, April 13 (Saturday)

Morning Seminar 1

08:10-09:10

Chairperson : **Hideo YAMANOUCHI** (Saitama, Japan)

Sponsored by Kowa Pharmaceutical Co., Ltd.

L08 **08:10-09:00 (+10min discussion)**

EARLY DEVELOPMENT OF EEG ACTIVITY: FUNCTION VS STRUCTURE

Sampsa VANHATALO (Helsinki, Finland)

Session III Clinico-Electrical Diagnosis

09:10-10:20 (+10min discussion)

Chairpersons : **Geraldine B. BOYLAN** (Cork, Ireland)
Yoshihiro TAKEUCHI (Shiga, Japan)

L09 **09:10-09:30 (+10min discussion)**

PROPOSAL OF SEMIOLOGICAL CATEGORIZATION OF NEONATAL SEIZURES

Akihisa OKUMURA (Tokyo, Japan)

L10 **09:40-10:10 (+10min discussion)**

AUTOMATED SEIZURE DETECTION IN NEWBORNS

Geraldine B. BOYLAN (Cork, Ireland)

Session IV Clinical Diagnosis & Outcome

10:20-11:40

Chairpersons : **Phillip L. PEARL** (Washington DC, USA)
Yoko OHTSUKA (Okayama, Japan)

L11 **10:20-10:50 (+10min discussion)**

NEW PARADIGMS IN NEONATAL METABOLIC EPILEPSIES

Phillip L. PEARL (Washington DC, USA)

L12 **11:00-11:30 (+10min discussion)**

NEW RECIPE? THE ROLE OF METABOLISM IN NEUROPROTECTION

Adam L. HARTMAN (Baltimore, USA)

Luncheon Seminar 1

11:50-12:50

Chairperson : **Shinichi NIIJIMA** (Tokyo, Japan)

Sponsored by Pfizer Japan Inc.

L13 **11:50-12:40 (+10min discussion)**

NEONATAL SEIZURES IN DEVELOPING COUNTRIES

Perrine PLOUIN (Paris, France)

Session V Management

12:50-14:40

Chairpersons : **Yong-Seung HWANG** (Seoul, Korea)
Kenji SUGAI (Tokyo, Japan)

L14 12:50-13:20 (+10min discussion)

TREATMENT OF NEONATAL SEIZURES
Hannah C. GLASS (San Francisco, USA)

L15 13:30-13:50 (+10min discussion)

MANAGEMENT OF NEONATAL SEIZURE WITH ANTIEPILEPTIC DRUGS
Hiroyuki KIDOKORO (Nagoya, Japan)

L16 14:00-14:30 (+10min discussion)

EEG MONITORING IN NEONATES: AMERICAN CLINICAL NEUROPHYSIOLOGY GUIDELINES
Nicholas S. ABEND (Philadelphia, USA)

Coffee and Poster Session 1 (at the Century Tower, University Campus) 14:40-16:20

AOCNA ND Meeting (at Room 203, 2F, Bldg. No10) 14:30-16:30

Session VI Further Deliberations

16:30-17:40

Chairpersons : **Hannah C. GLASS** (San Francisco, USA)
Toshisaburo NAGAI (Osaka, Japan)

L17 16:30-16:50 (+10min discussion)

NEONATAL SEIZURES IN INFANTS WITH NEUROINFECTION
Tetsuo KUBOTA (Aichi, Japan), Akihisa OKUMURA (Tokyo, Japan)

L18 17:00-17:30 (+10min discussion)

NEUROLOGICAL PROBLEMS IN FAMOUS MUSICIANS: IN WORDS AND MUSIC
Phillip L. PEARL (Washington DC, USA)

Tea Ceremony 17:00-19:00

Grand Social Party (at Tokyo Garden Palace Hotel) 19:00-21:00
KIYARI SHISHIMAI

Day 3, April 14 (Sunday)

Morning Seminar 2

08:00-09:00

Chairperson : **Taisuke OTSUKI** (Tokyo, Japan)

Sponsored by Kyowa Hakko Kirin Co., Ltd.

L19 **08:00-08:50 (+10min discussion)**

EARLY DEVELOPMENT OF EEG ACTIVITY: FROM BENCH TO CLINIC

Roustem KHAZIPOV (Kazan, Russia)

Session VII Basic Neurosciences 1

09:00-11:00

Chairpersons : **Kai-Ping CHANG** (Taipei, Taiwan)
Masaharu HAYASHI (Tokyo, Japan)

L20 **09:00-09:30 (+10min discussion)**

DEVELOPMENTAL AND PATHOGENIC MODAL SHIFTS OF GABA ACTIONS IN IMMATURE BRAIN

Atsuo FUKUDA (Hamamatsu, Japan)

L21 **09:40-10:10 (+10min discussion)**

PREDICTION OF BRAIN DAMAGE WITH MINIATURE TELEMETRY IN A RAT MODEL OF NEONATAL HYPOXIC-ISCHEMIC ENCEPHALOPATHY: BACKGROUND EEG VERSUS SEIZURES

F. Edward DUDEK (Salt Lake City, USA)

L22 **10:20-10:50 (+10min discussion)**

MRI FOR DIAGNOSIS AND PROGNOSIS OF LONG-TERM INJURY AFTER EXPERIMENTAL NEONATAL ARTERIAL STROKE

Zena VEXLER (San Francisco, USA)

Session VIII Basic Neurosciences 2

11:00-12:10

Chairpersons : **Harvey B. SARNAT** (Calgary, Canada)
Masashi MIZUGUCHI (Tokyo, Japan)

L23 **11:00-11:20 (+10min discussion)**

DEVELOPING COT-SIDE BIOMARKERS FOR ACUTE CEREBRAL INJURY

Osuke IWATA (Kurume, Japan)

L24 **11:30-12:00 (+10min discussion)**

DOES HYPOTHERMIA ALTER THE INCIDENCE OF EPILEPSY IN ASPHYXIATED NEONATES?

Marianne BERÉNYI (Budapest, Hungary)

Luncheon Seminar 2

12:20-13:20

Chairperson : **Hitoshi YAMAMOTO** (Kawasaki, Japan)

Sponsored by UCB Japan Co., Ltd. / Otsuka Pharmaceutical Co., Ltd.

L25 **12:20-13:10 (+10min discussion)**

FUTURE TARGETS FOR ANTISEIZURE THERAPY IN THE NEONATE : PRECLINICAL AND CLINICAL OBSERVATIONS

Raman SANKAR (Los Angeles, USA)

Session IX Genetics — 13:30-14:40

Chairpersons : **Zena VEXLER** (San Francisco, USA)
Mitsuhiro KATO (Yamagata, Japan)

L26 — 13:30-13:50 (+10min discussion)

GENETICS IN BENIGN NEONATAL SEIZURES
Shinichi HIROSE (Fukuoka, Japan)

L27 — 14:00-14:30 (+10min discussion)

NEONATAL EPILEPTIC ENCEPHALOPATHIES DUE TO KCNQ2 MUTATIONS: A NEW TWIST ON AN OLD STORY
Sarah WECKHUYSEN (Antwerp, Belgium)

Coffee and Poster Session 2 (at the Century Tower, University Campus) — 14:50-15:50

ISS Business Meeting (at Room 203, 2F, Bldg. No10) — 14:30-15:30

Session X New Syndromes & Perspectives — 16:00-17:30

Chairpersons : **Perrine PLOUIN** (Paris, France)
Hirokazu OGUNI (Tokyo, Japan)

L28 — 16:00-16:20 (+10min discussion)

VARIATION OF SUPPRESSION-BURST EEG PATTERNS SEEN IN EPILEPTIC ENCEPHALOPATHIES STARTING IN THE NEONATAL PERIOD
Hitoshi YAMAMOTO (Kawasaki, Japan)

L29 — 16:30-16:50 (+10min discussion)

AN EMERGING NEW CLINICO-GENETIC VARIANT OF WEST SYNDROME
Jun TOHYAMA (Niigata, Japan)

L30 — 17:00-17:20 (+10min discussion)

BRIDGING THE AGE GAP - APPLYING NEONATAL SEIZURE LESSONS TO ACUTE SYMPTOMATIC SEIZURES IN OLDER CHILDREN
Nicholas S. ABEND (Philadelphia, USA)

Closing Ceremony — 17:30-17:50

Best Poster Awarding : Toshisaburo NAGAI (Osaka, Japan)
Closing Address (1) : **Hitoshi YAMAMOTO** (President, 17th ISS Int'l Symposium)
Closing Address (2) : **Haluk TOPALOGLU** (President, 16th ISS Int'l Symposium)
Closing Address (3) : **Shinichi NIIJIMA** (President, 15th ISS Int'l Symposium)

Farewell Party (at the Century Tower, University Campus) — 18:00-20:00

Poster Program

Poster Session 1 Day 2, April 13 (Saturday)

Chairpersons: Sampsa VANHATALO (Helsinki, Finland), **Yasuhiro SUZUKI** (Osaka, Japan)

G1-01
14:40-14:48
DEVELOPMENT OF AMPLITUDE-INTEGRATED EEG COLORED ACCORDING TO SPECTRAL EDGE FREQUENCY
Katsuhiro KOBAYASHI (Okayama, Japan)

G1-02
14:48-14:56
THE ADEQUACY OF DENSITY SPECTRAL ARRAY AND AMPLITUDE-INTEGRATED EEG FOR NEONATAL SEIZURE IDENTIFICATION
Hiroyuki YAMAMOTO (Nagoya, Japan)

G1-03
14:56-15:04
GAMMA OSCILLATIONS SUPERIMPOSED ON SUBCLINICAL SEIZURE ACTIVITY IN A NEWBORN INFANT WITH HYPOXIC-ISCHEMIC ENCEPHALOPATHY
Akihito TAKEUCHI (Okayama, Japan)

G1-04
15:04-15:12
CLINICAL MANIFESTATIONS AND EEG FINDINGS OF NEONATAL SEIZURES: A SINGLE CENTER 5-YEAR REVIEW FROM SINGAPORE
Hian-Tat ONG (Singapore)

G1-05
15:12-15:20
EEG BACKGROUND ACTIVITIES IN PRETERM ANDTERM NEONATES WITH SEIZURES
Hasan TEKGUL (Izmir, Turkey)

G1-06
15:20-15:28
PROGNOSTIC VALUE OF EEG BACKGROUND IN TERM NEONATES WITH HYPOXIC-ISCHEMIC ENCEPHALOPATHY
Hasan TEKGUL (Manisa, Turkey)

Chairpersons: Kun-Long HUNG (Taipei, Taiwan), **Katsuhiro KOBAYASHI** (Okayama, Japan)

G1-07
15:28-15:36
OUTCOMES OF PERINATAL STROKE IN CHILDREN WITH INTRAUTERINE HERPESVIRUS INFECTION
Bakhytkul MYRZALIYEVA (Almaty, Kazakhstan)

G1-08
15:36-15:44
NEONATAL SEIZURES IN CHILDREN WITH INTRAUTERINE HERPES INFECTION
Bakhytkul MYRZALIYEVA (Almaty, Kazakhstan)

G1-09
15:44-15:52
A GENETIC ANALYSIS OF BENIGN NEONATAL EPILEPSY IN JAPAN
Yukiko IHARA (Fukuoka, Japan)

G1-10

15:52-16:00
BENIGN NEONATAL SLEEP MYOCLONUS: OUR EXPERIENCE OF 15 JAPANESE CASES
Yasuhiro SUZUKI (Osaka, Japan)

G1-11

16:00-16:08
EPILEPTIC ENCEPHALOPTHY WTH SUPPRESSION-BURST ACTIVITY IN THREE NEONATES
Hasan TEKGUL (Izmir, Turkey)

G1-12

16:08-16:16
NEUROLOGICAL PROGNOSIS FOR NEONATAL CEREBRAL INFECTION
Takashi SETOUE (Fukuoka, Japan)

Chairpersons: Jong Hee CHAE (Seoul, Korea)**, Shin-ichiro HAMANO** (Saitama, Japan)

G2-01

14:40-14:48
CATASTROPHIC INTRACTABLE EPILEPSY IN NEONATES FROM FOCAL CORTICAL DYSPLASIA
Vasu GOOTY (Boston, USA)

G2-02

14:48-14:56
CLINICAL AND ELECTROENCEPHALOGRAPHIC ANALYSIS OF INFANTILE SPASMS : A MEDICAL CENTER EXPERIENCE
Ai-Tyng LIM (Taipei, Taiwan)

G2-03

14:56-15:04
PRECOCIOUS AND DELAYED NEOCORTICAL SYNAPTOGENESIS IN FETAL HOLOPROSENCEPHALY
Harvey B. SARNAT (Calgary, Canada)

G2-04

15:04-15:12
USEFULNESS OF EARLY DIFFUION-WEIGHT-IMAGING IN NEONATAL NONKETOTIC HYPERGLYCINEMIA: A CASE REPORT
Tetsuo KUBOTA (Aichi, Japan)

G2-05

15:12-15:20
MOLYBDENUM COFACTOR DEFICIENCY: FOUR CASES FROM TURKEY
Pinar GENCPINAR (Antalya, Turkey)

G2-06

15:20-15:28
SUCCINATE SEMIALDEHYDE DEHYDROGENASE DEFICIENCY WITH SUBDURAL HAEMATOMA - A CAUSE OF SHAKEN BABY SYNDROME
Lai Choo ONG (Kuala Lumpur, Malaysia)

Chairpersons: Hiroshi OTSUBO (Toronto, Canada)**, Jun TOHYAMA** (Niigata, Japan)

G2-07

15:28-15:36
DOES EARLY ELECTROENCEPHALOGRAPHY (EEG) FINDING OF NEONATAL SEIZURES HAVE A PREDICTIVE VALUE TO LONG-TERM OUTCOME?
Tian SANG (Beijing, China)

G2-08

15:36-15:44
NEONATAL SEIZURES AND PROGNOSYS - CORRELATION BETWEEN CLINICO-ELECTROENCEPHALOGRAPHIC MANIFESTATIONS OF NEONATAL SEIZURES AND DEVELOPMENTAL OUTCOMES-
Kyoko HIRASAWA (Tokyo, Japan)

G2-09

15:44-15:52
PATTERN OF EEG IN NEONATAL SEIZURES (0-28 DAYS) AND ITS RELATION WITH NEURODEVELOPMENT AT 3 MONTH OF AGE IN FULL TERM NEONATES
Akhil SINGH (Bhopal, MP, India)

G2-10

15:52-16:00
FAST ACTIVITY DURING EEG SEI ZURES I N NEONATES : CHARACTERISTICS AND SIGNIFICANCE
Lakshmi NAGARAJAN (Perth, Australia)

G2-11

16:00-16:08
CLINICAL & ELECTROPHYSIOLOGICAL CHARACTERISTICS OF FULL-TERM NEONATAL SEIZURES CONFIRMED BY ELECTRICAL SIGNATURE
Hee HWANG (Seoul, Korea)

G2-12

16:08-16:16
EPILEPSY IN A NEONATE WITH MIGRATION DISORDER ASSOCIATED WITH MECKEL'S DIVERTICULUM - A NEW SYNDROME ? OR SPORADIC CASE
Shyi-Jou CHEN (Taipei, Taiwan)

Poster Session 2 Day 3, April 14 (Sunday)

Chairpersons: Sarah WECKHUYSEN (Antwerp, Belgium), **Shinji SAITOH** (Hokkaido, Japan)

G3-01 14:40-14:48
MALIGNANT MIGRATING PARTIAL SEIZURES IN INFANCY CONTROLLED WITH CLORAZEPATE
Mutsuki SHIODA (Tokyo, Japan)

G3-02 14:48-14:56
ISCHEMIA-MODIFIED ALBUMIN LEVELS IN CHILDREN HAVING SEIZURE
Pinar GENCPINAR (Antalya, Turkey)

G3-03 14:56-15:04
A NEW SYNDROME OF MICROCEPHALY WITH SIMPLIFIED GYRATION, WEST SYNDROME AND INFANTILE DIABETES
Cathryn POULTON (Rotterdam, Netherlands)

G3-04 15:04-15:12
CLINICAL PRESENTATION, TREATMENT AND PROGNOSIS IN CHILDREN WITH REYE-LIKE SYNDROME
Meltem UZUN (Konya, Turkey)

G3-05 15:12-15:20
DEVELOPMENTAL OUTCOME OF CHILDREN WITH WEST SYNDROME
Venkataraman VISWANATHAN (Chennai, India)

G3-06 15:20-15:28
NEURODEVELOPMENTAL AND EPILEPSY OUTCOME IN CHILDREN AGED ONE TO FIVE YEARS WITH INFANTILE SPASMS ONE OR MORE YEARS AFTER ONSET - A CROSS-SECTIONAL STUDY
Rachna SEHGAL (New Delhi, India)

Chairpersons: Federico VIGEVANO (Rome, Italy), **Shintaro YAMASHITA** (Tokyo, Japan)

G3-07 15:28-15:36
PROGNOSTIC UTILITY OF CLINICAL EPILEPSY SEVERITY SCORE IN CHILDREN WITH INFANTILE SPASMS VERSUS KRAMMER' S GLOBAL SCORE FOR HYPSARRHYTHMIA SCORING BEFORE INITIATING THERAPY
Sheffali GULATI (New Delhi, India)

G3-08 15:36-15:44
LONG TERM EPILEPSY OUTCOME OF CHILDREN WITH WEST SYNDROME: RETROSPECTIVE ANALYSIS OF 135 CHILDREN TREATED AT A TERTIARY CARE CENTRE
Sheffali GULATI (New Delhi, India)

G3-09 15:44-15:52
CLINICAL CHARACTERISTICS OF EPILEPSY AFTER NEONATAL ARTERIAL ISCHEMIC STROKE
Kenjiro KIKUCHI (Saitama, Japan)

G3-10

15:52-16:00
ROLE OF HYPOXIA IN THE GENESIS OF EPILEPSY IN CHILDREN
Alma ISSABEKOVA (Almaty, Kazakhstan)

G3-11

16:00-16:08
SHAKEN BABY SYNDROME MANISFESTING AS INFANTILE SPASMS: A CASE REPORT
Chih-Fen HU (Taipei, Taiwan)

Chairpersons: Hian-Tat ONG (Singapore)**, Katsunori FUJII** (Chiba, Japan)

G4-01

14:50-14:58
MUTATION SCREENING OF THE GABRG2 GENE IN KOREAN PATIENTS WITH CHILDHOOD ABSENCE EPILEPSY
Young Ok KIM (Gwangju, Korea)

G4-02

14:58-15:06
A NOVEL PCDH19 MUTATION IN A PATIENT WITH MICROCEPHALY, INFANTILE MYOCLONIC EPILEPSY AND PSYCHOMOTOR RETARDATION
Jao-Shwann LIANG (Taipei, Taiwan)

G4-03

15:06-15:14
CONTINOUS EEG MONITORING IN PEDIATRIC INTENSIVE CARE UNIT IN TAIWAN
Kuang-Lin LIN (Taoyuan, Taiwan)

G4-04

15:14-15:22
NEIGHBORHOOD, FAMILY AND RISK OF CHILDHOOD AND ADOLESCENT EPILEPSY: A NATIONWIDE EPIDEMIOLOGICAL STUDY FROM SWEDEN
Xinjun LI (Lund, Sweden)

G4-05

15:22-15:30
HYPERBARIC OXYGEN THERAPY IN CHILDREN WITH HYPOXIC ENCEPHALOPATHY USING VENTILATOR - A MEDICAL CENTER EXPERIENCE IN TAIWAN
Shyi-Jou CHEN (Taipei, Taiwan)

G4-06

15:30-15:38
AGE-DEPENDENT NEUROLOGIC VULNERABILITY TO CYCLOSPORINE AMONG CHILDREN
Li-Wen CHEN (Taipei, Taiwan)

G4-07

15:38-15:46
CORRELATION BETWEEN DIFFUSION TENSOR IMAGING AND DEVELOPMENTAL PROGNOSIS IN CRYPTOGENIC WEST SYNDROME
Tatsuya FUKASAWA (Aichi, Japan)

Chairpersons: Hannah C. GLASS (San Francisco, USA)**, Jun NATSUME** (Nagoya, Japan)

G5-01
14:50-14:58
A SURVEY ON THE USE OF NEWER ANTIEPILEPTIC DRUGS FOR NEONATAL SEIZURES AMONG FILIPINO PEDIATRIC NEUROLOGISTS
Thaddeus Jose BAYANA (Manila, Philippines)

G5-02
14:58-15:06
NEONATAL STATUS EPILEPTICUS CONTROLLED WITH LEVETIRACETAM
Hasan TEKGUL (Izmir, Turkey)

G5-03
15:06-15:14
HIGH DOSE INTRAVENOSIS LEVATIRACETAM IN EARLY MYOKLONIC ENCEPHALOPATHY DUE TO NON KETOTIC HIPERGLYCINEMIA
Orkide GUZEL (Izmir, Turkey)

G5-04
15:14-15:22
VARIATION OF THERAPEUTIC HYPOTHERMIA PRACTICES IN U.S. NEONATAL INTENSIVE CARE UNITS: A NATIONAL SURVEY
Marc ELLSWORTH (Rochester, USA)

G5-05
15:22-15:30
EFFECTS OF LAMOTRIGINE ON A PATIENT WITH EARLY MYOCLONIC ENCEPHALOPATHY
Takahito INOUE (Fukuoka, Japan)

Chairpersons: Phillip L. Pearl (Washing ton DC, USA)**, Yoshiaki SAITO** (Tokyo, Japan)

G6-01
14:50-14:58
THE EFFICACY OF TOPIRAMATE IN TREATMENT OF INFANTILE SPASMS
Mariya Ilyasovna SIGATULLINA (Tashkent, Uzbekistan)

G6-02
14:58-15:06
EFFECT OF ANTI EPILEPTIC THERAPY ON SERUM HOMOCYSTEINE IN CHILDREN
Praveen KISHORE (New Delhi, India)

G6-03
15:06-15:14
VAGUS NERVE STIMULATION IN PEDIATRIC REFRACTORY EPILEPSY MANAGEMENT (EXPERIENCE OF A METROPOLITAN TEACHING HOSPITAL IN TAIWAN)
Chuan-Yu WANG (Taipei, Taiwan)

G6-04
15:14-15:22
SIDE EFFECTS OF CHRONIC VAGUS NERVE STIMULATION FOR EPILEPSY IN CHILDREN
Batwala PHILIP (Kampala, Uganda)

G6-05

15:22-15:30

EVALUATION OF DIETARY THERAPIES (CLASSIC KETOGENIC AND MODIFIED ATKINS DIET) IN VIGABATRIN-RESISTANT INFANTILE SPASMS

Suvasini SHARMA (New Delhi, India)

G6-06

15:30-15:38

FAVOURABLE SEIZURE CONTROL WITH KETOGENIC DIET FOR TWO CHILDREN WITH MIGRATING PARTIAL SEIZURES OF INFANCY

Sangita Darshini TERUMALAY (Kuala Lumpur, Malaysia)

Profile of Lecturers

L 01

Kazuyoshi WATANABE

Present Position

Professor Emeritus, Nagoya University Graduate School of Medicine, Aichi, Japan
Professor, Faculty of Health and Medical Science, Aichi Shukutoku University, Aichi, Japan

Education

1. 1963 Graduated from Nagoya University School of Medicine
2. 1963-1964 Internship at Nagoya University Hospital
3. 1964-1968 Nagoya University Graduate School of Medicine
4. 1968-1970 Resident, Department of Pediatrics, Nagoya University Hospital

Appointments

1970-1979 Chief, Department of Child Neurology, Central Hospital, Aichi Prefectural Colony
1979-1984 Assistant Professor, Department of Pediatrics, Nagoya University School of Medicine
1984-2002 Professor and Chairman, Department of Pediatrics, Nagoya University School of Medicine
2002 Professor emeritus, Nagoya University Graduate School of Medicine
2002-2010 Professor, Faculty of Medical Welfare, Aichi Shukutoku University
2010-Present Professor, Faculty of Health and Medical Science, Aichi Shukutoku University

Selected Publications

1. Watanabe K, Hara K, Miyazaki S, Kuroyanagi M, Asano S, Kondo K, Kuno K, Jose H, Iwase K. Electroclinical studies of seizures in the newborn. Folia Psychiatr Neurol Jpn 1977; 31: 383-392.
2. Watanabe K, Hara K, Miyazaki S, Hakamada S. Neurophysiological study of newborns with hypocalcemia. Neuropediatrics 1982; 13: 34-38.
3. Watanabe K, Hara K, Miyazaki S, Hakamada S, Kuroyanagi M. Apneic seizures in the newborn. Am J Dis Child 1982; 136: 980-984.
4. Watanabe K. The neonatal electroencephalogram and sleep cycle patterns. In: Eyre JA, editor. The Neurophysiological Examination of the Newborn Infant. London: Mac Keith; 1992. P.11-47.
5. Watanabe K. Introduction to Neonatal Electroencephalography (in Japanese). Tokyo: Shinkou-igaku Publ. Co. 2002.

L 02

Toru KATO

Present Position

Pediatric Neurologist, Okazaki City Hospital, Okazaki, Japan

Education

1991 MD, Kanazawa University School of Medicine, Kanazawa, Japan
1991-1992 Resident, Chukyo Hospital, Nagoya, Japan

Appointments

1993 Pediatrician, Nagoya University Hospital, Nagoya, Japan
1994-2002 Pediatric Neurologist, Anjo Kosei Hospital, Anjo, Japan
2002-2003 Pediatric Neurologist, Nagoya University Hospital, Nagoya, Japan
2004-present Pediatric Neurologist, Okazaki City Hospital, Okazaki, Japan

Selected Publications

1. Kato T, Okumura A, Hayakawa F, Kuno K, Watanabe K. Electroencephalographic aspects of periventricular hemorrhagic infarction in preterm infants. Neuropediatrics 2004; 35: 161-6.
2. Kato T, Okumura A, Hayakawa F, Kuno K, Watanabe K. The evolutionary change of flash visual evoked potentials in preterm infants with periventricular leukomalacia. Clin Neurophysiol 2005; 116: 690-5.
3. Kato T, Watanabe K. Visual evoked potential in the newborn: does it have predictive value? Semin Fetal Neonatal Med 2006; 11: 459-63.
4. Kato T, Hayakawa F, Tsuji T, Natsume J, Okumura A. Early diffusion-weighted images in infants with subcortical leukomalacia. Pediatr Neurol 2010; 42: 375-9.
5. Kato T, Okumura A, Hayakawa F, Tsuji T, Hayashi S, Kubota T, Fukasawa T, Suzuki M, Maruyama K, Oshiro M, Hattori T, Kidokoro H, Natsume J, Hayakawa M, Watanabe K. Prolonged EEG depression in term and near-term infants with hypoxic ischemic encephalopathy and later development of West syndrome. Epilepsia 2010; 51: 2392-6.

L 03

Lena HELLSTRÖM-WESTAS

Present Position

Professor of Perinatal Medicine, Department of Women's and Children's Health, Uppsala University, Uppsala, Sweden

Education

1979 MD, Karolinska Institutet Stockholm, Sweden
1990 PhD, Lund University, Lund, Sweden

Appointments

1996	Associate Professor, Lund University
1998	Senior Consultant in Neonatology, NICU, Lund University Hospital
2003-2004	Director of Neonatology, Queen Silvia Children's Hospital, Göteborg
2006-present	Scientific advisor in Neonatology, National Board of Health and Welfare
2007	Senior lecturer, Uppsala University
2010	Professor of Perinatal Medicine, Department of Women's and Children's Health, Uppsala, Sweden

Selected Publications

1. Hellström-Westas L, Rosén I, Svenningsen NW. Silent seizures in sick infants in early life. Acta Paediatr Scand 1985; 74: 741-8.
2. Hellström-Westas L, Rosén I, Svenningsen NW. Cerebral function monitoring during the first week of life in extremely small low birthweight (ESLBW) infants. Neuropediatrics 1991; 22: 27-32.
3. Hellström-Westas L, Blennow G, Lindroth M, Rosén I, Svenningsen NW. Low risk of seizure recurrence after early withdrawal of antiepileptic treatment in the neonatal period. Arch Dis Child 1995; 72: F97-101.
4. Toet MC, Hellström-Westas L, Groenendaal F, Eken P, de Vries LS. Amplitude integrated EEG at 3 and 6 hours after birth in fullterm neonates with hypoxic ischaemic encephalopathy. Arch Dis Child 1999; 81: F19-23.
5. Hellström-Westas L, de Vries LS, Rosén I. An Atlas of Amplitude-Integrated EEG's in the Newborn. 2nd revised edition. Informa Healthcare: London, UK, 2008, pp 1-187.

L 04

Federico VIGEVANO

Current Position and Affilication

Head of Neurosciences and Neurorehabilitation Department
Ospedale Pediatrico Bambino Gesù, Scientific Institute, Rome, Italy

Education

1974 Degree in Medicine and Surgery at University of Rome.
1977 Specialization in Neurology and Psychiatry at University of Rome.
1977 Postgraduate course in Neurophysiology at University of Marseille, France.

Appointments

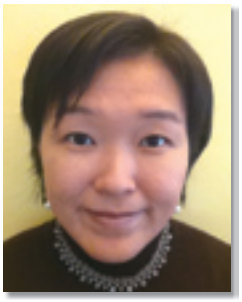
1982 Head, Section of Neurophysiology, Bambino Gesù Children's Hospital, Rome
1994 Professor of Neurology; Department of Neurological Sciences, University of Rome "La Sapienza", Italy
1996 Secretary of Italian League Against Epilepsy
1997 Head, Neurology Division, Bambino Gesù Children's Hospital, Rome
1999 Head of Italian League Against Epilepsy
2001 Professor of Neurology, Department of Paediatrics, University of Rome "La Sapienza", Italy.
2001 Professor of Neurology, Department of Infantile Neuropsychiatry, Catholic University of Rome, Italy.
2001 Chair of European Advisory Council of ILAE
2010 Head of Neurosciences and Neurorehabilitation Department

Selected Publications

1. Vigevano F. Benign familial infantile seizures. Brain Dev 2005; 27(3): 172-7
2. Ferrie C, Caraballo R, Covanis A, Demirbilek V, Dervent A, Kivity S, Koutroumanidis M, Martinovic Z, Oguni H, Verrotti A, Vigevano F, Watanabe K, Yalcin D, Yoshinaga H. Panayiotopoulos syndrome: a consensus view. Dev Med Child Neurol 2006; 48(3): 236-40
3. Specchio N, Fusco L, Claps D, Vigevano F. Epileptic encephalopathy in children possibly related to immune-mediated pathogenesis. Brain Dev 2010; 32(1): 51-6.
4. PRRT2 is mutated in familial and non-familial benign infantile seizures. Specchio N, Terracciano A, Trivisano M, Cappelletti S, Claps D, Travaglini L, Cusmai R, Marras CE, Zara F, Fusco L, Bertini E, Vigevano F. Eur J Paediatr Neurol. 2013 Jan;17(1):77-81.
5. Specchio N, Terracciano A, Trivisano M, Cappelletti S, Claps D, Travaglini L, Cusmai R, Marras CE, Zara F, Fusco L, Bertini E, Vigevano F. PRRT2 is mutated in familial and non-familial benign infantile seizures. Eur J Paediatr Neurol. 2013 Jan;17(1):77-81.

L 05

Emi TAKAHASHI



Present Position

Boston Children's Hospital Harvard Medical School Boston, MA, USA

Education

1998 Hubrecht Laboratory, The Institute for Developmental Biology, Utrecht, Netherlands (Visiting student)
1997 –2000 Chiba University School of Medicine, Chiba, Japan
2000 Nuclear Reprogramming Laboratory, Roslin Institute, Edinburgh, UK (Visiting student)
2000 –2002 Physiology University of Tokyo, Graduate School of Medicine, Department of Physiology, Tokyo, Japan
2003 Ph.D. in Neuroscience (Thesis advisor: Dr. Yasushi Miyashita) Chiba University Graduate School of Medicine, Chiba, Japan
2004 –2006 Postdoctoral Associate, Department of Anatomy and Neurobiology, Boston University School of Medicine
2006 –2007 Fellow, Uehara Memorial Foundation, Japan Athinoula A. Martinos Center for Biomedical Imaging
2007 –2009 Research Fellow, Department of Radiology, Harvard Medical School Massachusetts General Hospital, Athinoula A. Martinos Center for Biomedical Imaging
2009 –2010 Research Scholar, Division of Newborn Medicine, Department of Medicine, Boston Children's Hospital, Harvard Medical School Fetal-Neonatal Neuroimaging & Developmental Science Center

Appointments

2007 –2009 Research Fellow Department of Radiology, Massachusetts General Hospital
2009 –2010 Research Scholar Division of Newborn Medicine, Department of Medicine, Boston Children's Hospital
2010 – present Instructor in Pediatrics, Harvard Medical School
2010 – present Associate Scientific Research Stuff Division of Newborn Medicine, Department of Medicine, Boston Children's Hospital

Selected Publications

1. Takahashi E, Folkerth RD, Galaburda AL, Grant PE. Emerging cerebral connectivity in the human fetal brain: An MR tractography study. Cerebral Cortex 2012. 22; 455-464.
2. Takahashi E, Dai G, Wang R, Ohki K, Rosen GD, Galaburda AL, Grant PE, Wedeen VJ. Development of cerebral fiber pathways in cats revealed by diffusion spectrum imaging. Neuroimage 2010. 49; 1231-1240.
3. Takahashi E, Ohki K, Kim DS. Dissociation and convergence of the dorsal and ventral visual streams in the human prefrontal cortex. Neuroimage 2013. 65:488-498.
4. Takahashi E, Song JW, Folkerth RD, Grant PE, Schmahmann JD. Detection of cerebellar cortex and white matter pathways using high angular resolution diffusion tractography. Neuroimage 2013. 68: 105–111.
5. Xu G*, Takahashi E*, Folkerth RD, Haynes RL, Volpe JJ, Grant PE, Kinney HC. Radial coherence of diffusion tractography in the cerebral white matter of the human fetus: Neuroanatomic insights. *co-first authors. Cerebral Cortex 2012.

L 06

Jeffrey J. NEIL

Present Position

Professor of Neurology, Pediatrics and Radiology, St. Louis Childrens's Hospital,
Washington University School of Medicine, St. Louis, MO, USA

Education

1977 B.A., Washington University, St. Louis, Biology
1984 M.D., Washington University, St. Louis, Medicine
1984 Ph.D., Washington University, St. Louis, Neurobiology

Appointments

1986-1990 Fellow in Pediatric Neurology at St. Louis Children's Hospital, St. Louis, MO
1990-1992 Instructor in Pediatrics and Neurology, ditto
1992-1999 Assistant Professor of Neurology and Pediatrics, ditto
1999-2004 Associate Professor of Neurology, Pediatrics and Radiology, St. Louis Childrens's Hospital, Washington University School of Medicine, St. Louis, MO.
2004-present Professor of Neurology, Pediatrics and Radiology, ditto

Selected Publications

1. Neil JJ, Shiran SI, McKinstry RC, Schefft G, Snyder AZ, Almli CR, et al. Normal brain in human newborns: Apparent diffusion coefficient and diffusion anisotropy measured using diffusion tensor MR imaging. Radiology 1998; 209: 57-66.
2. McKinstryRC, Miller JH, Snyder AZ, Schefft GL, Mathur A, Almli CR, Akbudak E, Conturo TE, Shiran SI, Neil JJ. A prospective, longitudinal diffusion tensor imaging study of brain injury in newborns. Neurology 2002; 59: 824-33.
3. McKinstry RC, MD, Mathur A, Miller JH, Ozcan A, SnyderAZ, Schefft GL, Almli CR, Shiran SI, Conturo TE, Neil JJ. Radial organization of developing human cerebral cortex revealed by non-invasive water diffusion anisotropy MRI. Cerebral Cortex 2002; 12: 1237-43.
4. Kroenke CD, Bretthorst GL, Inder TE, Neil JJ. Diffusion MR imaging characteristics of the developing primate brain. NeuroImage 2005; 25: 1205-19.
5. Smyser CD, Inder TE, Shimony JS, Hill JE, Degnan AJ, Snyder AZ, Neil JJ. Longitudinal analysis of neural network development in preterm infants. Cerebral Cortex 2010; 20: 2852-62. PMC2978240

L 07

Terrie E. INDER

Present Position

Professor in Pediatrics, Radiology and Neurology, St Louis Childrens Hospital, Washington University in St Louis, MO, USA

Education

MBChB University of Otago, New Zealand
MD University of Otago, New Zealand

Appointments

2010-	current Professor of Pediatrics, Neurology and Radiology, St. Louis Children's Hospital, Washington University, St Louis
2006-2010	Associate Professor of Pediatrics, Neurology and Radiology, St. Louis Children's Hospital, Washington University, St Louis
2001-2005	Associate Professor of Pediatrics, Royal Children's Hospital, Melbourne, Australia
1999-2000	Senior Research Fellow in Pediatrics, Christchurch School of Medicine, New Zealand
1996-1999	Fellow in Pediatric Neurology at Boston Children's Hospital, Harvard Medical School
1989-1996	General Pediatric Residency and Neonatal Fellowship, University of Otago, New Zealand
1983-1989	University of Otago completing Bachelor of Medicine and Surgery (top graduate in year).

Selected Publications

1. Woodward LJ, Clark CA, Bora S, Inder TE. Neonatal White Matter Abnormalities an Important Predictor of Neurocognitive Outcome for very Preterm Children. PLOS One 2012;7(12):e51879.doi: 10.1371 PMID:23284800
2. Smith G, Gutovich J, Smyser C, Pineda R, Newnham C, Tjoeng T, Vavasseur C, Wallendorf M, Neil J, Inder T. Neonatal Intensive Care Unit Stress is Associated with Brain Development in Preterm Infants. Annals of Neurology 2011;70(4):541-9 PMID:21976396
3. Shah DK, Mackay MT, Lavery S, Watson S, Harvey S, Zempel J, Mathur A, Inder TE. The Accuracy of Bedside EEG Monitoring for Seizure Detection in Term Infants. Pediatrics 2008;121(6):1146-54. PMID: 18519484
4. Woodward LJ, Anderson PJ, Austin NC, Howard K, Inder TE. Cerebral Abnormalities on Neonatal Magnetic Resonance Imaging and Neurodevelopmental Outcomes in Preterm Infants. New England Journal of Medicine 2006; 355(7): 685-694. PMID: 16914704
5. Inder TE, Warfield SK, Wang HX, Huppi PS, Volpe JJ. Abnormal Cerebral Structure at Term in Premature Infants. Pediatrics 2005 115(2):286-294. PMID: 15687434

L 08

Sampsa Kullervo VANHATALO

Present Position

1a-Clinical Research Fellow, Finnish Academy, 2009-2010
1b-Head of Pediatric Clinical Neurophysiology, University Hospital of Helsinki, Finland
2- Docent (Ass.prof) in Clinical Neurophysiology, University of Helsinki, Finland

Education

1995 Doctoral Thesis
1998 MD and PhD degrees, University of Helsinki
2001-3 Postdoctoral Research Training, University of Washington, Seattle, WA, USA

Appointments

1993-2000 Research Associate (Department of Anatomy, University of Helsinki)
1999 Docent (Ass prof) in neurobiology, University of Helsinki
2000-01 Dept.of Pediatrics, Univ.Hospital of Helsinki, 6mo
2004- Dept. of Clin.Neurophys., Univ.Hospital of Helsinki
2009 Docent (Ass prof) in Clin.Neurophysiology, University of Helsinki

Selected Publications

1. Vanhatalo S, Palva M, Andersson S, Rivera C, Voipio J, Kaila K. Slow endogenous activity transients and developmental expression of K-Cl cotransporter 2 in the immature human cortex. Eur J Neurosci 2005; 22: 2799-804.
2. Vanhatalo S, Jousmäki V, Andersson S, Metsäranta M. An easy and practical method for routine, bedside testing of somatosensory systems in extremely low birth weight infants (ELBW). Pediatr Res 2009; 66: 710-.
3. Vanhatalo S, Kaila K. Spontaneous and evoked activity in the early human brain. In: Lagercrantz H, Hanson MA, Ment LR, Peebles DM, editors. The Newborn Brain: Neuroscience & Clinical Applications, 2nd edition. Cambridge University Press, 2009, Chapter 15, 229-243.
4. Stjerna, S., Voipio, J., Metsäranta, M., Kaila, K., Vanhatalo, S., 2012. Preterm EEG: A Multimodal Neurophysiological Protocol. J. Vis. Exp., e3774, DOI : 10.3791/3774.
5. Tokariev A, Palmu K, Lano A, Metsäranta M, Vanhatalo S. Phase synchrony in the early preterm EEG: Development of methods for estimating synchrony in both oscillations and events. Neuroimage, 2012, 60:1562-1573.

L 09

Akihisa OKUMURA

Present Position

Associate Professor, Department of Pediatrics, Juntendo University School of Medicine, Tokyo, Japan

Education

1989 Graduated from Nagoya University School of Medicine, Nagoya, Japan
1989-1990 Resident at Tokai Chuo Hospital
1991-1992 Department of Pediatrics, Nagoya University Hospital

Appointments

1992-1998 Department of Pediatrics, Anjo Kosei Hospital
1998-2005 Assistant, Department of Pediatrics, Nagoya University Graduate School of Medicine, Nagoya, Japan
2005-2006 Assistant Professor, Department of Pediatrics, Nagoya University Graduate School of Medicine
2006-present Associate Professor, Department of Pediatrics, Juntendo University School of Medicine, Tokyo, Japan

Selected Publications

1. Okumura A, Komatsu M, Abe S, Kitamura T, Matsui K, Ikeno M, Shimizu T. Amplitude-integrated electroencephalography in patients with acute encephalopathy with refractory, repetitive partial seizures. Brain Dev 2011; 33: 77-82.
2. Okumura A, Yamamoto T, Kidokoro H, Kato T, Kubota T, Shoji H, Sato H, Shimojima K, Shimizu T. Altered gene expression in umbilical cord mononuclear cells in preterm infants with periventricular leukomalacia. Early Hum Dev 2010; 86: 665-7.
3. Okumura A, Kidokoro H, Shoji H, Nakazawa T, Mimaki M, Fujii K, Oba H, Shimizu T. Kernicterus in preterm infants. Pediatrics 2009; 123: e1052-8.
4. Okumura A, Suzuki M, Kubota T, Hisada K, Kidokoro H, Kato T, Hayakawa F, Shimizu T.West syndrome in extremely preterm infants: its relation to postnatal events. Arch Dis Child Fetal Neonatal Ed 2008; 93: F326-7.
5. Okumura A, Hayakawa F, Kato T, Itomi K, Maruyama K, Kubota T, Suzuki M, Kidokoro H, Watanabe K. Ictal electroencephalographic findings of neonatal seizures in preterm infants. Brain Dev 2008; 30: 261-8.

L 10

Geraldine B. BOYLAN

Present Position

Senior Lecturer and Clinical Scientist in the Department of Paediatrics & Child Health, School of Medicine, University College Cork, and Director of the Neonatal Research Centre at Cork University Maternity Hospital, Cork, Ireland

Education

2008 MA: University College Cork, Ireland
2002 PhD: University of London, UK
1991 MSc: Physiology, University of London, UK
1989 BSc: Physiology, University of London, UK
1987 Higher Diploma in Medical Physics & Physiological Measurement, City of Westminster College, London, UK.

Appointments

2006 - Present Senior Lecturer in Medical Education & Paediatrics, University College Cork
2004 - 2006 Lecturer in Paediatrics & Child Health, University College Cork
2002 - 2004 Senior Research Fellow, Dept. of Paediatrics & Child Health, University College Cork
1996 - 2002 Clinical Scientist, Neonatal Intensive Care Unit, Kings College Hospital, London, UK
1987 - 1996 Chief Clinical Physiologist, Middlesex Hospital, London, UK

Selected Publications

1. Temko A, Thomas E, Marnane W, Lightbody G, Boylan G. EEG-based neonatal seizure detection with Support Vector Machines. Clin Neurophysiol 2011; 122(3): 464-73.
2. Temko A, Thomas E, Marnane W, Lightbody G, Boylan GB. Performance assessment for EEG-based neonatal seizure detectors. Clin Neurophysiol 2011; 122(3): 474-82.
3. Walsh BH, Low E, Bogue CO, Murray DM, Boylan GB. Early continuous video electroencephalography in neonatal stroke. Dev Med Child Neurol. 2011; 53(1): 89-92.
4. Thomas EM, Temko A, Lightbody G, Marnane WP, Boylan GB. Gaussian mixture models for classification of neonatal seizures using EEG. Physiol Meas. 2010; 31(7): 1047-64.
5. Murray DM, Bala P, O'Connor CM, Ryan CA, Connolly S, Boylan GB. The predictive value of early neurological examination in neonatal hypoxic-ischaemic encephalopathy and neurodevelopmental outcome at 24 months. Dev Med Child Neurol 2010; 52(2): 1-9.

L 11 L 18

Phillip Lawrence PEARL

Present Position

1. Professor of Pediatrics, Neurology, and Music, Director of Medical Student Education, Dept Neurology, The George Washington University School of Medicine and Columbian College of Arts and Sciences, Washington, D.C., USA
2. Division Chief, Child Neurology and Neurodevelopmental Disabilities, Director of Education, Department of Neurology, Member, Children's Research Institute, Children's National Medical Center, Washington, D.C., USA
3. Clinical Epilepsy Branch, Office of the Clinical Director, Division of Intramural Research, National Institute of Neurological Disorders and Stroke, National Institutes of Health, Bethesda, Maryland, USA

Education

1984	University of Maryland School of Medicine, Baltimore, Maryland
1984-1986	Pediatric Internship and Residency, Baylor College of Medicine, Houston, TX
1986-1989	Neurology and Child Neurology, Baylor College of Medicine, Houston, TX
1989-1990	Fellowship in Clinical Neurophysiology, Children's Hospital, Beth Israel Hospital, Harvard Medical School, Boston, MA
2003	Graduate Certificate in Leadership Development, Graduate School of Education and Human Development, George Washington University, Washington, DC

Honors

2012-Present	Professors of Child Neurology, President
2009-Present	ACGME Neurology Residency Review Committee
2008	Michael T. Ty Memorial Lecturer, Massachusetts General Hospital, Department of Neurology, Boston
2006	American Neurological Association
2006	American Pediatric Society

Selected Publications

1. Pearl PL and Yuezhou, Y. Inherited Pediatric Metabolic Epilepsies. Exp Opinion Orphan Drugs. Epub 8 Jan 2013.
2. Pearl PL, Hyland K, Chiles J, McGavin C, Yuezhu Y, Taylor D: Partial Pyridoxine Responsiveness in PNPO Deficiency. J Inherit Metab Dis: in press.
3. Freilich ER, Jones JM, Gaillard WD, Conry JA, Tsuchida TN, Reyes C, Dib-Hajj S, Waxman SG, Meisler MH, Pearl PL: Novel SCN1A Mutation in a Proband With Malignant Migrating Partial Seizures of Infancy. Arch Neurol 2011; 68:650-671.
4. Pearl PL: Neurological problems of jazz legends. J Child Neurol 2009; 24:1037-1042.
5. Pearl PL: New Treatment Paradigms in Neonatal Seizures. J Inherit Metab Dis 2009; 32:204-213.

L 12

Adam L. HARTMAN

Present Position

Assistant Professor of Neurology, Pediatrics, and Molecular Microbiology and Immunology, Johns Hopkins Hospital, Baltimore, USA

Education

1985-1989 Bachelor of Arts, Northwestern University, Evanston, IL, Chemistry
1989-1994 Doctor of Medicine, Northwestern University, Chicago, IL
1994-1995 Internship, National Naval Medical Center, Bethesda, MD, Pediatrics
1995-1997 Residency, National Capital Uniformed Services Residency Program, National Naval Medical Center, Bethesda, MD and Walter Reed Army Medical Center, Washington, DC, Pediatrics
2002-2005 Residency, Johns Hopkins Hospital, Baltimore, MD, Pediatric Neurology
2005-2007 Fellowship, Johns Hopkins Hospital, Baltimore, MD, Clinical Neurophysiology (Pediatric Epilepsy)
2009 Statistics for Laboratory Scientists I & II (Fall & Spring Semesters), Department of Biostatistics, Johns Hopkins Bloomberg School of Public Health, Baltimore, MD
July, 2009 Molecular Biology Summer Workshop, Smith College, Northampton, MA

Appointments

Assistant Professor of Neurology and Pediatrics (July 2007 - present)
Assistant Professor of Molecular Microbiology and Immunology (July 2012 - present)
Johns Hopkins University School of Medicine, Baltimore, MD (July 2007 - present)
Attending Physician, Johns Hopkins Hospital, Baltimore, MD (July 2007 - present)
Active Staff, Johns Hopkins Bayview Medical Center, Baltimore, MD (July 2007 - present)

Selected Publications

1. Gasior M, French A, Joy M, Tang R, Hartman AL, M. A. Rogawski. The Anticonvulsant Activity of Acetone, the Major Ketone Body in the Ketogenic Diet, Is Not Dependent on Its Metabolites Acetol, 1,2-Propanediol, Methylglyoxal or Pyruvic Acid. Epilepsia 2007;48:793-800.
2. Hartman AL, Lyle M, Rogawski MA Gasior M. Efficacy of the Ketogenic Diet in the 6-Hz Seizure Test. Epilepsia 2008;49:334-339.
3. Hartman AL, Zheng XR, Bergbower E, Kennedy M, Hardwick JM. Seizure tests distinguish intermittent fasting from the ketogenic diet. Epilepsia 2010;51:1395-1402.
4. Gasior M, Yankura J, Hartman AL, French A, Rogawski MA. Anticonvulsant and proconvulsant actions of 2-deoxy-d-glucose. Epilepsia 2010;51:1385-1394.
5. Hartman AL, Santos P, Dolce, A, Hardwick JM. The mTOR inhibitor rapamycin has limited acute anticonvulsant effects in mice. PLoS One 2012;7:e45156.

L 13

Perrine PLOUIN

Present Position

Head, Department of Clinical Neurophysiology, Necker Enfants Malades Hospital, Paris, France

Education

1963 Baccalauréat, Paris University
1971 M.D., Paris University
1971 Master in Physiology, Paris University

Appointments

1968-73 Lecturer in Physiology, Paris University
1973-81 Practicing in Clinical Neurophysiology, different hospitals in Paris
1981-2003 Head, Department of Clinical Neurophysiology, Saint-Vincent-de-Paul Hospital, Paris
2003-2011 Head, Department of Clinical Neurophysiology, Necker Enfants Malades Hospital, Paris

Selected Publications

1. Coppola G, Plouin P, Chiron C, Robain O, Dulac O. Migrating partial seizures in infancy: a malignant disorder with developmental arrest. Epilepsia 1995; 36: 1017-24.
2. Lamblin MD, Andre M, Challamel MJ, Curzi-Dascalova L, d'Allest AM, De Giovanni E, Moussalli-Salefranque F, Navelet Y, Plouin P, Radvanyi-Bouvet MF, Samson Dollfus D, Vecchierini-Blineau MF. Electroencephalography of the premature and term newborn. Maturational aspects and glossary. Neurophysiol Clin 1999: 29: 123-219.
3. Mikaeloff Y, Jambaque I, Hertz-Pannier L, Zamfirescu A, Adamsbaum C, Plouin P, Dulac O, Chiron C. Epilepsy Res 2006; 69(1): 67-79.
4. Co JPT, Elia M, Engel J Jr., Guerrini R, Mizrahi EM, Moshe SL, Plouin P. Proposal of an algorithm for diagnosis and treatment of neonatal seizures in developing countries. Epilepsia; 2007: 48(6): 1158-64.
5. Velis D, Plouin P, Gotman J, da Silva FL; ILAE DMC Subcommittee on Neurophysiology. Recommendations regarding the requirements and applications for long-term recordings in epilepsy. Epilepsia 2007; 48(2): 379-84.

L 14

Hannah Cranley GLASS

Present Position

Assistant Professor of Neurology, Step II, Neurology & Pediatrics, Department of Neurology, University of California, San Francisco, CA, USA

Education

1991-95 Columbia College of Columbia University, New York, NY. BA(honors), Magna cum laude, Biology/Psychology

1997-2001 McGill University, Montréal, Québec, Canada Doctor of Medicine and Master of Surgery (MDCM)

2001-05 Resident, Child Neurology, University of Calgary, Alberta, Canada

2005-06 Chief Resident, Child Neurology, University of Calgary, Alberta, Canada

2006-08 University of California, San Francisco, CA, USA Master of Advanced Studies (MAS), Training in Clinical Research

Principle Positions Held

2008- University of California, San Francisco
Assistant Professor, Neurology & Pediatrics

2008- University of California, San Francisco
Co-Director, Neurological Intensive Care Nursery

Selected Publications

1. Glass HC, Bonifacio SL, Chau V, Glidden D, Poskitt K, Barkovich AJ, Ferriero DM, Miller SP. Recurrent postnatal infection is associated with progressive white matter injury in premature infants. Pediatrics 2008; 122(2): 299-305.
2. Glass HC, Pham TN, Danielson B, Towner D, Glidden D, Wu Y. Antenatal and intrapartum risk factors for neonatal seizures in term infants: A population-based study, California 1998-2002. J Pediatr 2009; 154(1): 24-28. e1.
3. Glass HC, Glidden D, Jeremy RJ, Barkovich AJ, Ferriero DM, Miller SP. Clinical neonatal seizures are independently associated with outcome in infants at risk for hypoxic-ischemic brain injury. J Pediatr 2009: 155(3): 318-23.
4. Glass HC, Bonifacio SL, Sullivan J, Rogers E, Ferriero DM, Goldstein R, Barkovich AJ. Magnetic resonance imaging and ultrasound injury in preterm infants with seizures. J Child Neurol 2009; 24(9): 1105-11.
5. Glass HC, Bonifacio SL, Peloquin S, Shimotake T, Sehring S, Sun Y, Sullivan J, Rogers E, Barkovich AJ, Rowitch D, Ferriero DM. Neurocritical care for neonates. Neurocrit Care 2010; 12(3): 421-9.

L 15

Hiroyuki KIDOKORO

Present Position

Department of Pediatrics, Nagoya University Graduate School of Medicine, Nagoya, Japan

Education

2009-2010 Research fellow, Department of Pediatrics, Washington University in St. Louis, St. Louis, USA
2007-2010 PhD degree, Nagoya University Graduate School of Medicine, Nagoya, Japan
2002-2003 Resident, Division of Neonatology, Saitama Children's Medical Center, Saitama, Japan.
1998-2000 Resident, Anjo Kosei Hospital, Anjo, Japan
1992-1998 Doctor of Medicine, Nagoya University Graduate School of Medicine, Nagoya, Japan

Appointments

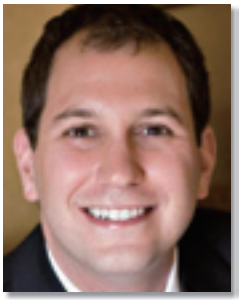
2006-2009 Chief pediatric neurologist, Division of Pediatrics, Anjo Kosei Hospital, Anjo, Japan
2005-2006 Chief , Division of Pediatrics, Kasugai Municipal Hospital, Kasugai, Japan
2004-2005 Clinical staff, Division of Neonatology, Saitama Children's Medical Center, Saitama, Japan.

Selected Publications

1. Kidokoro H, Okumura A, Watanabe K. Abnormal brushes in preterm infants with periventricular leukomalacia. Neuropediatrics 2006; 37(5): 265-8.
2. Kidokoro H, Kubota T, Ohe H, Hattori T, Kato Y, Miyajima Y, Ogawa A, Okumura A, Watanabe K, Kojima S. Diffusion-weighted magnetic resonance imaging in infants with periventricular leukomalacia. Neuropediatrics 2008; 39(4): 233-8.
3. Kidokoro H, Okumura A, Kato T, Hayakawa F, Natsume J, Kubota T, Watanabe K, Kojima S. Electroencephalogram and flash visual evoked potentials for detecting periventricular leukomalacia. Neuropediatrics 2008; 39(4): 226-32.
4. Kidokoro H, Okumura A, Hayakawa F, Kato T, Maruyama K, Kubota T, Suzuki M, Natsume J, Watanabe K, Kojima S. Chronologic Changes in Neonatal EEG Findings in Periventricular Leukomalacia. Pediatrics 2009; 124(3): e468-75.
5. Kidokoro H, et al. Absent Cyclicity on aEEG within the first 24 h is associated with brain damage in preterm infants. Neuropediatrics 2011 (in press).

L 16 L 30

Nicholas Scott ABEND



■ Present Position

Medical Director, Clinical Neurophysiology, The Children's Hospital of Philadelphia PA, USA

■ Education

2007-2009 The Children's Hospital of Philadelphia, Epilepsy and Neurophysiology Fellow
2007-2008 Clinical Research Certificate Program, Center for Epidemiology and Biostatistics, The University of Pennsylvania School of Medicine
2004-2007 Hospital of the University of Pennsylvania & Children's Hospital of Philadelphia, Neurology Resident
2002-2004 The University of Chicago Children's Hospital, Pediatrics Residency
1998-2002 The University of Chicago, Pritzker School of Medicine
1994-1998 Washington University (Major: Biology Minor: Psychology) B.S., May 1998

■ Appointments

10/09-present Assistant Professor of Neurology & Pediatrics, University of Pennsylvania Perelman School of Medicine
7/09-present Attending Physician, Division of Neurology, The Children's Hospital of Philadelphia

■ Selected Publications

1. Wusthoff CJ, Dlugos DJ, Guteirrez-Colina AM, Wang A, Cook N, Donnelly M, Clancy RR, Abend NS. Incidence of electrographic seizures during therapeutic hypothermia for neonatal encephalopathy. J Child Neurol (in press).
2. Abend NS, Guteirrez-Colina AM, Monk HM, Dlugos DJ, Clancy RR. Levetiracetam for treatment of neonatal seizures. J Child Neurol (in press).
3. Abend NS, Gutierrez-Colina AM, Topjian AA, Zhao H, Guo R, Donnelly M, Clancy RR, Dlugos DJ. Non-convulsive seizures are common in critically ill children. Neurology. (in press).
4. Abend NS, Topjian AA, Gutierrez-Colina AM, Donnelly M, Clancy RR, Dlugos DJ. Impact of Continuous EEG Monitoring on Clinical Management in Critically Ill Children. Neurocritical Care. (in press).
5. Abend NS, Topjian A, Ichord R, Herman ST, Helfaer M, Donnelly M, Nadkarni V, Dlugos D, Clancy R. Electroencephalographic monitoring during hypothermia after pediatric cardiac arrest. Neurology 2009; 72(22) 1931-40.

L 17

Tetsuo KUBOTA

Present Position

Chief, Department of Pediatric Neurology, Anjo Kosei Hospital, Aichi, Japan

Education

1990-1996 Nagoya University School of Medicine
2000-2003 Ph.D., Pediatric Neurology, Nagoya University Graduate School of Medicine

Appointments

1996-1997 Intern, Anjo Kosei Hospital
1997-1998 Resident, Pediatrics, Anjo Kosei Hospital
1998-2000 Clinical fellow, Pediatrics, Anjo Kosei Hospital
2001-2003 Research fellow in Department of Sensory-Motor Integration, National Institute for Physiological Sciences
2003- Clinical fellow, Department of Pediatrics, Anjo Kosei Hospital

Selected Publications

1. Kubota T, Okumura A, Hayakawa F, Kato T, Itomi K, Kuno K, Watanabe K. Relation between the date of cyst formation observable on ultrasonography and the timing of injury determined by serial electroencephalography in preterm infants with periventricular leukomalacia. Brain Dev. 2001 Oct; 23(6): 390-394.
2. Kubota T, Okumura A, Hayakawa F, Kato T, Itomi K, Kuno K, Watanabe K. Combination of neonatal electroencephalogrpahy and ultrasonography: sensitive means of early diagnosis of periventricular leukomalacia. Brain Dev. 2002 Oct; 24(7): 698-702.
3. Kubota T, Kaneoke Y,Maruyama K, Watanabe K and Kakigi R. Temporal structure of the apparent motion perception: a magnetoencephalographic study. Neuro. Res. 2004 Jan48(1): 111-118.
4. Kubota T, Okumura T, Kato T, Kakizawa H, Kondo Y, Negoro T, Ochi T, Watanabe K. Symptomatic Localized-Related Epilepsy Associated with Periventricular Leukomalacia. Epilepsia, 2005 Feb; 46 s2: 11-14
5. Kubota T, Fukasawa T, Kitamura E, Magota M, Kato Y, Natsume J, Okumura A. Epileptic seizures induced by dexmedetomidine in a neonate. Brain Dev. 2013 Apr;35(4):360-2.

L 19

Roustem KHAZIPOV

Present Position

Director of Research, INMED/INSERM U901, Marseille, France &
Leading Scientist, Laboratory of Neurobiology, Kazan Federal University, Russia

Education

1998	Doctor of Science, Kazan Medical University, Russia
1988-1991	PhD (physiology) Kazan Medical University, Russia
1982-1988	MD (general medicine) Kazan Medical University, Russia
1978-1982	BS (physics) Kazan School in Mathematics and Physics, Russia

Appointments

1997- present	INSERM Researcher and Research director (INSERM U901, Marseille, France)
2011- present	Leading Scientist, Laboratory of Neurobiology (Kazan Federal University, Russia)
2003	Visiting scientist, Rutgers University (NJ), USA (G. Buzsaki)
2001-2002	Research Associate in Neurology, Children's Hospital (G.L. Holmes), Harvard Medical School, Boston, MA, USA
2002-2003	Research Associate, Department of Neurology (G.L. Holmes), Dartmouth Medical School, Hanover, NH, USA

Selected Publications

1. KHAZIPOV R., SIROTA A., LEINEKUGEL X, HOLMES G., BEN-ARI Y., BUZSAKI G. Early motor activity drives spindle-bursts in developing somatosensory cortex. Nature, 2004, 432: 758-761.
2. TYZIO R, COSSART R, KHALILOV I, MINLEBAEV M, HUBNER CA, REPRESA A, BEN ARI Y, KHAZIPOV R (2006) Maternal Oxytocin Triggers a Transient Inhibitory Switch in GABA Signaling in the Fetal Brain During Delivery Science. 314: 1788-1792.
3. COLONNESE M , A. KAMINSKA, M.MINLEBAEV, M. MILH, B. BLOEM, S. LESCURE, G. MORIETTE, C. CHIRON, Y. BEN-ARI & R. KHAZIPOV. (2010) A conserved switch in sensory processing prepares developing neocortex for vision. Neuron 67: 480-498.
4. MINLEBAEV M, COLONNESE M, TSINTSADZE T, SIROTA A, KHAZIPOV R. (2011) Early gamma oscillations synchronize developing thalamus and cortex. Science 334: 226-9.
5. COLONNESE M, KHAZIPOV R (2012) Spontaneous activity in developing sensory circuits: Implications for resting state fMRI. Neuroimage 62:2212-2221.

L 20

Atsuo FUKUDA

Present Position

Professor and Head
Department of Neurophysiology Hamamatsu University School of Medicine

Education

1977-1983, M.D.
Faculty of Medicine, Kyushu University, Fukuoka, Japan
1983-1985, Resident
Department of Gynecology and Obstetrics
Kyushu University Hospital, Fukuoka, Japan
1985-1989, Ph.D. (Advisor: Y. Oomura)
Department of Physiology
Graduate School of Medical Sciences, Kyushu University, Fukuoka, Japan

Appointments

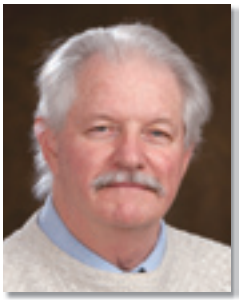
1989-1992, Postdoctoral Fellow (Mentor, D. A. Prince)
Department of Neurology and Neurological Sciences
Stanford University School of Medicine, Stanford, CA, USA
1993-1994, Assistant Professor; 1995-1998, Associate Professor
Department of Physiology, Nagoya City University Medical School, Nagoya, Japan
1998- , Professor and Chairman
Department of Neurophysiology, Hamamatsu University School of Medicine

Selected Publications

1. Fukuda, A. Diuretic soothes seizures in newborns. ***Nat. Med.*** 11: 1153-1154, 2005.
2. Saitsu, H., Kato, M., Mizuguchi, T., Hamada, K., Osaka, H., Tohyama, J., Uruno, K., Kumada, S., Nishiyaman, K., Nishimura, A., Okada, I., Yoshimura, Y., Hirai S-i., Kumada, T., Hayasaka, K., Fukuda, A., Ogata, K. and Matsumoto, N. De novo mutations in the gene encoding STXBP1 (MUNC18-1) cause early infantile epileptic encephalopathy. ***Nat. Genet.*** 40: 782-788, 2008.
3. Inoue, K., Furukawa, T., Kumada, T., Yamada, J., Wang, T., Inoue, R. and Fukuda A. Taurine inhibits the K+-Cl- cotransporter KCC2 to regulate embryonic Cl- homeostasis via the with-no-lysine (WNK) protein kinase signaling pathway. ***J. Biol. Chem.*** 287: 20839-20850, 2012.
4. Egawa, K., Kitagawa, K., Inoue, K., Takayama, M., Takayama, C., Saitoh, S., Kishino, T., Kitagawa, M. and Fukuda, A. Decreased tonic inhibition in cerebellar granule cells causes motor dysfunction in a mouse model of Angelman syndrome. ***Sci. Transl. Med.*** 4: 163ra157, 2012.
5. Wang, T., Kumada, T., Morishima, T., Iwata, S., Kaneko, T., Yanagawa, Y., Yoshida, S. and Fukuda, A. Accumulation of GABAergic neurons, causing a focal ambient GABA gradient, and downregulation of KCC2 are induced during microgyrus formation in a mouse model of polymicrogyria. ***Cereb. Cortex***: in press.

L 21

F. Edward DUDEK



Present Position

Professor and Vice-Chair for Research, Department of Neurosurgery University of Utah School of Medicine Salt Lake City, UT, USA

Education

1969 B.Sc., Biological Sciences, University of California, Irvine, CA, USA
1973 Ph.D., Dev. & Cell Biol. (Physiol.), University of California, Irvine, CA, USA

Appointments

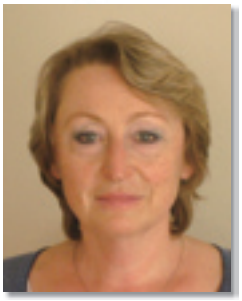
1980-84 Associate Professor, Dept. of Physiology, Tulane Univ. School of Medicine, New Orleans, LA, USA
1984-87 Professor, Dept. of Physiology, Tulane Univ. School of Medicine, New Orleans, LA, USA
1987-92 Professor, Mental Retardation Research Center, UCLA School of Medicine, Los Angeles, CA, USA
1992-05 Professor, Dept. of Anatomy & Neurobiol., Colorado State Univ., Fort Collins, CO (Chair: 7/92-6/00) , USA
2005-2012 Professor and Chair, Dept. of Physiology, Univ. of Utah School of Medicine, Salt Lake City, UT, USA
2012- Professor and Vice Chair for Research, Department of Neurosurgery, University of Present Utah School of Medicine, Salt Lake City, UT

Selected Publications

1. White AM, Williams PA, Hellier JL, Clark S, Dudek FE, Staley KJ. EEG spike activity precedes epilepsy after kainate-induced status epilepticus. Epilepsia 2010; 51: 371-83.
2. Kadam S, White AM, Staley KJ, Dudek FE. Continuous electroencephalographic monitoring with radio-telemetry in a rat model of perinatal hypoxia-ischemia reveals progressive post-stroke epilepsy. J Neurosci 2010; 30: 404-15.
3. Shao L-R, Dudek FE. Both synaptic and intrinsic mechanisms underlie the different properties of population bursts in the hippocampal CA3 area of immature versus adult rats. J Physiol 2009; 587(24): 5907-23.
4. Statler KD, Scheerlinck P, Pouliot W, Hamilton M, White HS, Dudek FE. A potential model of pediatric post-traumatic epilepsy. Epilepsy Res 2009; 86: 221-3.
5. Lehmkuhle MJ, Thomson KE, Scheerlinck P, Pouliot W, Greger B, Dudek FE. A simple quantitative method for analyzing electrographic status epilepticus in rats. J Neurophysiol 2009; 101: 1660-70.

L 22

Zena VEXLER



Present Position

Professor in Neurology, University of California SanFrancisco, CA, USA

Education

1979	Moscow State University, Dept. of Chemistry
1990	Institute of Biophysics, Ministry of Health
1990-1995	University of California, San Francisco, CA
1995-1996	University of California, San Francisco, CA

Appointments

1979-1981	Fellow Institute of Biophysics, Ministry of Health, USSR
1981-1990	Research Scientist Institute of Biophysics, Ministry of Health, USSR
1990-1995	Postgraduate Researcher University of California, San Francisco, CA
1995-1999	Assistant Researcher Neurobiologist University of California, San Francisco, CA
1999-2002	Assistant Professor University of California, San Francisco, CA
2002-2010	Associate Professor University of California, San Francisco, CA
2010-	Professor University of California, San Francisco, CA
2003-	Present Director of Research, Neonatal Brain Disorders Center University of California, San Francisco, CA
2008-	Member, Biomedical Sciences Graduate Program University of California, San Francisco, CA

Selected Publications

1. Wendland M, Faustino J, West T, Holtzman D, and Vexler ZS "Early diffusion-weighted MRI as predictor of caspase-3 activation following hypoxia-ischemia in neonatal rodents". Stroke, 2008, 39:1862-1868. PMID: 18420950
2. Shimotake J, Derugin N, Wendland M, Vexler ZS* and Ferriero DM*. "Vascular Endothelial Growth Factor Receptor2 inhibition promotes cell death and limits endothelial cell proliferation in a neonatal rodent model of stroke". Stroke, 2010, 41:343-349. * - co-senior authors. PMID: 20101028
3. Faustino J, Wang X, Jonhson C, Klibanov A, Derugin N, Wendland M, Vexler ZS. "Microglial cells contribute to endogenous brain defenses after acute neonatal focal stroke". J. Neurosci. 2011: 31:12992-13001. PMID: 21900578
4. Fernandez-Lopez D, Faustino J, Daneman R, Zhou L, Derugin N, Wendland M, and Vexler ZS. "Blood-brain barrier permeability is increased after acute adult stroke but not neonatal stroke" J. Neurosci, 2012, Jul 11; 32(28): 9588-9600. PMID: 22787045
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L 23

Osuke IWATA

■ Present Position

Centre for Developmental & Cognitive Neuroscience, Department of Paediatrics & Child Health, Kurume University School of Medicine 67 Asachimachi, Kurume, Fukuoka

■ Education

1987-1993 Bachelor of Medicine
Nagoya University School of Medicine, Nagoya, Japan

■ Appointments

2000-2001 Consultant Neonatologist, Perinatal Centre, Division of Neonatology Nagano Children's Hospital, Nagano, Japan
2001-2002 Honorary Research Fellow, Centre for Perinatal Brain Research, Institute for Women's Health, University College London
2002-2007 Senior Research Fellow, ditto
2007-present The present position

■ Selected Publications

1. Iwata S, Bainbridge A, Nakamura T, Tamura M, Takashima S, Matsuishi T, Iwata O. Subtle white matter injury is common in term-born infants with a wide range of risks. Int J Dev Neurosci 2010; 28(7): 573-80.
2. Kawano G, Iwata O, Iwata S, Kawano K, Obu K, Kuki I, et al. Determinants of outcomes following acute child encephalopathy and encephalitis: pivotal effect of early and delayed cooling. Arch Dis Child. (in press).
3. Iwata S, Iwata O, Olson L, Kapetanakis A, Kato T, Evans S, et al. Therapeutic hypothermia can be induced and maintained using either commercial water bottles or a "phase changing material" mattress in a newborn piglet model. Arch Dis Child 2009; 94(5): 387-91.
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5. Robertson NJ, Nakakeeto M, Hagmann C, Cowan FM, Acolet D, Iwata O, et al. Therapeutic hypothermia for birth asphyxia in low-resource settings: a pilot randomised controlled trial. Lancet 2008; 372(9641): 801-3.

L 24

Marianne BERÉNYI

Present Position

Director of the Department of Developmental Neurology, St. John's Teaching Hospital, Budapest, Hungary

Education

1973 M.D.-Semmelweis Medical University, Budapest
1978 Board Certif. in Pediatrics -Postgraduate Medical School, Budapest
1984 Board Certif. in Pediatric Neurology -Postgraduate Medical School, Budapest
1987 Board Certif. in Physiatry-Rehabilitation -Postgraduate Medical School, Budapest
1993 Ph.D. -Hungarian Academy of Sciences, Budapest

Appointments

Deputy department leader	Pediatric Institute -Svábhegy	
	Dept. of Developmental Neurology and Neurohabilitation	1978 - 2002
Assistant professor	Pediatric Institute -Svábhegy	
	Dept. of Developmental Neurology and Neurohabilitation	1982 - 1991
Associate professor	Pediatric Institute -Svábhegy	
	Dept. of Developmental Neurology and Neurohabilitation	1991 - 2002
Director of the Dept.	Pediatric Institute -Svábhegy	
	Dept. of Developmental Neurology and Neurohabilitation	2002 - 2007
Director of the Dept.	St. John's Teaching Hospital	
	Dept. of Developmental Neurology	2007 - present

Selected Publications

1. Katona F.-Berényi M.: Clinical developmental neurology – diagnostics (in Hungarian with English abstract) Clinical Neuroscience, 54: 142-155, 2001.
2. Katona F.-Berényi M.: „How early is too late?" Early therapeutic programs of developmental neurology (in Hungarian with English abstract) Clinical Neuroscience, 54: 196-206, 2001.
3. Katona F.-Berényi M.: The role of studies of JANOS SZENTAGOTHAI in developmental neurology (in Hungarian with English abstract) Clinical Neuroscience, 56: 422-429. 2003.
4. Berényi, M., Katona, F., Madersbacher, H.G.: Intravesical electrical stimulation in newborn, infants and children (in English) in: Textbook of the Neurogenic Bladder 2nd Edition Eds: Corcos, J., Schick, E. Informa UK, pp:770-778, 2008 (ISBN:0415423163)
5. Berényi, M., Katona,.: Diagnostic and therapy of brain injured infants (in Hungarian) in: Textbook of Pediatrics 2nd Ed: Oláh É., MEDICINA. pp.:1641-1656, 2009 (ISBN:978-963-226-176-8)
6. Berényi M, Katona F DEVELOPMENTAL NEUROLOGY: The development of consciousness, communication and sensorimotor behaviour (in Hungarian) Budapest: MEDICINA, 2012. 478 p. (ISBN:978 963 226 331 1)

L 25

Raman SANKAR

Present Position

Professor of Neurology and Pediatrics and Chief of Pediatric Neurology at the David Geffen School of Medicine at the University of California, Los Angeles, CA, USA

Education

1968-1974 Ph.D., University of Washington, Seattle, Washington
1982-1986 M.D., Tulane Medical School, New Orleans, Louisiana
1986-1988 Pediatric Intern-Resident, Children's Hospital of Los Angeles
1988-1989 Resident, Department of Neurology, UCLA School of Medicine, Los Angeles, CA
1989-1991 Fellow, Division of Pediatric Neurology, ditto

Appointments

1992-1999 Assistant Professor, UCLA School of Medicine
1999-2005 Associate Professor, Director of Residency Training in Child Neurology, David Geffen School of Medicine at UCLA
2005-Present Professor and Chief, Rubin Brown Distinguished Chair Division of Pediatric Neurology, David Geffen School of Medicine at UCLA

Selected Publications

1. Sankar R, Curran JG, Kevill J, Rintahaka PJ, Shewmon DA, Vinters HV. Microscopic cortical dysplasia in infantile spasms: evolution of white matter abnormalities. AJNR Am J Neuroradiol 1995; 16:1265-72.
2. Sankar R, Shin DH, Liu H, Mazarati AM, Pereira de Vasconcelos A, Wasterlain CG. Patterns of status epilepticus-induced neuronal injury during development and long-term consequences. J Neurosci 1998; 18: 8382-93.
3. Sankar R, Shin DH, Mazarati AM, Liu H, Katsumori H, Lezama R, Wasterlain CG. Epileptogenesis following status epilepticus reflects age- and model-dependent plasticity. Ann Neurol 2000; 48: 580-9.
4. Mazarati AM, Shin D, Kwon YS, Bragin A, Pineda E, Tio D, Taylor A, Sankar R. Elevated plasma corticosterone level and depressive behavior in experimental temporal lobe epilepsy. Neurobiol Dis 2009; 34: 457-61.
5. Auvin S, Mazarati A, Shin D, Sankar R. Inflammation enhances epileptogenesis in the developing rat brain. Neurobiol Dis 2010; 40:303-310.

L 26

Shinichi HIROSE

Present Position

Professor and Head, Department of Pediatrics, School of Medicine, Fukuoka University, Fukuoka, Japan

Education

1980 M.D., Fukuoka University, School of Medicine
1988 Ph.D., Fukuoka University, School of Medicine (Biochemistry)
1980-1982 Resident, Fukuoka University Hospital
1982-1984 Clinical Fellow in Pediatrics, Fukuoka University Hospital
1984-1988 Graduate Research in Biochemistry, Fukuoka University School of Medicine
1988-1992 Research Associate, Institute of Pathology, Case Western Reserve University, Cleveland, Ohio, USA

Appointments

1992-94 Instructor in Pediatrics, Fukuoka University Hospital
1994-97 Assistant Professor, Department of Pediatrics, School of Medicine Fukuoka University
1997-2005 Associate Professor, ditto
2006-present Professor and Chairman, ditto; Director, Center for Maternal, Fetal and Neonatal Medicine, Fukuoka University Hospital

Selected Publications

1. Sugiura Y, Nakatsu F, Hiroyasu K, Ishii A, Hirose S, Okada M, Jibiki I, Ohno H, Kaneko S, Ugawa Y. Lack of potassium current in W309R mutant *KCNQ3* channel causing benign familial neonatal convulsions (BFNC). Epilepsy Res. 2009;84(1):82-5.
2. Kurahashi H, Wang JW, Ishii A, Kojima T, Wakai S, Kizawa T, Fujimoto Y, Kikkawa K, Yoshimura K, Inoue T, Yasumoto S, Ogawa A, Kaneko S, Hirose S. Deletions involving both *KCNQ2* and CHRNA4 present with benign familial neonatal seizures. Neurology. 2009;73(15):1214-7.
3. Kanaumi T, Takashima S, Iwasaki H, Itoh M, Mitsudome A, Hirose S. Developmental changes in *KCNQ2* and *KCNQ3* expression in human brain: possible contribution to the age-dependent etiology of benign familial neonatal convulsions. Brain Dev. 2008;30(5):362-9.
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L 27

Sarah WECKHUYSEN

Present Position

Postdoctoral Researcher: Neurogenetics Group, Department of Molecular Genetics, VIB, University of Antwerp, Campus Drie Eiken, Antwerp, Belgium
Neurologist, Epilepsy Centre Kempenhaeghe, Hans Berger Kliniek, The Netherlands

Education

1996-2003 Medicine, Katholieke Universiteit Leuven (KUL), Leuven, Belgium
Doctor in Medicine with highest distinction, KUL, Leuven, Belgium
2003-2008 Residency in Neurology, KUL, Leuven, Belgium

Appointments

2008-preesnt Neurologist, Epilepsy Centre Kempenhaeghe, Hans Berger Kliniek, the Netherlands
2008-present Postdoctoral Researcher, Neurogenetics Group, Department of Molecular Genetics, VIB, Antwerp, Belgium

Selected Publications

1. Dupont P, Van Paesschen W, Palmini A, Ambayi R, Van Loon J, Goffin J, Weckhuysen S, et al. Ictal perfusion patterns associated with single MRI-visible focal dysplastic lesions: Implications for the noninvasive delineation of the epileptogenic zone. Epilepsia 2006; 47(9): 1550-7.
2. Weckhuysen S, Deprez L, Peeters K, Deconinck T, Claeys KG, Claes LRF, et al. Novel ATP1A2 mutations associated with familial hemiplegic migraine, common forms of migraine and epileptic seizures. Epilepsia 2007 Nov 19.
3. Dermaut B, Seneca S, Dom L, Smets K, Ceulemans L, Smet J, De Paepe B, Tousseyn S, Weckhuysen S, et al. Progressive myoclonic epilepsy as an adult-onset manifestation of Leigh syndrome due to m.14487T>C. J Neurol Neurosurg Psychiatry 2010; 81(1): 90-3.
4. Deprez L, Weckhuysen S, Holmgren P, Suls A, Van Dyck T, Goossens D, et al. Clinical spectrum of early onset epileptic encephalopathies associated with STXBP1 mutations. Neurology 2010 28; 75(13): 1159-65.
5. Weckhuysen S, Mandelstam S, Suls A, Audenaert D, Deconinck T, Claes LR, Deprez L, Smets K, Hristova D, Yordanova I, Jordanova A, Ceulemans B, Jansen A, Hasaerts D, Roelens F, Lagae L, Yendle S, Stanley T, Heron SE, Mulley JC, Berkovic SF, Scheffer IE, de Jonghe P. KCNQ2 encephalopathy: emerging phenotype of a neonatal epileptic encephalopathy. Ann Neurol. 2012 Jan;71(1):15-25. doi: 10.1002/ana.22644.

L 28

Hitoshi YAMAMOTO

Present Position

Professor, Department of Pediatrics, St. Marianna University School of Medicine, Kawasaki, Japan

Education

1974-1979 Graduate course, St. Marianna University School of Medicine, Kawasaki, Japan
1979-1981 Resident in Pediatrics, ditto
1981-1984 General Pediatrics Fellow, ditto
1989 PhD, ditto

Appointments

1990-1991 Research Fellow, Department of Neuroscience, University of California, San Diego, California
1994-2004 Instructor, Department of Pediatrics, St. Marianna University School of Medicine
2004-2008 Associate Professor, ditto
2008 Secretary general of AOCNA (Asian & Oceanian Child Neurology Association)
2009-present Professor, Department of Pediatrics, St. Marianna University School of Medicine

Selected Publications

1. Yamamoto H, Okumura A, Fukuda M. Epilepsies and epileptic syndromes starting in the neonatal period. Brain Dev 2011; 33(3): 213-20.
2. Yamamoto H, Okumura A. Neonatal seizures as chronic epilepsy. Medimond 2009; L612R9026: 51-6.
3. Fukuda M, Yamauchi H, Yamamoto H, Aminaka M, Murakami H, Kamiyama N, Miyamoto Y, Koitabashi Y. The evaluation of oxidative DNA damage in children with brain damage using 8-OHdG levels. Brain Dev 2008; 30: 131-6.
4. Yamamoto H, Aihara M, Niijima S, Yamanouchi H. Treatments with midazolam and lidocaine for status epilepticus in neonates. Brain Dev 2007; 29: 559-64.
5. Yamamoto H, Fukuda M, Miyamoto Y, Murakami H, Kamiyama N. A new trial liposteroid therapy for intractable epileptic seizures in infancy. Brain Dev 2007; 29: 421-4.

L 29

Jun TOHYAMA

Present Position

Section Chief, Department of Pediatrics, Epilepsy Center, Nishi-Niigata Chuo National Hospital, Niigata, Japan; Contract Associate Professor, Department of Pediatrics, Niigata University Medical and Dental Hospital, Niigata, Japan

Education and Appointments

1986	MD: Niigata University School of Medicine, Niigata, Japan
1986-1990	Department of Pediatrics, Niigata University, Niigata, Japan
1990-1993	Division of Child Neurology, Tottori University, Yonago, Japan
1993-1995	Department of Inherited Metabolic Disease, National Center for Psychiatry and Neurology, Kodaira, Japan
1995-1997	Section Chief, Department of Pediatrics, Niigata National Hospital, Niigata, Japan
1997-1999	Postdoctoral Fellow, Neuroscience Center, UNC Hospital, University of North Carolina at Chapel Hill, NC, USA
2000-present	Department of Pediatrics, Epilepsy Center, Nishi-Niigata Chuo National Hospital, Niigata, Japan
2009- present	Contract Associate Professor in Department of Pediatrics, Niigata University Medical and Dental Hospital, Niigata, Japan

Selected Publications

1. Tohyama J, Nanba E, Ohno K. Hypoxyanthine-guanine phosphoribosyltransferase (HPRT) deficiency: Identification of point mutations in Japanese patients with Lesch-Nyhan syndrome and hereditary gout and their permanent expression in an HPRT-deficient mouse cell line. Human Genetics 1994; 93: 175-81.
2. Tohyama J, Vanier MT, Suzuki K, Ezoe T, Matsuda J, Suzuki K. Paradoxical influence of acid β-galactosidase gene dosage on phenotype of the twitcher mouse (genetic galactosylceramidase deficiency). Human Molecular Genetics 2000; 9: 1699-707
3. Tohyama J, Akasaka N, Osaka H, Maegaki Y, Kato M, Saito N, Yamashita S, Ohno K. Early onset West syndrome with cerebral hypomyelination and reduced cerebral white matter. Brain Dev 2008; 30: 349-55
4. Saitsu H, Kato M, Mizuguchi T, Hamada K, Osaka H, Tohyama J, et al. De novo mutations in the gene encoding STXBP1 (MUNC18-1) cause early infantile epileptic encephalopathy. Nature Genetics 2008; 40: 782-7
5. Saitsu H, Tohyama J, Kumada T, Egawa K, Hamada K, Okada I, et al. Dominant-negative mutations in alpha-II spectrin cause West syndrome with severe cerebral hypomyelination, spastic quadriplegia, and developmental delay. Am J Hum Genet 2010; 86: 881-91.

L 01

NEUROPHYSIOLOGICAL ASPECTS OF NEONATAL SEIZURES

Kazuyoshi WATANABE
Faculty of Health and Medical Science, Aichi Shukutoku University, Nagoya, Aichi, Japan

The advent of amplitude-integrated EEG (aEEG) and its increasing use in NICU has revolutionized the diagnosis and treatment of neonatal seizures. However, as seizure detection with the use of aEEG is often inaccurate, it is necessary for seizures noted on aEEG to be confirmed with standard EEG before treating them. Continuous aEEG does not replace, but is complementary to standard EEG. This lecture reviews some of the findings obtained with standard EEGs, and tries to interpret them with recent findings in the field of basic science.

Ictal EEG characteristics: In addition to well known features of neonatal seizures, seizures mainly occur in active-REM sleep in neonates. This is in sharp contrast to those in older children and adults, in whom epileptic seizures occur mainly in NREM sleep. This may be explained by neurotransmitter effects on sleep mechanisms of neonatal brain different from those of older individuals.

Pathophysiology of neonatal seizures: When all clinical seizures have no electrical correlates, they are non-epileptic, but when the correlation between clinical seizures and frequent electrical discharges are inconsistent, they should rather be considered epileptic, reflecting progression of status epilepticus causing electromechanical dissociation.

Background EEG alteration in neonatal seizures: In neonates without pre-existing brain damage, frequent seizures per se may cause mild depression characterized by loss of high voltage slow pattern (an important constituent of slow wave sleep reflecting cortico-cortical connectivity). Mild depression only in the acute stage is not associated with neurological sequelae, but previously damaged brain may be more vulnerable than normal brain.

L 02

CONVENTIONAL ELECTROENCEPHALOGRAM IN NEONATAL SEIZURES

Toru KATO
Department of Child Neurology, Okazaki City Hospital, Okazaki, Aichi, Japan

The most important feature of neonatal seizures is an electro-clinical dissociation. Non-epileptic events with seizure-like movements would be overestimated, whereas subclinical seizures with EEG correlates would be underestimated. Therefore, EEG is essential for the correct evaluation of neonatal seizures. Recently, amplitude-integrated EEG (aEEG) has been widely used as a continuous brain functional monitoring in neonatal intensive care units. Although it is a simple and convenient tool for evaluation of neonatal seizures, the sensitivity of detection of neonatal seizures is lower on aEEG than on conventional EEG (cEEG).

The ictal EEG changes were usually defined as rhythmic, repetitive, and stereotyped activities lasting more than 10 s with evolutional changes of frequency, amplitude, and morphology. Commonly, the onset of ictal discharges is focal or regional in each seizure, although the foci are usually derived from multiple regions. Migration of ictal discharges is usually seen. The morphologies of ictal discharges are various such as rhythmic delta/theta/alpha waves or repeated sharp/spike/complex waves. Sometimes, the morphologies of the ictal discharges change evolutionally during a single seizure. The correlation between ictal discharges, and seizures semiology or their etiology has not been fully elucidated.

Several reports showed the correlation between cEEG findings and adverse outcome. In these reports, interictal EEG background, delta status, seizure frequency, seizure duration, numbers of seizure foci, and absence of sleep state cycling were correlated with adverse outcome. I will discuss the utilities of cEEG for evaluation of prognosis on the basis of our experience.

L 03

AMPLITUDE INTEGRATED EEG FOR SEIZURE DETECTION

Lena HELLSTRÖM-WESTAS
Department of Women's and Children's Health, Uppsala University, Uppsala, Sweden

The amplitude-integrated EEG (aEEG) is a time-compressed EEG trend, created with the purpose of giving an easy interpretable tracing for intensive-care staff during long-term monitoring of high-risk patients. The aEEG trend is recorded from a limited number of electrode positions, creating a singlechannel aEEG (usually P3-P4 or C3-C4), or two to three channels of aEEG (often F3-P3/C3 and F4-P4/C4, sometimes also a cross-cerebral channel, P3-P4 or C3-C4). The raw EEG is displayed in parallel with the aEEG trend, usually a limited-channel EEG but sometimes also a full EEG.
It has been shown in studies assessing conventional EEG, that the number of recording channels and electrodes influence the number of neonatal seizures that can be detected. It can therefore be expected that a single-channel or two-channel trend recording will detect fewer seizures than a full EEG. The very compressed aEEG trend also makes brief seizures more difficult to detect. It has consequently been shown that if only the aEEG trend is assessed, a majority of seizures will not be detected. However, if the corresponding single- or two channel EEG trace is examined, the chance of detecting seizures is around 75-80% as compared to a standard EEG. Methods for improved automated seizure detection systems are currently developed and will aid bedside evaluation of possible seizures. The most common locations for neonatal epileptic seizure activity are the central and temporal areas, followed by the parietal region. This is probably a main reason why the single- or two channel aEEG/EEG can detect around 80% of seizures appearing in a conventional EEG. Seizures, often subclinical, are common in very preterm infants with acute brain injury, e.g. intraventricular hemorrhages or periventricular leukomalacia. These seizures may be very slow and not possible to detect in the aEEG trend.
In conclusion, the aEEG trend with simultaneous display of the raw EEG, detects seizures in many high-risk neonates and is associated with earlier confirmation of electrographic seizure activity. The aEEG method is also very useful for assessing effects of antiepileptic medications bedside in the NICU.
Due to the limited number of channels and the filtered aEEG trend not all seizures can be detected by the aEEG. For clinical monitoring of high-risk infants we use aEEG/EEG together with repeated conventional EEGs.

L 04

NON EPILEPTIC MANIFESTATIONS AMONG NEONATES

Federico VIGEVANO
Department of Neurosciences and Neurorehabilitation, Bambino Gesù Children's Hospital, Rome, Italy

Non Epileptic Manifestations (NEM) indicate a series of events characterized by clinical elements that may simulate epileptic seizures and therefore leading to a differential diagnosis. NEM may appear at any age, although their major incidence is in pediatric age, during the child's first years of life. Frequently, neonatologists and pediatricians notice unusual behaviors in patients with differential diagnosis with epileptic seizures. Provide a right diagnosis will avoid a wide number of useless and sometimes dangerous tests, and especially a prolonged drug therapy with severe side effects, such as the antiepileptic therapy. Diagnosis requires a careful collection of anamnesis of the episode and modalities of their appearance, mainly the factors that may favor or induce the episode and those that may stop it. Particularly relevant is also the relationship with wake and sleep. A video-EEG recording of the episode may provide a useful support as it will prove the absence of epileptiform discharges. Many difficulties may occur while doing differential diagnosis. A high similarity can appear between epileptic seizures and NEM; for example, an epileptic tonic seizure is very similar to a non epileptic tonic seizure; an epileptic myoclonus is hardly distinguishable from a non epileptic myoclonus only by clinical observation.
Among neonates, a non epileptic myoclonus may be part of a benign entity, as in benign neonatal sleep myoclonus, or expression of a severe condition as in hyperekplexia. Other NEM are jitteriness and ocular disorders.

L 05

DIFFUSION IMAGING OF THE FETAL AND NEONATAL BRAIN

Emi TAKAHASHI
Boston Children's Hospital Harvard Medical School, Boston, MA, USA

Diffusion Magnetic Resonance Imaging (MRI) has provided insight into major white matter pathways in the adult human brain. Recent technical developments enabled us to image fiber pathways also in the fetal and neonatal brain. The talk will summarize the principles of diffusion MRI and tractography and then introduce our recent tractography studies on white matter and gray matter brain development. Challenges and potential uses of diffusion tractography along with EEG/MEG for the epilepsy study will be discussed.

L 06

NEUROIMAGING IN NEONATAL SEIZURES

Jeffrey J. NEIL
Departments of Neurology, Pediatrics and Radiology, St. Louis Children's Hospital, St. Louis, MO, USA

Seizures in neonates have a myriad of causes, the majority of which have MRI correlates. We will review the MRI signatures of the more common causes of neonatal seizures, including hypoxic ischemic injury, sinovenous thrombosis, stroke, brain malformation, and metabolic disorders. We will also consider the metabolic effects of seizures as detected by 1H spectroscopy suggesting lactate accumulation during seizures. Finally, we will touch on the use of MRI as an outcome measure of brain injury in relation to seizure monitoring and control.

L 07

NEONATAL SEIZURES IN THE PRETERM INFANT

Terrie E. INDER
Departments of Neurology, Pediatrics and Radiology, St. Louis Children's Hospital, St. Louis, MO, USA

The incidence of seizures in the preterm infant depends on the gestational age and intracranial pathology of study population alongside the electroencephalographic method and diagnostic criteria used for a seizure. A single channel EEG may underestimate the incidence of seizures, since it may not detect focal seizures. In a study by Shah et al, 51 infants less than 30 weeks gestation were monitored with two channel EEG recording, raw EEG trace and amplitude integrated EEG. In this study the application of limited channel EEG facilitated longer continuous recordings (mean 74hours). One quarter of these infants (mean gestational age 26wks) had seizures identified on the raw EEG trace during their hospitalization at another site. Staudt et al examined a cohort of 156 infants < 2,500g with a 21% incidence of seizures. Of note, 66% of infants who experienced seizures were <31weeks gestation (16). Hellstrom-Westas et al reported a seizure incidence of 75% in a group of ELBW infants with intracranial haemorrhage (17).
Seizures in the preterm infant occur in the sickest and smallest infants, but improved delineation of seizures and their impact on brain injury and development in the preterm infant is required.

L 08

EARLY DEVELOPMENT OF EEG ACTIVITY: FUNCTION VS STRUCTURE

Sampsa VANHATALO
Department of Clinical Neurophysiology, University Hospital of Helsinki, Helsinki, Finland

For the past half a century, preterm and neonatal EEG has relied on recordings with only few electrodes using a limited frequency range. The EEG analysis has remained phenomenological, based on experience from clinical correlates rather than on any physiological mechanisms. Recent developments in EEG recording and analysis techniques have opened new vistas to what may be lost in the conventional EEG. Combining these recordings to known anatomical data or animal work has opened a pathway to genuine translational studies.

The main observation is, that there is a striking amount of infraslow, spontaneous activity transients (SAT), which are unique to the preterm period. In the youngest premies, they are organized into mostly focal events, whereas the size of co-active (SAT) cortical areas is increased towards full term, corresponding very closely to the reported development of thalamo-cortical and cortico-cortical connections. These events are no longer seen after term age. At the same time, there is a gradual build-up of an ongoing cortical activity that continues for the rest of the life.

Recent findings demonstrate that the current clinical recording practise in preterm babies leads to a significant ignorance of prominent EEG activities. Most importantly, current views in developmental neuroscience suggest that SATs are likely the most important type of activity in the developing brain, believed to drive brain wiring at this stage. Detection and monitoring the nature of SATs may give a straightforward, and neurophysiologically reasoned method for monitoring and assessment of brain function during prematurity.

L 09

PROPOSAL OF SEMIOLOGICAL CATEGORIZATION OF NEONATAL SEIZURES

Akihisa OKUMURA
Department of Pediatrics, Juntendo University School of Medicine, Tokyo, Japan

A large majority of neonatal seizures are acute symptomatic. Acute symptomatic seizures in older children, such as seizures in acute encephalitis, are not usually categorized using a framework of epileptic seizures. Historically, neonatal seizures have been described according to several classifications. Volpe proposed a simple classification mainly based on motor phenomena during seizures. Although this classification is easy to understand, seizures with no or minimal motor phenomena may not be categorized appropriately. Moreover, the concept of subtle seizures is not always understood correctly. Mizrahi et al. proposed a classification based on electroclinical findings. This classification divided neonatal seizures into two plus one categories: clinical seizures with and without consistent electrocortical signature, and electrical seizures without clinical seizure activity. The appropriate application of this classification is presumed to be rather easy for pediatric neurologists, but is not always easy for neonatologists. A recently revised terminology by ILAE described that neonatal seizures are no longer regarded as a separate entity and can be classified within the proposed terminology of seizures and epilepsies. However, as stated above, a large majority of neonatal seizures are acute symptomatic, not chronic epilepsy. Seizure manifestations of neonates are different from those of older children. We would like to propose semiological categorization of neonatal seizures consisting of four categories; motor symptoms, hypomotor symptoms, autonomic symptoms, and subclinical seizures. The appropriateness of this categorization is now under verification.

L 10

AUTOMATED SEIZURE DETECTION IN NEWBORNS

Geraldine B. BOYLAN
Medical School, Brookfield Health Sciences Complex, University College Cork, Cork, Ireland

Seizures are the hallmark of neurological dysfunction in the newborn but can be very difficult to detect without EEG monitoring. Current methods used in neonatal intensive care units (NICUs) worldwide are 1-2 channel amplitude EEG (aEEG) systems, aEEG systems with simultaneous raw EEG display or conventional multichannel EEG monitoring (cEEG). aEEG monitoring has the advantage of ease of use particularly for newborns but cEEG does have the advantage of being able to detect more seizures. Whichever method is used in the NICU for EEG monitoring, very specific expertise is required to interpret the recordings. Unfortunately, most NICUs do not have access to a 24 hour neurophysiology service and as a result, EEGs in the NICU are quite often interpreted by health professionals with little or no expertise in EEG. This leads to both under and over reporting of neonatal seizures. A compelling solution to the problem is to use automated algorithms to improve the accuracy of seizure detection in the NICU. This is not a trivial task and requires a multidisciplinary team approach including clinicians, scientists and engineers. In addition, considerable good quality neonatal EEG training data is essential for these algorithms. In this talk, the current state of the art in automated newborn seizure detection will be reviewed and a new real-time algorithm developed at Cork University Maternity Hospital will be demonstrated.

L 11

NEW PARADIGMS IN NEONATAL METABOLIC EPILEPSIES

Phillip L. PEARL
Pediatrics, Neurology, and Music, Children's National Medical Center, The George Washington University School of Medicine and Health Sciences and Columbian College of Arts and Sciences, Washington DC, USA

Neonatal seizures represent a major challenge among the epilepsies vis a vis seizure classification, electroclinical correlation, inherent excitability of neocortex, ontogenic characteristics of neurotransmitter receptors, and responsiveness to standard antiepileptic drugs. Depolarizing properties of GABA-A receptors lead to unique therapeutic challenges and opportunities. Conversely, specific metabolic disorders have inherent therapeutic responsiveness if the correct diagnosis is recognized along with rapid therapeutic intervention. The prototype is pyridoxine dependency, which is allelic with folinic acid dependency. Pyridoxal-5-phosphate dependency is an additional diagnosis that requires earmarked distinction. Clinicians must remain vigilant for these possibilities, including atypical cases where apparent seizure-free intervals may occur. Serine dependent seizures and glucose transporter deficiency may present with neonatal seizures and have specific therapy. A vital potassium channel regulated by serum ATP/ADP ratios in the pancreas and brain may be mutated with a resultant neuroendocrinopathy characterized by development delay, epilepsy, and neonatal diabetes (DEND). This appears to respond optimally to oral hypoglycemic therapy. Urea cycle disorders also require prompt recognition and a combination of dietary and ammonia lowering therapy. The startle syndrome of hyperekplexia, a glycine receptor or transport related disorder which mimics neonatal epilepsy, has been associated with laryngospasm and sudden death but is treatable with benzodiazepines.

L 12

NEW RECIPE? THE ROLE OF METABOLISM IN NEUROPROTECTION

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Neuronal injury (including that induced by hypoxic-ischemic injury) can be prevented through a variety of pharmacological and physical methods. A growing body of literature also has shown that altering metabolism can be neuroprotective. Because of the unique metabolic state of the newborn (due to the importance of ketone bodies), there are novel opportunities for neuroprotection, as well. Protection against a variety of insults (with an emphasis on seizures) using different metabolic substrates, pathways, and clinical/translational strategies will be discussed.

L 13

NEONATAL SEIZURES IN DEVELOPING COUNTRIES

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Few data have been reported concerning the incidence, the etiology, the treatment procedure and the prognosis of epileptic seizures occuring among neonates in developing countries. Some studies have been reported from India and Africa. Incidence of neonatal seizures is higher than in developed countries, and the most common diagnoses associated with seizures were neonatal encephalopathy and meningitis. This may reflect the high prevalence of risk factors for neonatal brain insult in these communities. Most of the time, it is only possible to document seizures that are clinically evident as EEG and of course long term EEG monitoring are not available. Subclinical seizures may have been missed and, given the fact that electro-clinical dissociation is more common in neonates than in any other age groups the incidence may be higher. In developing countries, as many of the births occur at home and there is a lack of any consistent data on gestational age, the Apgar scores are not available and only the history and the physical state of the child on admission may help to make the diagnosis of neonatal encephalopathy. Neuro-imaging may be unavailable and it is not possible to establish other causes of seizures such intracranial and periventricular haemorrhages. Phenobarbital is the only available antiepileptic drug in these countries, sometimes with phenytoin and diazepam. Prognosis is currently worse than in developed countries, and this may be related to etiology and diagnostic delay, and lack of efficient treatment.

L 14

TREATMENT OF NEONATAL SEIZURES

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Seizures in the newborn period are common, and frequently indicate serious underlying brain injury such as global hypoxic-ischemic injury, stroke or infection. Though accumulating evidence suggests seizures may harm the developing brain, there are currently no evidence-based guidelines for evaluation and treatment of neonatal seizures. In this talk, I will address management of neonatal seizures, including controversies regarding how to define, monitor and choose an anti-convulsant agent for neonatal seizures.

L 15

MANAGEMENT OF NEONATAL SEIZURE WITH ANTIEPILEPTIC DRUGS

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Seizure detection using continuous electroencephalogram (EEG) monitoring in clinical practice has brought us the accurate recognition of neonatal seizure (NS) and the alteration of the management of NS. When we consider the treatment for infants with NS, accurate diagnosis of NS with EEG is the first essential step, but not necessarily sufficient. The most effective treatment for each infant with NS is determined based on the etiologies. Thus, the appropriate way to use antiepileptic drugs (AEDs) for NS is also different according to the etiologies. It is practically useful to classify the NSs into the following 3 categories based on etiological mechanism; acute symptomatic, remote symptomatic or idiopathic NS. The majority of NSs are acute symptomatic seizures which result from hypoxic-ischemic encephalopathy, intraventricular or parenchymal hemorrhage infectious diseases and so on. The seizure burdens are transient and AED can be ceased soon after stopping NS, although neonatologists tend to prefer prophylactic use of AEDs. In contrast, longer administration of AEDs is often appropriate treatment of remote symptomatic NSs which are generated from congenital cerebral abnormalities or neurocutenous diseases.
In this session, we will show our clinical experience of the infants with NS who were managed according to our strategy for NS with continuous EEG monitoring. Then, we will discuss about the better management of NS focusing on the antiepileptic treatment for the infants with acute symptomatic NSs.

L 16

EEG MONITORING IN NEONATES: AMERICAN CLINICAL NEUROPHYSIOLOGY GUIDELINES

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EEG monitoring is being used with increasing frequency to identify electrographic seizures in critically ill neonates. In 2011 the American Clinical Neurophysiology Society published a guideline focused on EEG monitoring in neonates which aimed to provide "an expression of idealized goals." The guideline addressed indications for conventional EEG monitoring in neonates (differential diagnosis of abnormal paroxysmal events, detection of electrographic seizures, judging the severity of encephalopathy), procedures for EEG monitoring (electrodes, montages, video monitoring, bedside observer responsibilities, and monitoring duration), interpretation and reporting, and data retention and storage. The guideline also discusses the role of digital trending (such as amplitude integrated EEG).

L 17

NEONATAL SEIZURES IN INFANTS WITH NEUROINFECTION

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The estimated rate of neonatal seizures in term newborns is 1 to 3 per 1000 live births. Neonatal seizures occur during the first 28 days of age, often comprise the first clinical manifestation of neurologic dysfunction, and frequently reflect serious underlying brain injury. The more common disorders that present with neonatal seizures include hypoxic-ischemic encephalopathy, cerebrovascular disorders, cerebral dysgenesis, inborn error of metabolism and transient metabolic disturbances such as hypocalcemia, hyponatremia, and hypoglycemia. In addition, neuroinfection is also the main cause for neonatal seizures. In a Canadian population-based study by Ronen et al., neuroinfection was 19% of all neonatal seizures. Neoroinfections remain an important cause of acute and long-term neurological morbidity regardless of congenital, intrapartum or postnatal infection. The actual clinical presentations of infection in the neonate are different for bacteria, viruses and parasites. Infants with bacterial infections tend to present with sepsis, while those with cytomegalovirus or toxoplasmosis infections may be clinically asymptomatic at birth despite their obvious intracranial involvement. Neonates with viral infections such as herpes simplex virus may present with active hepatitis, skin vesicles, petechiae or seizures. The mechanism of infection and damage is different amongst the infectious agents. For example, viruses tend to produce a selective necrosis of specific cell types, whereas bacteria and fungi are less selective. Clinical and imaging differentiation helps us diagnose clinical etiology. Neonatal seizure is also the key findings for differential diagnosis of neuroinfection. Here, we present case reports and literatures focused on bacterial infection including brain abscess, herpes simplex virus, and parechovirus.

L 18

NEUROLOGICAL PROBLEMS IN FAMOUS MUSICIANS: IN WORDS AND MUSIC

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This presentation will review the musical and medical biographies of selected musicians and composers, with an emphasis on the relationship between their neurological disorders and their artistic productivity. The order is chronological. Live performance of selections from each artist will accompany the discussion.

Medical vignettes of great musicians are examined with a focus on the relationship between the medical disorder and artistic creativity. Robert Schumann's (1810-1856) compositional productivity correlated with bipolar affective disorder and he ultimately died of neurosyphilis, widowing Clara Schumann (1819-1896), herself a great pianist and composer who suffered severe rheumatism, neuralgia, and two strokes. Maurice Ravel (1875-1937) died of familial Pick's dementia. Dmitri Shostakovich (1906-1975) suffered a prolonged illness most consistent with motor neuron disease. Cole Porter (1891-1964) sustained severe orthopedic and neurological injuries following an equestrian accident, was among the first patients to receive curare during ECT for depression, and had severe phantom limb pain until his death. George Gershwin (1898-1937) presented with personality change and uncinate seizures attributed to hysteria but died from herniation secondary to a low grade glioma, as opposed to the long chronicled reports of glioblastoma multiforme. We use these vignettes to discover the history of neurology through the lens of great musicians' lives and to enhance our appreciation of music by combining medical biographies with live musical performance of the masters' works.

L 19

EARLY DEVELOPMENT OF EEG ACTIVITY: FROM BENCH TO CLINIC

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Studies in neonatal rodents revealed discontinuous temporal organization of activity and immature cortical EEG patterns that are reminiscent of the activity observed in preterm infants (delta-brushes). In the very immature animals (the two first days after birth) deltabrushes are essentially composed of a delta wave associated with an activation of glutamate NMDA and AMPA receptors at the thalamocortical synapses that drive firing of neurons in the dense cortical plate. GABAergic synapses develop late and contribute little to cortical neuronal network activity during this early developmental stage. Later on in development, delta-brushes start to display rapid oscillatory component in gamma and alpha/beta frequency bands. Early cortical gamma oscillations are initially driven by gamma-rhythmic glutamatergic input from thalamus to the cortical layer 4, they enable multiple replay of sensory input in the thalamocortical network and support long-term potentiation at thalamocortical synapses. With further maturation, intracortical GABAergic synapses start to contribute to synchronization of cortical gamma oscillations. Mechanisms of generation of alpha/beta oscillations are still unknown, but they likely involve intracortical and corticothalamic mechanisms. In sensory cortices, delta-brushes are driven by spontaneous activity at sensory periphery: in somatosensory cortex, delta-brushes are driven by sensory feedback resulting from physiological myoclonies and in visual cortex, delta-brushes, often organized in slow activity transients, are driven by spontaneous waves of activity in retina. Similar paradigm also operates in preterm human infants during the corresponding developmental stages. This suggests that human fetus, which grows in utero in the conditions of virtually complete sensory deprivation, possesses endogenous mechanisms of sensory stimulation to drive delta-brushes that are instrumental for the development of thalamocortical sensory maps during the critical developmental period.

L 20

EVELOPMENTAL AND PATHOGENIC MODAL SHIFTS OF GABA ACTIONS IN IMMATURE BRAIN

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One of the recent topics in neuroscience is that the major inhibitory neurotransmitter GABA necessarily evokes excitation in immature brain, in contrast to inhibition in adult brain. Since $GABA_A$ receptor is a Cl^- channel, such a developmental switch of GABA action from excitation (Cl^- efflux) to inhibition (Cl^- influx) is induced by changes in Cl^- gradient produced by cation-chloride cotransporters. In immature neurons, NKCC1, a Na^+, K^+-$2Cl^-$ cotransporter, takes up Cl^- to keep $[Cl^-]i$ high, promoting depolarizing GABA action by efflux of Cl^- along with its electrical gradient, which exert excitation typically. After the development, KCC2, a K^+-Cl^- cotransporter, extrudes Cl^- out of cells to keep $[Cl^-]i$ low, hence GABA hyperpolarizes postsynaptic neurons by Cl^- influx along with its chemical gradient and act as inhibitory transmitter.
Ambient GABA before synaptogenesis exhibits tremendous action on developing brain, such as neurogenesis and migration. Then vesicular GABA release induces synaptogenesis and temporal excitatory transmission. Therefore, modal shifts of $GABA_A$ receptor-actions either by changes in Cl^- gradient or GABA/$GABA_A$ receptors could greatly influence brain development at any stage of development. In this concern, maternal taurine could be a modulator of $GABA_A$ receptor and Cl^- homeostasis during perinatal period. Although neither a fetus nor a neonate can synthesize taurine but it is transferred from maternal blood via either placenta or maternal milk ingestion. Interestingly, ambient taurine in cerebral cortex was more than 1000 times of ambient GABA. Taurine could be an endogenous agonist for embryonic tonic $GABA_A$ receptor in the neocortex, which might serve as a stop signal for radial migration. In addition, taurine activate WNK-SPAK/OSR1 signaling which reciprocally regulates NKCC1 and KCC2 functions to keep intracellular Cl^- high. Therefore, taurine may contribute to embryonic neuronal Cl^- homeostasis which is required for normal brain development.
Thus, some deleterious consequence might occur in children following prenatal and/or neonatal exposure to pharmacological and/or environmental stress which perturbs necessary modal shifts of GABA actions during development.

L 21

PREDICTION OF BRAIN DAMAGE WITH MINIATURE TELEMETRY IN A RAT MODEL OF NEONATAL HYPOXIC-ISCHEMIC ENCEPHALOPATHY: BACKGROUND EEG VERSUS SEIZURES

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The relationship between acute electrographic seizures and subsequent brain damage in neonates is unclear, if not controversial. Here, electrographic seizures at postnatal day 7 were compared in neonatal-rat models of hypoxia alone (Ha) and hypoxia-ischemia (HI). While both models display acute seizure activity during the 2-h hypoxia treatment, only HI causes an encephalopathy (HIE) with severe neuronal degeneration. Single-channel EEG was recorded during Ha and HI treatment with a novel miniature telemetry device. The electrographic seizures (and their behavioral correlates) appeared virtually identical in both Ha and HI models during the hypoxia treatment, and were identified as discrete events with power in the traditional delta (0.1-2 Hz) and/or alpha (8-12 Hz) bands. Although both the initial pattern and the waveforms of acute seizures were quite similar in Ha and HI animals during the first hour of treatment, Ha caused a more severe electrographic seizure profile than HI during the second hour of the hypoxia treatment. Based on analyses of seizure frequency and power spectral density, the EEG-recorded seizures progressively increased during the 2-h treatment in Ha animals, while HI led to a progressive decrease in seizures with significant suppression of the EEG background. However, during the sub-acute period after the hypoxic-gas exposure, electrographic seizures were detected only after HI, but not after Ha. Background suppression was also observed only after HI and not Ha. These data suggest the following conclusions: (1) the hypoxic-gas exposure common to both of the models (i.e., Ha and HI) drives the seizures during treatment, but these seizures per se do not lead to overt brain damage; (2) the seizures during Ha are substantially more robust than those during HI, apparently because ongoing neuronal damage during HIE actually blunts the electrographic seizures, and (3) although electrographic seizures were preferentially detected after HI versus HA, the decrease in background EEG during and after HI better predicts frank brain damage during HIE. Because background suppression of the beta and gamma bands could be detected with only a few minutes of EEG recording, background suppression is potentially a rapid and reliable biomarker for non-invasive detection of previous and/or ongoing brain injury in human neonates. Future clinical studies in the neonatal ICU with comparable recording strategies should test these hypotheses in at-risk neonates.

Disclosure: MJL and FED have financial interest in Epitel, Inc.

L 22

MRI FOR DIAGNOSIS AND PROGNOSIS OF LONG-TERM INJURY AFTER EXPERIMENTAL NEONATAL ARTERIAL STROKE

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Arterial ischemic stroke in the neonate occurs at a rate similar to that in adults. The excitotoxic, inflammatory and oxidative responses are the major contributors to ischemic injury in both the immature and adult brain but there are several areas where these responses diverge. We will discuss emerging data that the status of the neurovascular integrity and local neuroinflammation are vastly different after neonatal and adult focal stroke. We will demonstrate that following transient middle cerebral artery occlusion (tMCAO) in neonatal rats blood-brain barrier (BBB) integrity is markedly disrupted in acute adult stroke but not in acute neonatal stroke and that endothelial-leukocyte interactions play an important role in maintaining BBB integrity and minimizing ischemic injury in neonates. We will then show that the inflammatory component plays a significant role in injury but that pharmacological deletion of microglial cells before induction of tMCAO enhances neuroinflammation, alters the mode of neuronal death on the apoptotic/necrotic continuum, and makes early injury worse, rather than protects. We will discuss the value of multiple MRI modalities for diagnosing early injury (DWI), including the presence of apoptotic neuronal death, and for monitoring injury progression (T2W and DTI), including effects of treatments that target short-term protection and neurorepair.

L 23

DEVELOPING COT-SIDE BIOMARKERS FOR ACUTE CEREBRAL INJURY

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Despite advances in understanding of neonatal seizures over past decades, the mechanism of neurodevelopmental impairments in affected infants remain mostly unknown. Application of newly developed cerebral biomarkers may help delineate causal relationships between variables such as acute hypoxia-ischaemia, seizures, treatments, tissue damage and neurological functions. Quantitative magnetic resonance techniques are useful to predict functional and histo-pathological outcomes following acute events. In a large animal model of perinatal asphyxial encephalopathy, energetic metabolites obtained using 31-phosphorous MRS have been shown to be predictive of later metabolic derangements as early as two hours after the commencement of resuscitation. In a similar translational model, temporal changes of the apparent diffusion coefficient of water after transient hypoxia-ischaemia and resuscitation have been demonstrated to surrogate the level of energetic metabolites and histo-pathological cerebral damages in the corresponding region. While these magnetic resonance biomarkers are useful to assess metabolic, functional and histopathological conditions of the cerebral tissue, serial data collection is still difficult for most institutions. Recently, our group has utilised time-resolved near-infrared spectroscopy (TR-NIRS) to serially monitor microstructural changes in the brain tissue. In addition to the absorption coefficient of near-infrared light, which gives information about tissue haemoglobin oxygenation, TR-NIRS provides the scatter coefficient, which has similar, but opposite nature with water diffusion. In the newborn infant, the scatter coefficient of the brain is associated with the maturational status of the infant, and its temporal changes after birth vary according to clinical courses. Using these handy, non-invasive techniques, the evaluation of direct relationships between acute events, seizures, treatments, and tissue damages may be significantly improved, which may contribute to further advances in therapeutic strategies for perinatal asphyxia and neonatal seizures.

L 24

DOES HYPOTHERMIA ALTER THE INCIDENCE OF EPILEPSY IN ASPHYXIATED NEONATES?

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Objective: Symptomatic epilepsy usually indicates the severity of brain injuries, though. this correlation is indirect because direct causal connection is presently unknown. Moderate body hypothermia may diminish consequences of hypoxic injuries of developing brain. The objective of our clinical research was to detect the possible interrelation of hypothermia and the frequency of symptomatic epilepsy. The Dept. of Developmental Neurology is responsible to detect early signs of abnormal brain development, to accomplish early diagnosis, and initiate individual neurotherapy with clinical follow up.
Method: Data of the last 5 years (2008-2012) were analyzed. During this time 2.223 baby were referred to us, among them 1.302 were > 36 weeks of gestation. Moderate body hypothermia tends to be more frequent in Hungary, but still not universally accepted. Enrolling protocol follows the international recommendation. Data of 170 asphyxiated neonates with hypothermia were compared with 66 normotherm. Their case history, admission time (2 – 4 postnatal weeks), items of diagnostic procedure (neuroimaging, evoked brainstem potential, functional evaluation of motor and sensory performance and precognitive functions) were similar. Video-EEG with 24 channels were performed at admission and repeated during the follow-up period of 12-18 months when either clinical signs or abnormal electric signs were detected.
Results: 16 % among normotherm and 20 % among hypothermic developed epilepsy during the observed period. Statistical significance was not present, the difference in percentage does not indicate the ameliorating effect of hypothermia.
Conclusion: Total moderate body hypothermia does not seem to diminish the occurrence of symptomatic epilepsy, but prevents its intractability and enhances the efficacy of intensive complex neurotherapy.

L 25

FUTURE TARGETS FOR ANTISEIZURE THERAPY IN THE NEONATE : PRECLINICAL AND CLINICAL OBSERVATIONS

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Traditional treatment of neonatal seizures evolved before the evolution of knowledge pertaining to targets relevant to the control of neuronal excitability. Further, the adaptation of medications like phenobarbital and phenytoin for treating neonates was based on the ability of those drugs to control seizures in adults with the implicit assumption that similar mechanisms would be operant in the immature brain. In this presentation, we shall discuss the theoretical concerns about the safety of traditional agents that modulate GABA-A or voltage-gated sodium channel function. We shall consider novel targets such as non-NMDA glutamatergic sites (AMPA), synaptic vesicle protein SV2A, sodium-potassium-chloride co-transporter-1 (NKCC1), voltagegated potassium channel of the Q-type (Kv 7.2/7.3) and the accumulating preclinical knowledge about their suitability for use in neonatal seizures. We shall include preclinical knowledge pertaining to the potential for development-specific neuronal injury, efficacy against seizures, as well as the potential for neuroprotection and antiepileptogenic promise. The concept that both efficacy and safety of therapy may be guided by knowledge of the ontogeny of specific targets during development will be the main theme of this presentation. The presentation will then review clinical data pertaining to the efficacy of anticonvulsant medications, old and new, with comments on the level of evidence available to guide practice. Finally, design of emerging clinical trials for novel approaches to the treatment of neonatal seizures will be covered.

L 26

GENETICS IN BENIGN NEONATAL SEIZURES

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Benign neonatal seizures are defined as seizures which occur in the neonatal period and remit spontaneously within a few months after birth. Benign familial neonatal Epilepsy (BFNE) is an autosomal dominant inherited epilepsy characterized by benign neonatal seizures in which mutations of either *KCNQ2* or *KCNQ3* are found in approximately half of patients. *KCNQ2* and *KCNQ3* are genes encoding voltage gated potassium channels, K_v7.1 and K_v7.2, respectively. The mutations identified were all heterozygous and found to hamper the K_v7.1/Kv7.2 functions. The dominant inheritance is attributed to haploinsufficiency. K_v7.1/Kv7.2 channels are believed to control neuronal excitabilities in the brains of those newborns whose GABAergic inhibitory system is yet to be established. K_v7.1/Kv7.2 deficiency thus induces neuronal hyper-excitation, i.e., seizures; these remit as GABAergic neurotransmission becomes the main inhibitory element in the mature brain. Similarly, heterozygous missense *SCN2A* mutations were found to result in benign familial neonatal infantile Epielpsy (BFNIE). BFNIE is an autosomal dominant inherited epilepsy in which seizures may develop not only in the neonatal period but also in infancy. *SCN2A* is the gene encoding α2 subunit of a neuronal voltage gated sodium channel, Na_v1.2, which is expressed in inhibitory interneurons. While interneuron dysfunction resulting from *SCN2A* mutations leads to seizures, spontaneous remission of seizures occurs later in life as Na_v1.6 compensate for the mutational deficiencies. The molecular pathomechanisms of the development and cessation of benign neonatal seizures are being revealed, and further studies may provide necessary therapeutic measures against the commoner epilepsies.

L 27

NEONATAL EPILEPTIC ENCEPHALOPATHIES DUE TO KCNQ2 MUTATIONS: A NEW TWIST ON AN OLD STORY

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Mutations in *KCNQ2* and *KCNQ3*, encoding the voltage-gated potassium channel Kv7.2 and Kv7.3 are since long known to be present in 60-70% of families with benign familial neonatal seizures (BFNS). Surprisingly, de novo *KCNQ2* mutations were recently reported to be present in 10% of patients with a neonatal epileptic encephalopathy in which MRI and metabolic workup was negative. As has been seen in other genetic epilepsies with for example *SCN1A* or *GLUT1* mutations, the phenotypic spectrum associated with *KCNQ2* mutations appears to be very broad. In this talk clinical and EEG features of patients with a *KCNQ2* encephalopathy will be presented, and current knowledge on pathophysiology and treatment options will be discussed.

L 28

VARIATION OF SUPPRESSION-BURST EEG PATTERNS SEEN IN EPILEPTIC ENCEPHALOPATHIES STARTING IN THE NEONATAL PERIOD

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Epileptic encephalopathies (EEs) in the neonatal period are age-related epileptic syndromes characterized by a variety of behavioral seizure manifestations, distinctive electroencephalogram (EEG) patterns, and poor outcomes. As seizures in the newborn 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 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 EEs. A suppression-burst EEG pattern (SBP) is usually seen in neonates with serious brain damage or neonatal epileptic syndromes. We will highlight our recent experience of EEs and also propose a precise definition for SBP which has not correctly been identified in the literatures. EEs with SBP in neonatal period are known to evolve into relatively few types of epileptic syndromes. We will also emphasize the importance of ictal EEGs for diagnosis and treatment of neonatal epilepsies and epileptic syndromes.

L 29

AN EMERGING NEW CLINICO-GENETIC VARIANT OF WEST SYNDROME

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Objective: We previously reported the patients of West syndrome with cerebral hypomyelination and reduced cerebral white matter as a new clinical condition. And *SPTAN1* mutations have been identified in West syndrome with this condition. We aimed to investigate the genotype-phenotype correlation in patients with *SPTAN1* aberration.
Methods: Among the previously reported patients of West syndrome, 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 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 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 and more severe clinical conditions of West syndrome with severe cerebral hypomyelination and developmental delay, suggesting the dominant negative effects of the mutations.

L 30

BRIDGING THE AGE GAP – APPLYING NEONATAL SEIZURE LESSONS TO ACUTE SYMPTOMATIC SEIZURES IN OLDER CHILDREN

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Many similarities exist between neonatal seizures and acute symptomatic seizures in older, critically ill children. Extensive research has focused on seizure detection and management in neonates, and this framework may be useful in studying and managing acute symptomatic seizures in older critically ill children. Like neonates, about half of critically ill children with an acute encephalopathy experience seizures. Further, as in neonates, electromechanical uncoupling of seizures is common even without neuromuscular blockade. EEG monitoring is also required for seizure identification. While full-array EEG remains the gold standard, trend detection techniques are helpful in measuring seizure burden in sick older children, just as amplitude-integrated EEG has been utilized in neonates. While existing anticonvulsants appear efficacious in treating acute symptomatic seizures, management is based largely on soft data and randomized clinical trials are needed. Finally, as in neonates, it is unclear if acute symptomatic seizures are simply a biomarker or worse brain injury or whether they independently worsen outcome per se in critically ill children. Trials studying acute symptomatic seizures in neonates and older critically ill children face similar challenges and may require similar study designs.

G1-01

DEVELOPMENT OF AMPLITUDE-INTEGRATED EEG COLORED ACCORDING TO SPECTRAL EDGE FREQUENCY

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Objectives: Amplitude-integrated EEG (aEEG) is a technique to compress EEG data that is utilized as a means of seizure detection not only in neonates but also in older infants. To improve the interpretability of aEEG, we attempted to devise a color scale that allows us to incorporate spectral edge frequency (SEF) information into aEEG.

Methods: In the ictal EEGs of focal seizures recorded from a neonate and a three-month-old infant, the aEEG trend was built based on the bilateral electrode derivations, including the seizures. The EEG data were first filtered with a band-pass frequency range of 2 to 15 Hz. Next, the minimum and maximum absolute amplitudes of the EEG envelope were plotted using a semilogarithmic amplitude scale with respect to every 15-s and 1-s data segment. We built a density spectral array (DSA) as well as an aEEG trend that incorporated a color scale indicating the SEF (hereinafter called aEEG/SEF) for comparison.

Results: Of the three types of EEG spectra, including the representative seizure data, the figures of aEEG/SEF appeared most easy to interpret. The coloring of aEEG trends did not hinder the interpretability of aEEG.

Conclusions: The aEEG/SEF data presentation technique might be a valid option in the aEEG recording of seizures. An extensive study involving many neonates and infants is needed in the future to fully evaluate the usefulness of aEEG/SEF.

G1-02

THE ADEQUACY OF DENSITY SPECTRAL ARRAY AND AMPLITUDE-INTEGRATED EEG FOR NEONATAL SEIZURE IDENTIFICATION

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Objective: To evaluate the diagnostic accuracy of density spectral array (DSA) and amplitude-integrated EEG (aEEG), and the adequacy of reduced channel DSA and aEEG for neonatal seizure identification.

Method: We transformed a set of 46 original eight-channel EEGs including 26 records with seizures and 20 without seizures into four different deviations of a single-channel DSA and aEEG recording (Fp1–Fp2, C3–C4, T3–T4, O1–O2) and four different deviations of a double-channel DSA and aEEG recording (Fp1–C3 and Fp2–C4, C3–O1 and C4–O2, Fp1–T3 and Fp2–T4, T3–O1 and T4–O2). Two authors with sufficient experience investigated the series of DSA and aEEG records, blinded to the raw EEG, and marked events suspected to be seizures. Their performance was compared to seizures identified on the underlying raw EEG.

Results: The 46 original EEG recordings contained 104 discrete seizures over 62 hours. The median (range) sensitivity for seizure identification across all recordings was 38 (28-46) % using DSA and 60 (45-75) % using aEEG. Mean number of false-positive diagnosis were 1.5 (times/hour) using DSA and 0.39 using aEEG. Sensitivity on aEEG was higher, and the false-positive diagnosis was lower than those on DSA ($p < 0.001$). In comparison between single and double-channel recordings, sensitivity with double channels was higher both on DSA and aEEG ($p = 0.01$) without significant difference in false-positive diagnosis.

Conclusion: Our results demonstrated that aEEG has an advantage over DSA for seizure identification in neonates. Multiple channel aEEG monitoring is recommended to improve the seinsitivity of diagnosis.

G1-03

GAMMA OSCILLATIONS SUPERIMPOSED ON SUBCLINICAL SEIZURE ACTIVITY IN A NEWBORN INFANT WITH HYPOXIC-ISCHEMIC ENCEPHALOPATHY

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Objective: High frequency electroencephalogram (EEG) oscillations are thought to be associated with ictogenesis in adults and children. We therefore explored fast activities, which were beyond the traditional EEG activity bands, from the ictal EEGs of neonatal patients' seizures.
Case Report: The subject was a female newborn infant with a severe hypoxic-ischemic encephalopathy. The patient was born at a gestational age of 39 weeks by an emergency cesarean section. Background EEG activity on the first day of life showed a burst-suppression pattern. MRI disclosed diffusely decreased apparent diffusion coefficient values on day 2 and multi-cystic encephalomalacia on day 17.
Method: We recorded a long-term 8-channel video-EEG with a sampling frequency of 500Hz from days 0 to 15 after birth. We examined the ictal and subclinical-ictal EEGs through temporal expansion of traces with a low-cut frequency filter and time-frequency spectral analysis.
Results: In total, 64 ictal and 90 subclinical-ictal events were recorded until day 11. We found gamma oscillations with approximate frequencies of 30Hz to 50Hz superimposed over delta waves in the subclinical focal seizures recorded on days 8 to 11. The occurrence of gamma oscillations temporally coincided with the emergence of subclinical seizures. The spectral power of the gamma oscillations reached a peak on day 11. These subclinical seizures were multifocal with dominance over the right frontal head region.
Conclusions: The detection of gamma oscillations in neonatal seizure activity is a novel finding. Further investigation is needed to determine the clinical significance of these fast activities during the neonatal period.

G1-04

CLINICAL MANIFESTATIONS AND EEG FINDINGS OF NEONATAL SEIZURES: A SINGLE CENTER 5-YEAR REVIEW FROM SINGAPORE

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Objective: With the easy availability of simultaneous video-EEG recordings, there is better characterization of clinical seizures and correlation with the EEG seizure pattern. This improvement has also allowed more accurate description and classification of neonatal seizures.
Method: We retrospectively reviewed the video-EEG recordings of neonates who had neonatal seizures or suspicious paroxysmal events in the 5-year period from 2008 to 2012 inclusive. We categorized the seizure manifestation based on the J Volpe's neonatal seizure classification.
Results: We had 26 neonates with a total of 50 neonatal EEGs recorded. Seven neonates had clinico-electrographic seizures captured and these included tonic, clonic and myoclonic and subtle seizures. Three had EEG seizures without any clinical manifestations. All 10 neonates who had seizures were already on treatment with phenobarbitone. The EEG seizure patterns were varied, with most seizure onsets characterized by localized rhythmic activity or repetitive spikes of delta, alpha or beta frequency, which evolved and spread to wider areas. Repetitive myoclonic jerks were associated with generalized polyspike-wave discharges. Six neonates had non-epileptic events; 2 had apneas, 2 had non-epileptic myoclonus (sleep myoclonus), and 2 had eye deviation associated with extensor posturing. The remaining 10 neonates had no seizures or events captured.
Conclusions: A simultaneous video-EEG study is very helpful towards differentiating neonatal seizures from non-epileptic movement disorders and abnormal behaviors in preterm and term babies. The clinical manifestations and EEG of neonatal seizures in our series are similar to previous reports.

G1-05

EEG BACKGROUND ACTIVITIES IN PRETERM ANDTERM NEONATES WITH SEIZURES

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Objective: To investigate EEG background activity in preterm and term neonates with clinical seizures.
Method: Forty six neonatal EEG recording were collected from clinical neurophysiology laboratory archive of Children's Hospital, Ege University School of Medicine. Each EEG background was retrospectively classified into four grades (Grade 0 : Normal, Grade I: Mildly abnormal, Grade II: Moderately abnormal, Grade III: Severely abnormal) by EEG reader blinded to the semiology and etiology of seizures. The relatoionship between EEG background activity and the etiology of neonatal seizures was investigated.
Results: Etiology was identified in 22 (48%) neonates: hypoxic ischemic encephalopathy in 8 (17.3%) neonates, transient metabolic distrubances in 5 (10.8%), neonatal menengitis in 5 (10.8%), intracranial hemorrhage in 3 (%6.5) and inherited metabolic disorder in 1 (2.1%). No etiologic reason was found in 24 (52%) neonates. Moderate / severely abnormal EEG background acivities were found in neonates with identified etiology. Normal / mildly abnormal neonatal EEG background activities were found in neonates with subtle type seizure semiology.
Conclusion: The grading of EEG background activity might be used as prognostic indicator in neonates with clinical seizures.

G1-06

PROGNOSTIC VALUE OF EEG BACKGROUND IN TERM NEONATES WITH HYPOXIC-ISCHEMIC ENCEPHALOPATHY

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Objective: To determine the prognostic values of neonatal electroencephalography in term neonates with hypoxic ischemic encephalopathy.
Method: Electroencephalography, neuroimaging, periodic neurological exams and a developmental test at 44-48 months after discharge from the hospital were performed on twenty five term newborn infants with clinical evidence of hypoxic ischemic encephalopathy.
Results: Normal/mildly abnormal neonatal electroencephalography correlated with favorable outcome, particularly if neuroimaging was normal. The cranial MRI sensitivity was 83.3%, while the specificity was 57.9%, the positive predictive value was 38.5%, and the negative predictive value was 91.6%. Moderate/severely abnormal electroencephalography and multifocal/diffuse cortical or deep gray matter lesions correlated with poor outcome.
Conclusion: Newborn infants with hypoxic ischemic encephalopathy should be treated in neonatal intensive care units, assessed with periodic neurological examination, electroencephalogram and brain imaging. This would help to initiate early intervention and improve the outcome of patients.

G1-07

OUTCOMES OF PERINATAL STROKE IN CHILDREN WITH INTRAUTERINE HERPESVIRUS INFECTION

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Objective: To explore the neurological outcome of perinatal stroke in children with intrauterine herpesvirus infection
Method: 26 children with intrauterine herpesvirus infection in age from 2 to 12 months, who suffered a stroke in the neonatal period.
Results: Hemorrhagic forms constituted the majority - 22 cases (84,6%), ischemic strokes were diagnosed in 4 children.Outcomes of hemorrhagic forms of stroke were neurological syndromes with varying degrees of severity. In 16 children (72,7%) was formed cerebral palsy, in conjunction with symptomatic epilepsy - 11 cases, isolated epileptic syndrome in 1 child. Ophthalmologic examination revealed: atrophy of optic nerves (10 - 45,5%); neuroangiopathia (4 - 18,2%), strabismus (3 -13.6%). Syndrome of movement disorder is presented in the form of: muscular dystonia - 2 (9,1%), pyramidal insufficiency - 3 (13,6%) children. The results of neuropsychological testing showed: 15 children (68,2% of cases) had a severe degree of psychomotor delay; 6 (27.2%) - the average degree, and only 1 child diagnosed with mild delays. Outcomes of ischemic forms of perinatal stroke were presented in the form of syndrome of movement disorder: muscular hypotonia (1), pyramidal insufficiency (1), easy hemiparesis (1), moderate (2) and light (1) the degree of neurodevelopmental delay; focal epilepsy (1). Rough neurological symptom in the form of spastic-hyperkinetic forms of cerebral palsy, symptomatic epilepsy, severe psychomotor retardation and pathological vision formed in 1 child (25%).
Conclusion: Outcomes of perinatal stroke in children with intrauterine herpetic infection were various neurological syndromes, which led to the formation of early neurological disability.

G1-08

NEONATAL SEIZURES IN CHILDREN WITH INTRAUTERINE HERPES INFECTION

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Aim: To explore the character and course of neonatal seizures in children with intrauterine herpes infection (IHI).
Methods: We examined 160 children with IHI. 42 children with neonatal seizures were the study group (M=26, F=16).
Results: Intraventricular hemorrhage of 2-3 degrees was found in 27 cases (64.2%), intracerebral hemorrhage - 12 (28.5%), herpes meningoencephalitis - 11 (26.2%), congenital abnormalities of the brain – 6 (14.3%), subarachnoid hemorrhage - 5 (11.9%), cerebral infarction - 2 (4.7%). At the same time, the HIE was diagnosed in 17 (40.4%) infants of the study group. Semiotics of neonatal seizures: multifocal clonic - 5, generalized tonic - 2; focal tonic: asymmetrical truncal posturing - 7, sustained eye deviation - 4; spasms: flexor – 5, mixed - 6; myoclonic: focal - 2, fragmentary - 4; motor automatisms - 7. Convulsions were resistant to therapy and had of high frequency in 19 children (45.2%). Generalized nature of the infection was confirmed in all children: the positive result of ELISA (anti-CMV, anti-HSV Ig M) and a positive PCR for CMV/ HSV DNA. Catamnesis over the next 6 months showed that 15 (35.7%) children had symptomatic epilepsy, 3 patients (7.1%) - epileptic encephalopathy.
Conclusion: Analysis of etiological factors showed the combined effect of viral infection and hypoxia-ischemia in the genesis of neonatal seizures. The main pathomorphological changes were intracranial hemorrhage (intraventricular, intracerebral). Neonatal seizures in most cases (69%) were epileptic and were resistant to therapy in 45.2% cases. Outcome of neonatal seizures was postneonatal epilepsy in 18 (42.9%) children.

G1-09

A GENETIC ANALYSIS OF BENIGN NEONATAL EPILEPSY IN JAPAN

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Objective: Benign familial or non-familial neonatal epilepsy (BFNE or BNE) is characterized by neonatal onsets, focal partial seizures, spontaneous remissions in neonates, and normal psychomotor developments. BFNE shows autosomal dominant inheritance and high penetrance. Single nucleotide variations (SNVs) or micro-deletions in *KCNQ2* or *KCNQ3* cording a voltage gated KCNQ potassium channel, are both considered to be possible causes of BFNE or BNE. The proportion of families or patients with mutations has been estimated at above 50% in *KCNQ2*, and less than 7% in *KCNQ3*. Our study presents the frequency of *KCNQ2* and *KCNQ3* mutations in Japanese BFNE and BNE.
Methods: A total 53 patients were recuruited: 37 with BFNE; 9 with BNE; and 7 with unclear family histories.) The SNVs and micro-deletions were identified using Sanger sequencing and MLPA.
Results: We identified *KCNQ2* or *KCNQ3* mutations in 14 patients (26.4%) with BFNE or BNE. While the mutations of *KCNQ2* were identified in 12 patients (22.6%), the mutations of *KCNQ3* were identified in only 2 patients (3.8%). Although the 12 *KCNQ2* mutations included 5 SNVs (41.7%), 3 small indels (25.0%), and 4 micro-deletions (33.3%), both *KCNQ3* mutations were SNVs.
Conclusion: Our study that gene abnormalities other than KCNQ2 and KCNQ3 also cause BFNE and BNE.

G1-10

BENIGN NEONATAL SLEEP MYOCLONUS: OUR EXPERIENCE OF 15 JAPANESE CASES

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Backgrounds: Benign neonatal sleep myoclonus (BNSM) is a self-limited non-epileptic movement disorder that may mimic neonatal seizures. Little is known about Japanese cases of BNSM.
Method: We retrospectively reviewed the clinical manifestations and outcome in 15 patients with BNSM (male 10), including three paired siblings, who were referred to our center between 1996 and 2012.
Results: All were full-term infants (AFD 13, and SFD 2). Pregnancy was uneventful in all but one infant (choroid plexus cyst). The age at onset ranged from day 1 to 18 (mean day 8). The diagnosis of BNSM was based on neonatal onset, characteristic myoclonic jerks that occurred during sleep (videotape review) and normal EEG findings. Prior to referral to our center (3-8 weeks, mean 4.2 weeks), lumbar puncture was performed in 3 infants. Two infants were placed on antiepileptic drugs under a misdiagnosis of neonatal seizure. Family history was positive for febrile convulsion in 4 children, migraine in 3, epilepsy in 1 and hyperthyroidism in 1. During the clinical course, the myoclonic jerks resolved by 6 months in 14 of 15 patients. Of seven patients whose follow-up EEGs were available, one showed epileptiform discharges at the age of 3 years. At the final evaluation (mean 21 months), five patients had developed neurological complications (febrile seizure 1, migraine 2, and speech delay 2).
Conclusion: Inconsistent with the previous reports, our study showed a high incidence of familiar cases. Recognition of BNSM is imperative to avoid needless diagnostic studies, and unnecessary treatments.

G1-11

EPILEPTIC ENCEPHALOPTHY WTH SUPPRESSION-BURST ACTIVITY IN THREE NEONATES

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Objective: Epileptic encephalopathy with suppression-burst in electroencephalography (EEG) in neonates can be associated with a few types of infantile epileptic syndromes.
Method: We present here three newborns with supression-burst activity in neonatal EEG.
Results: First female neonate had erratic myoclonus movements, hiccups, and a suppressionburst pattern in EEG that was compatible with early myoclonic encephalopathy (EME). The seizures were controlled with dextromethorphan (20 mg/kg), and a suppression-burst pattern in EEG was reverted to relatively normal background activity. Second male neonate had tonic spasms with continuous suppression-burst pattern in both waking and sleeping states of neonatal EEG. MRI revealed diffuse cortical pacygria compatible with early infantile epileptic encephalopathy with suppression-burst (Ohtahara syndrome). The seizures responded poorly to several antiepileptic drugs. Third female neonate had alternating focal tonic / focal tonic-clonic seizures, compatible with migrating partial seizures in infancy (MMPEI). Continuous migrating polymorphous focal seizures, combined with multifocal ictal EEG discharges, responded poorly to several antiepileptic drugs. Ketogenic diet was stated with significant seizure reduction in second years of life.
Conclusion: MMPEI shoud be considered between EME and EIEE in neonates with epileptic encephalopathy with suppression-burst in electroencephalography.

G1-12

NEUROLOGICAL PROGNOSIS FOR NEONATAL CEREBRAL INFARCTION

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Objectives: In recent years, advances in diagnostic imaging have increased reports of neonatal cerebral infarction (NCI). It has been suggested that neurological prognosis of NCI is relatively good, but some controversy still exists. To investigate whether NICs are generally without serious / life threatening sequelae or not.
Methods: Between Jan. 2005 and Jun. 2010, we retrospectively analyzed 8 neonates admitted to Fukuoka University Hospital. Considered were clinical information, radiological findings, EEGs, and developmental and seizure outcomes.
Results: Six cases showed neonatal seizures or apnea within 2 days after birth. The remaining two, despite their low Apgar scores, were asymptomatic during the neonatal period (in one case the Apgar score was the result of abruption placentae; in the other the fetus had a monozygotic twin who died before birth). Several months later, however, these two presented with hemiparesis and epileptic seizures. In five of 8 cases NCI lesions were identified in the right middle cerebral artery (MCA) region. Three of the 8 cases showed abnormal EEGs; of these, there was one case of combined mental retardation (MR) and epilepsy, one of combined hemiparesis and epilepsy, and one without neurological damage. Four of the 8 cases showed normal EEGs; two of them being with hemiparesis, and the other two without. One of the 8 cases showed hemiparesis, but EEG was not examined.
Conclusion: In contrast to earlier studies, our results suggest that NCI prognosis is not always favorable. New therapeutic measures are required.

G2-01

CATASTROPHIC INTRACTABLE EPILEPSY IN NEONATES FROM FOCAL CORTICAL DYSPLASIA

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Objectives: Focal cortical dysplasia (FCD) is a major cause of neonatal intractable seizures, often with catastrophic presentation of very frequent seizures and developmental delay; however, predictors of seizure intractability are not well established.
Methods: We retrospectively reviewed features including Neuroimaging and EEGs in two neonates with FCD, who had care for intractable seizures at Children's Hospital Boston.
Results: Common features include 1) presentation within 2 weeks of life, with frequent seizures (>25/day), 2) large dysplasia size, 3) failing ≥3 antiepileptic drugs (AEDs), 4) subsequent developmental delay.
Patient 1 was a girl with right parieto-temporo-occipital FCD, presenting at a few days of life with complex partial seizures of eyelid fluttering, swimming and bicycling movements. Due to incomplete seizure control after first resection at 4 months, repeat resection of FCD was performed at 12 months and with AEDs adjustment she gained complete seizure freedom.
Patient 2 was a boy with left temporo-parietal FCD, presenting at two weeks of age, with tonic spasms, right gaze deviation and eyelid fluttering. Inspite of resection of FCD at 8 months, he had incomplete seizure control.
Initial surgical resection resulted in incomplete seizure control in both cases, which reflects difficulty in delineating the large dysplasia, likely due to limitations of current technology.
Conclusion: The identified features may reflect the severity of epilepsy and difficulty in management of such neonatal focal cortical dysplasia. Although early surgical intervention results in effective seizure control and developmental outcome, there are multiple challenges in management of neonatal catastrophic onset epilepsy from FCD.

G2-02

CLINICAL AND ELECTROENCEPHALOGRAPHIC ANALYSIS OF INFANTILE SPASMS : A MEDICAL CENTER EXPERIENCE

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Objective: Infantile spasms or West syndrome is an age-dependent seizure. It has classic electroencephalographic(EEG) pattern with hypsarrhythmia recorded in both awake and sleep records. However, some of the cases presented with variations of hypsarrhythmia. This study is to clarify the EEG findings and clinical correlation.
Method: We retrospectively review the clinical features and interictal EEG studies of 48 infants diagnosed as infantile spasms in our hospital. We analyzed the basic demographic data, including the age of seizure onset, seizure pattern and EEG findings.
Results: The male-to-female ratio was 28:20. The mean age of seizure was 4.93 month-old. Of 48 infants, 29 cases were symptomatic, 16 were cryptogenic and 3 cases were idiopathatic. Most of the cases presented with flexor spasm (72.9%) while classic salaam pattern (68.8%) was the next. The EEG studies showed typical hypsarrhythmia in 18 cases, atypical hypsarrhythmia in 26 cases while the rest with focal or multifocal epileptic discharges. The atypical hypsarrhythmia included pseudo-periodic discharges resembling burst-suppression (17), asymmetric hypsarrhythmia (3), hypsarrhythmia with persistent focus or increased interhemispheric synchronization (3). Infantile spasms with atypical hypsarrhythmia have a younger age tendency (mostly before 4 month-old) in 15 symptomatic infants.
Conclusion: In our study we found that most of the symptomatic infantile spasms with atypical hypsarrhythmic EEG finding occurred in a younger age. There is a female preponderance of atypical hypsarrhythmia in EEG recording.

G2-03

PRECOCIOUS AND DELAYED NEOCORTICAL SYNAPTOGENESIS IN FETAL HOLOPROSENCEPHALY

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Objectives: Predictable temporal and spatial patterns of synaptophysin expression previously were demonstrated in the normally developing fetal cerebral cortex, reflecting synaptogenesis without regard to neurotransmitter type. We attempt to determine whether timing and distribution are factors in holoprosencephaly (HPE).
Methods: We studied 6 human fetal brains with HPE in the mid-2nd and 3rd trimesters with immunocytochemical antibodies against synaptophysin and other markers of neuronal maturation.
Results: All brains were micrencephalic for gestational age. We found not only abnormal patchy patterns of synaptic vesicle reactivity within the disorganized cortical plate, but also loss of precise timing of genetically programmed synaptogenesis. Precociousness in synaptophysin reactivity relative to age-matched controls was demonstrated in 5 cases, and delayed reactivity in one. Abnormalities were more severe in paramedian than in more lateral regions of the cerebral cortex, following a mediolateral gradient in the horizontal axis. Extrapial heterotopia exhibited early synaptogenesis.
The nodular histological architecture of paramedian zones of cortex contains randomly oriented branching microcolumns of neurones, a unique feature. The hippocampus and subcortical structures did not show precocious synapses, except for arrest in our single case of cortical delay.
Conclusions: Synaptic precociousness occurs often in HPE but is limited to the cerebral cortex; synaptic delay occurs in other cases and is more global. Precociousness may limit synaptic plasticity and contribute to earlier epileptogenic circuitry with a poorer prognosis. Faulty genetic programming alters not only cortical histological architecture but also the timing of onset of synapse formation.

G2-04

USEFULNESS OF EARLY DIFFUION-WEIGHT-IMAGING IN NEONATAL NONKETOTIC HYPERGLYCINEMIA: A CASE REPORT

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Objective: Nonketotichyperglycinemia (NKH) is a disorder of glycine metabolism caused by a defect in the glycine cleavage enzyme system. Glycine is an inhibitory neurotransmitter in the spinal cord and a modulator of excitation at the N-methyl-D-aspartate receptors in cerebral cortex, hippocampus, and cerebellum. In neonatal-onset NKH, neonates present hypotonia, apnea, lethargy, and then intractable epileptic seizures. We present a case of neonatal-onset NKH focusing on EEG and diffusion-weighted-imaging (DWI) in neonatal period.

Patient: A 2-day-old baby boy was transferred to our hospital for evaluation and management of hypotonia and poor sucking. EEG showed a continuous burst suppression pattern. Neurologic examination revealed diffuse hypotonia with symmetrically hypoactive deep tendon reflex. Moro, sucking and rooting reflexes were also poor. His apneic episodes were increased and needed mechanically ventilation for 30 days. MRI revealed partial agenesis of the corpus callosum. In addition, DWI revealed high signal intensity in the posterior limbs of the internal capsules. Later performed MR spectroscopy showed elevated peak consistent with glycine on long echo time. Analyses of blood and cerebrospinal fluid(CSF) amino acids revealed elevated glycine levels and ratios of CSF to plasma glycine. Repetitive EEG recordings showed increased some activities in the suppression phase but his spasms were continued in spite various antiepileptic drug administrations.

Conclusion: High signal intensity in the posterior limbs of the internal capsules on DWI probably reflect the neuropathologic finding of vacuolating myelinopathy in NKH. In addition to characteristic EEG findings, this DWI finding might be the key of early diagnosis of NKH.

G2-05

MOLYBDENUM COFACTOR DEFICIENCY: FOUR CASES FROM TURKEY

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Molybdenum cofactor deficiency is a very rare autosomal recessive inborn error of metabolism, which appears from the early neonatal period with severe mental and motor retardation, feeding difficulties, refractory seizures, and hypotonia. In the neonatal period the patients may be misdiagnosed as having disease such as hypoxicischemic encephalopathy because of their clinical features and MRI findings. Serum uric acid and urine sulphite concentrations are keys to the diagnosis. We present clinical, radiological, electroencephalographic and genetic features of four Turkish patients diagnosed in early infancy.

G2-06

SUCCINATE SEMIALDEHYDE DEHYDROGENASE DEFICIENCY WITH SUBDURAL HAEMATOMA - A CAUSE OF SHAKEN BABY SYNDROME

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Introduction: The shaken baby syndrome commonly produces subdural haematomas without external signs of violence. Some rare metabolic diseases, like glutaric aciduria type 1 and Menkes disease, can sometimes produce subdural haematomas with a nontraumatic origin. We report here an infant with succinate semialdehyde dehydrogenase (SSADH) deficiency whose clinical course was complicated by the development of subdural haematoma.

Case Report: A male infant was the youngest child of consanguineous parents; his elder sister was healthy. There was no family history of neurological disease. Seizures started at one month of age, evolving to intractable focal clonic and asymmetric tonic spasms at 4 months. He had global developmental delay with hypotonia. Urine organic acid chromatography showed high peaks of 4-hydroxybutyric acid. MRI brain was unremarkable. Seizures were partially controlled with vigabatrin and levetiracetam. However, the baby developed sleep disturbances, prolonged episodes of inconsolable crying with dystonic posturing, and refusal to feed with vomiting over the next 3 months. Multiple admissions to investigate and treat these symptoms were unsuccessful. A repeat MRI brain at 8 months of age showed generalised cerebral atrophy with a subacute left fronto-parietal subdural haematoma. The family admitted to vigorous rocking to calm the the baby and put him to sleep, and received counselling and education to change their knowledge and behaviour related to shaking.

Conclusion: SSADH deficiency should be included in the differential diagnosis of subdural haematomas due to nonaccidental head injury.

G2-07

DOES EARLY ELECTROENCEPHALOGRAPHY (EEG) FINDING OF NEONATAL SEIZURES HAVE A PREDICTIVE VALUE TO LONG-TERM OUTCOME?

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Objective: Considering the fact that most neonatal seizures are limited within neonatal period, there are still debates on whether aggressive treatment should be given when the ictal event first occurs. The aim of this study was to determine whether characteristics of EEG recorded shortly after first seizure happened indicate the long-term neurodevelopmental outcomes as well as the presence of post-neonatal seizures (PNS), and to provide an evaluation of prognosis at a certain extent.
Methods: Among 247 infants with clinical neonatal seizures of diverse etiologies admitted to the neonatal ward from January 1997 to December 2009, 158 patients were followed up at an average age of 28 months for their neurodevelopmental outcomes and PNS. We analyzed the relationship between (1)EEG findings together with clinical information and (2)outcomes using binary logistic regression to figure out the predictive factors of prognosis.
Results: Neurodevelopmental 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 ($Sig=0.001$). However, epileptic discharge was not associated with PNS after setting background activity as the stratification variable.
Conclusion: Abnormal outcome occurs more likely in the patient with abnormal EEG background activity or with abnormal cranial image, with or without epileptic discharge. Background activity of EEG is the best predictive factor for long-term prognosis of neonatal seizures (both neurodevelomental outcome and PNS), whereas epileptic discharge is not an independent risk factor of unfavorable outcome in our cohort.

G2-08

NEONATAL SEIZURES AND PROGNOSIS - CORRELATION BETWEEN CLINICO-ELECTROENCEPHALOGRAPHIC MANIFESTATIONS OF NEONATAL SEIZURES AND DEVELOPMENTAL OUTCOMES-

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Objective: The excellent standard of neonatal medicine in Japan assures very low infant mortality. However, there is controversy regarding issues pertaining to neonatal seizures. We focused on prognostic differences among babies with neonatal seizures depending on clinical and electroencephalographic (EEG) manifestations.
Method: We analyzed clinical information, including neonatal seizure symptoms, the existence of seizure discharges and developmental changes in background EEG activities, in 49 patients with possible central nervous system damage. The EEG findings were evaluated based on conventional recordings obtained with a Nihon Koden EEG system, or simultaneous amplitude integrated and conventional EEG recordings made using a Nicoletone monitor for at least 1 hour. Seizure discharges were defined as rhythmic discharges lasting 10 seconds or longer and showing a clear start and finish. We checked developmental outcomes and seizure recurrence during a follow-up period of 68 ±14 months. We also investigated the relationships between outcomes and neonatal seizure states.
Results: Twenty-four of 49 babies had seizures, including clinical only, EEG only and both. Nine patients had both clinical and EEG, 4 only EEG and 11 only clinical seizures. Seven and three patients with EEG seizures, regardless of the presence/absence of clinical seizures, had neurological handicaps and seizures, respectively. Four of these patients had suffered from West syndrome. In contrast, the outcomes of patients without EEG seizures were variable and none had seizure recurrence.
Conclusion: Patients with EEG seizures showed poor developmental outcomes and high rates of seizure recurrence. EEG monitoring in the NICU, thus appears to be valuable.

G2-09

PATTERN OF EEG IN NEONATAL SEIZURES (0-28 DAYS) AND ITS RELATION WITH NEURODEVELOPMENT AT 3 MONTH OF AGE IN FULL TERM NEONATES

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Background: EEG is bedside tool to document seizure disorder. The study was aimed to corelate EEG changes with neurodevelopmental outcome, that will help to understand the prognostic value of EEG in newborns in regards to its neurodevelopmental outcome, hence to initiate early intervention therapy to minimize disability.

Objective: To Study EEG changes in neonatal seizures within 7 days of episode and to corelate these changes with neurodevelopment outcome at 3 month of age.

Materials and Methodology: Total number of 28 cases of neonatal seizures were studied, sample size calculated by temporal association. Only full term neonates (37 to 42 weeks) with documented seizure disorder were included in the study. HIE I cases, babies of mother on anti-epileptics, pre term neonates (<37 weeks), full term neonates (37 to 42 weeks) with undocumented seizure disorder were excluded. The neurodevelopment examination was performed periodically to assess the progress of encephalopathy. Neurodevelopment assessment was done by the DDST II scale.

Results:

- In 17 out of 22 EEG, generalized seizure disorder in form of generalized sharp waves was observed. 5 were normal.
- No developmental delay on DDST at 4 weeks in 10 cases was observed.
- 6 died due to other medical illness at mean age of 11 days.
- 8 cases to be follow up.
- Future developmental follow up 3 month with not only co relate with EEG pattern but also reveal sensitivity and specificity of DDST at 4 weeks of age.

G2-10

FAST ACTIVITY DURING EEG SEIZURES IN NEONATES: CHARACTERISTICS AND SIGNIFICANCE

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Introduction: Paroxysmal fast activity (FA), in the beta and gamma range, has been proposed as a marker for epileptic networks in childhood. Yamazaki (2009) reported interictal FA, on scalp EEG in neonates with hemimegelencephaly. We explore the presence and significance of Ictal FA (IFA) on scalp EEG recorded neonatal seizures (NS).

Methodology: Forty two babies (GA >35w) had NS during VEEG of one hour. Their clinical details have been previously described (Nagarajan 2010). The EEGs were recorded digitally (Compumedics), the NS analysed for ictal FA, with a HFF of 30 Hz (Insight II). The relationship between IFA and outcomes was evaluated .

Results: We analysed 159 NS in 42 babies. IFA was present in 17 babies in 62/69 NS. Five with IFA had electroclinical seizures (ECSz) only, 5 had electrographic seizures (ESz) only and seven had both ECSz and ESz. In the 25 with no IFA the distribution was: ECSz-6, ESz-13, ECSz+ESz-6.

IFA (mostly 30-50Hz), was frequently inconsistent and variable. During a bilateral Sz, IFA was at times unilateral, at times persistent on one side and variable on the other. Two neonates with multiple NS had both IFA + and - events.

There was no significant difference in the background EEG, neuroimaging abnormalities, neurodevelopmental impairment or post neonatal seizures between those with and without IFA.

Conclusions: Ictal FA in NS is often inconsistent and variable. IFA may not be a biological marker for epileptogenic tissue in neonates, because of the different aetiopathogenesis of NS compared to older children and adults.

G2-11

CLINICAL & ELECTROPHYSIOLOGICAL CHARACTERISTICS OF FULL-TERM NEONATAL SEIZURES CONFIRMED BY ELECTRICAL SIGNATURE

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Purpose: Neonatal seizure is the most significant parameter of neurological insult, however its pathophysiology and diagnostic consensus are still controversial. We performed this study to investigate the characteristics of electroencephalogram (EEG) in neonatal seizures, to validate the efficacy of radiological studies, and to estimate the occurrence of further subsequent epileptic seizures.
Methods: Seventeen patients with full-term neonatal seizure confirmed by electrical seizure were enrolled. Mean birth weight is 3.29 kg, and mean duration of follow-up is 22.7 months. Medical records and EEG were retrospectively reviewed.
Results: Subtle seizure is the most common seizure type: subtle seizure in 6(35%), subtle seizure with focal clonic seizure in 2(12%). In 12 patients (71%), abnormal background activities were observed, and trace discontinua is the most common abnormal finding. Ictal EEGs were mostly localized into unilateral posterior quadrant in 11 (64%), however, the remained were localized into the frontal area. Brain USG did not reveal any abnormal finding in 9 patients (53%), whereas brain MRI didn't in 4 patients (25%). Brain MRI found abnormal findings in 5 out of 9 patients with negative brain USG result. Subsequent epileptic seizures followed in 7 patients (44%).
Conclusion: Background activity is still useful as an indirect marker of neonatal seizure. Different generation or propagation mechanism can be suggested in that ictal EEGs were often localized into the frontal area in minor population. Brain MRI is more sensitive than brain USG, especially in case of cerebral infarct or hypoxic-ischemic encephalopathy.

G2-12

EPILEPSY IN A NEONATE WITH MIGRATION DISORDER ASSOCIATED WITH MECKEL'S DIVERTICULUM –A NEW SYNDROME? OR SPORADIC CASE

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Case Report: The timing of the migration period are believed to range from the 6th or 7th until the 20th or 24th gestational week. And Meckel's diverticulum is the most common anomaly of the residual omphalomesenteric (vitellointestinal) duct which normally becomes occluded and vanished completely by weeks 8-10 of gestation. Here we report a girl who had facial dysmorphism, neonatal seizure and migration disorder in terms of polymicrogyria and type 1 schizencephaly (fused lip) at her neonatal period and she started to suffer from massive bloody stool and was diagnosed as Meckel's diverticulum at her age of 10 months old. To our knowledge, this is the first case of migration disorder associated Meckel's diverticulum, that may be a sporadic case. However, based on the coordination of embryonic developmental stage in between migration disorder and Meckel's diverticulum this case may imply that the unknown insult(s) (environmental, infectious or genetic factor(s)) occurs during this concurred period. Nevertheless, further genetic study should be investigated.

G3-01

MALIGNANT MIGRATING PARTIAL SEIZURES IN INFANCY CONTROLLED WITH CLORAZEPATE

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Background: Malignant migrating partial seizures in infancy (MMPSI) constitute a rare form of epileptic encephalopathy of early infancy characterized by intractable multifocal seizures. MMPSI has recently become a topic of interest because a relationship with *KCNT1* gene mutation was found. We report the effective use of clorazepate (CLZ) in a patient with MMPSI.
Case Description: An 8-month-old girl began having seizures at 2 months of age. She had been born full-term by normal delivery without neurological events. She showed normal development before the seizure onset and was capable of both smiling and ocular pursuit. Her older half-brother had benign infantile convulsions. Focal polymorphous seizures soon became very frequent, occurring in clusters of 20-30 several times a day. She then showed arrest of psychomotor development with deterioration. Investigations found no evidence of a storage disorder, nor any identifiable metabolic and neurodegenerative disorders. Brain magnetic resonance imaging indicated mild frontal atrophic changes but no structural malformations. Ictal electroencephalography showed seizures originating from different areas of the same or the opposite hemisphere independently. She was given a diagnosis of MMPSI and underwent genetic testing for *KCNT1*. She was treated with multiple conventional anti-epileptic drugs including potassium bromide and Vitamin B6, but none showed any efficacy and even a ketogenic diet proved to be ineffective. Phenobarbital, Valproate and Nitrazepam were minimally effective. After starting CLZ, the seizures decreased to just a few per day.
Conclusion: These observations suggest that CLZ should be used in patients with MMPSI as it may exert somewhat greater efficacy than the other types of drugs currently available.

G3-02

ISCHEMIA-MODIFIED ALBUMIN LEVELS IN CHILDREN HAVING SEIZURE

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Objective: Convulsions are one of the frequently seen problems for a neurologist in the daily routine. It is difficult to distinguish the seizure from pseudo-seizure because of lack of conclusive tests. The aim of this study is to investigate the relationship between seizure types and seizure periods by studying IMA serum levels in children having seizure.
Method: Two groups were included in our study. The patient group consisted of the children admitted to Pediatric Emergency Care during January 2008-January 2010 with seizure and the control group consisted of healthy children.
Results: Serum Ischemia modified albumin (IMA) level in the group having seizures was 99.7U/ml and 83.2U/ ml in the control group. In the comparison of the patient and control groups, significant differences were found between their IMA values (p=0.000). There was a significant difference between IMA values of the group having generalized tonic-clonic seizures and those of the control group (p=0.001). In comparison of the IMA values of the group having febrile convulsions and those of the control group, a significant difference was determined (p=0.011). Additionally, It if the seizure was prolonged over 5 minutes, IMA level increased, and there was a significant difference between the groups experiencing over 5 minutes of seizures and the groups experiencing less than 5 minutes (p=0.001).
Conclusion: An increase in IMA levels in febrile convulsion supports the hypoxia development in the brain during the seizure. Serum IMA levels increased with the elongation of the seizure period and may be an indicator for status epilepticus.

G3-03

A NEW SYNDROME OF MICROCEPHALY WITH SIMPLIFIED GYRATION, WEST SYNDROME AND INFANTILE DIABETES

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Objectives: 40% of patients with epilepsy are estimated to have a genetic cause but only a small number of genes have been identified. West syndrome is the most common form of epilepsy causing neurological impairment in children under 1 year with so far only 4 genes associated. Microcephaly with simplified gyration (MSG) is a rare malformation of cortical development, not always associated with severe epilepsy and although apoptosis is thought to be a cause this is not described in patients. Infantile onset permanent diabetes (PND) is caused by inappropriate apoptosis of pancreatic beta cells e.g. in Wolcott-Rallison syndrome (WRS). We have collected two families with this rare phenotype of West syndrome, MSG and PND and aim to identify the novel genetic mutation.
Results: Genome Wide Linkage Analysis revealed a region on chromosome 18 with a significant LOD score of 4.3 linked to two patients from separate families. In this area, two homozygous non-conservative missense mutations in a novel gene were found in patients, but not in 300 ethnically matched controls, and were predicted as pathogenic. The gene is highly expressed in foetal brain and pancreas. Patient fibroblasts and gene knockdown experiments showed an increased susceptibility to apoptosis under stress conditions and an autopsy specimen showed increased apoptosis in the cerebral cortex.
Conclusion: We have identified a new epileptic syndrome with MSG, epilepsy and infantile diabetes caused by novel mutation with apoptosis as the causal mechanism. This works sheds light on the mechanisms of brain development and on the pathogenesis of West syndrome.

G3-04

CLINICAL PRESENTATION, TREATMENT AND PROGNOSIS IN CHILDREN WITH REYE-LIKE SYNDROME

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Objectives: We performed a retrospective study to explore the mortality rates and prognosis of the Reye like syndrome in patients treated at Konya Research and Education Hospital.
Method: Twenty two children with ages between 5 months and 7 years old were included in this study. All patients were treated with intensive supportive methods to manage body fluid, blood circulation, respiration, body temperature, and intracranial pressure.
Results: The main presenting features were history of fever (72.7%), profuse vomiting (63.6%), abnormal behavior and agitation (77.2%), and sudden onset of unconsciousness (100%). The etiologies of patients included viral illness, gastroenteritis, metabolic disorders, intoxication and hypoxia due to foreign body aspiration. No neurological deficit was seen in the children who survived the disease. In our patients the mortality rate was 31.8%.
Conclusion: Reye like syndrome occurs only rarely but should be a part of the differential diagnosis of any encephalopathy of unknown origin and above all if there is a history of ingestion of drugs, previous viral infection and vomiting. Our treatment protocol is safe and effective in children with Reye like syndrome.

G3-05

DEVELOPMENTAL OUTCOME OF CHILDREN WITH WEST SYNDROME

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Introduction: Biotinidase deficiency is an autosomal recessive disorder due to deficiency of the biotinidase enzyme. It is known to have varied presentations, the classical presentations being progressive neurological deterioration, cutaneous involvement and biochemical abnormality (metabolic acidosis, ketonuria and mild hyperammonemia). Being a treatable disorder a high index of suspicion is needed for identifying such patients

Materials and Methods: This prospective study was carried out at the Department of Pediatric Neurology, Kanchi Kamakoti Childs Trust Hospital, Chennai, India. A total of 8 patients with biotinidase deficiency were identified and their relevant clinical characteristics, laboratory, findings, neuroimaging and their neurological and developmental assessment at follow up was analyzed.

Results: 7 of the 8 patients presented with seizures beginning from 12 hours of life till 5 months of life. 5 of these were on 3 or 4 anticonvulsants prior to initiating biotin therapy. Skin or hair manifestations were present in 4 of these patients while the remaining did not have any skin manifestations. Hyperammonemia and acidosis were not seen in any of our patients while one had elevated iso-valerylcarnitine on metabolic screen. These patients remain seizure free and developmentally and neurologically normal at follow up on biotin therapy alone the oldest child being 7 years now.

G3-06

NEURODEVELOPMENTAL AND EPILEPSY OUTCOME IN CHILDREN AGED ONE TO FIVE YEARS WITH INFANTILE SPASMS ONE OR MORE YEARS AFTER ONSET – A CROSS-SECTIONAL STUDY

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Objective: There is paucity of outcome data of children with infantile spasms from India and other developing countries . Most results of developmental assessment in previous studies were often based on clinical impression and school placement rather than standardized, ageappropriate psychometric testing. Therefore, the present study was planned to objectively evaluate the outcome of these children treated with the current standard treatment option.

Methods: Ninety five children, aged one to five years under follow up for more than six months in Pediatric Neurology Clinic with the diagnosis of infantile spasm were enrolled in this crosssectional study if they had completed one or more years after the onset of spasms.

Neurodevelopment of each child was assessed using Development Profile III (DP III) and Gross Motor Function Classification System (GMFCS). History regarding epilepsy frequency and control in the last one year was taken using E-Chess scale.

Results: The mean age at onset of spasms was 5.6 ± 3.7 months. Perinatal factors were the commonest etiology in 54/95 children (56.8%). Favorable neurodevelopmental outcome was observed in 8/95 (8.4%) patients only. None of the assessed variables affected neurodevelopmental outcome significantly.Favorable epilepsy outcome was observed in 58/95 (61.1%) patients. On multivariate analysis, it was significantly associated with treatment lag ≤ 3 months between apparent onset of spasms and institution of therapy {OR (CI) 2 (1.1-3.8)} and response to first line antiepileptic drug {5 (2.6-10)} when adjusted for age.

Conclusions: The commonest etiology of infantile spasms noted were potentially preventable perinatal causes. Favorable Neurodevelopmental outcome was infrequent. Early appropriate treatment may have a favorable epilepsy outcome.

Key words: Infantile spasms, outcomes, neurodevelopment, epilepsy, motor

G3-07

PROGNOSTIC UTILITY OF CLINICAL EPILEPSY SEVERITY SCORE IN CHILDREN WITH INFANTILE SPASMS VERSUS KRAMMER'S GLOBAL SCORE FOR HYPSARRHYTHMIA SCORING BEFORE INITIATING THERAPY

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Introduction: Infantile spasms is an age dependent epileptic encephalopathy. The present study was done to assess whether clinical scoring of epilepsy severity or pretreatment EEG scoring of hypsarrhythmia can predict favorable epilepsy outcome.

Methods: Thirty three children, aged one to five years under follow up for more than 6 months, with the diagnosis of infantile spasms (ILAE), whose pre-appropriate treatment digital EEG records were available, were enrolled. Demographic, semiology related, etiological and therapy related variables were evaluated. Epilepsy severity in last 6 months was assessed using Early Childhood Epilepsy Severity Score (ECHESS) and EEG records were analyzed using Krammer's criteria.

Favorable epilepsy outcome was defined as seizure free or ≤ 1seizure/ month in last 3 months.

Results: Mean age at assessment was 25.9 ± 12.2 months with 87.8% males. The semiology of spasms was flexor (75.8%), extensor (9.1%) and mixed (15.2%). Perinatal asphyxia was causative in 54.5% children. 45.5% children had favorable epilepsy outcome. The presence of spikes at frequency of less than one per second in hypsarrhythmia correlated with favorable epilepsy outcome on multivariate analysis. Krammer's Global score (≤ 8) could predict favorable epilepsy outcome with high sensitivity of 100 (C. I. 78-100) and specificity of 94.4 (C.I. 72.6-99). Early childhood epilepsy severity score (E-Chess ≤ 9) in last one year was associated with favorable epilepsy outcome (p=0.001) and favorable motor outcome (p=0.02).

Conclusion: Krammers' EEG scoring at onset of spasms and epilepsy severity quantified with E-Chess may help in predicting epilepsy outcomes in children with West syndrome.

G3-08

LONG TERM EPILEPSY OUTCOME OF CHILDREN WITH WEST SYNDROME: RETROSPECTIVE ANALYSIS OF 135 CHILDREN TREATED AT A TERTIARY CARE CENTRE

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Background: West syndrome has a heterogeneous etiology and variable course. This study was undertaken to describe the clinical characteristics, treatment received and the epilepsy outcome of children with West syndrome in a developing country.
Methods: The case records of 135 children with west syndrome enrolled in Pediatric Neurology Clinic at a tertiary care referral centre in north India, from January 2009 to March 2011, were reviewed retrospectively.
Results: The mean age at presentation was 26.6 months (SD: 14.1 months) with 72% males. The mean age of onset of spasms was 5.5 months (SD: 4 months). The semiology of spasms was flexor (74.3%), extensor (13.2%) and mixed (12.5%). 81.5% patients were symptomatic West syndrome. Etiology could not be found in rest of the children. Underlying etiologies: hypoxic ischemic sequelae (56.3%), CNS infections (10.4%), cerebral malformations (4.5%), Tuberous sclerosis (3.7%). 80% children received hormonal therapy.
55.6% patients showed resolution of spasms, 13.3% children evolved to Lennox-Gastaut syndrome, 10% developed other seizure types,11.1% showed relapse of spasms after stopping hormonal therapy and 8.9% patients had persistent spasms at last follow up. 44% children with Lennox-Gastaut syndrome had hypoxic ischemic sequalae and 10.5% children with hypoxic ischemic sequalae developed Lennox-Gastaut syndrome.
Conclusions: Hypoxic ischemic sequalae and CNS infections are the major causes of west syndrome in a developing country. Half of these patients show resolution of spasms with appropriate therapy. A fraction of these children progress to Lennox-Gastaut syndrome. Early identification, aggressive management and good supportive care are critical for favourable epilepsy outcome.

G3-09

CLINICAL CHARACTERISTICS OF EPILEPSY AFTER NEONATAL ARTERIAL ISCHEMIC STROKE

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Objective: There are few reports concerning epilepsy after neonatal arterial ischemic stroke in Japan. The aim is to evaluate the clinical characteristics of epilepsy after neonatal arterial ischemic stroke.
Methods: This study was retrospective, and 16 patients (10 males and 6 females) were enrolled. The follow-up duration of all the patients was more than four years in this study. Neonatal arterial ischemic stroke was diagnosed after birth and on or before the 28th postnatal days. We divided the patients into two groups: epilepsy group (3 patients) and non-epilepsy group (13 patients). Two groups were compared about clinical manifestations, neuroimaging findings, and neurological outcomes. In addition, we described the clinical course of epilepsy in epilepsy group.
Results: There were no statistic differences about maternal factors, gestational age, delivery form, Apgar Score, birth weight, and lesions shown by neuroimaging findings between two groups. As neurological outcomes, the ratio of mental retardation in epilepsy group was higher than in non-epilepsy group ($p<0.05$). Patients with epilepsy had seizures at the age of 5.6 years (range 5.2-7.1 years). Their duration of treatment was 7.6 years (range 2.6-8.9 years), and none of them were seizure-free instead of using more than two antiepileptic drugs.
Conclusion: The frequency of epilepsy after neonatal arterial ischemic stroke was 18.8% and was higher in male. These results were compatible with previous reports. Mental retardation may be associated with developing epilepsy in patients with neonatal ischemic stroke. Furthermore, their epileptic seizures tended to be intractable.

G3-10

ROLE OF HYPOXIA IN THE GENESIS OF EPILEPSY IN CHILDREN

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Objective: To study the role of hypoxia in the genesis of epilepsy in children.
Methods: 150 children (M=91, F=59) under 2 years of age with varying degrees of hypoxic damage to the nervous system.
Results: I group - children with mild hypoxic damage to the nervous system - 13,3% (n = 20), II group - 23,33% (n = 35), III group - 63,33% (n = 95). Convulsive syndrome was not detected in this group. In group II children were born with gestational age 36-38 weeks, he pregnancy was against the background of anemia II-III degree, threatened abortion, 28,6% (n =10) children were born by Caesarean section. Score at birth by Apgar 3-5 points. Convulsive syndrome was diagnosed in 14,2% (n = 5) with a predominance of boys and submitted by local or generalized muscle contractions, vegetative-visceral disorders, tonic-clonic seizures accompanied by specific changes in the EEG. III group consisted of children were born with gestational age 28-38 weeks, weighed down by obstetrical history, were born by Caesarean section - 47,4% (n = 45) children. Score at birth by Apgar 1.3 points. Convulsive was diagnosed in 17,9% (n = 17), with a predominance of boys and characterized by polymorphic seizures: tonic, clonic (focal, multifocal, generalized), myoclonia with vegetative-visceral disorders accompanied by specific changes in the EEG.
Discussion: The revealed dependence allows you to mark a significant role of hypoxia in the early stages of ontogenesis, considering as a major factor in the realization of epilepsy with a delay rate of neuropsychological development.

G3-11

SHAKEN BABY SYNDROME MANISFESTING AS INFANTILE SPASMS : A CASE REPORT

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The diagnosis of child maltreatment leading to head injury is challenging. Here, we present the case of a 3-month-old female infant who presented with focal seizures that lasted for several minutes. After admission, she began to show intermittent clusters of head nods, irritable crying, arching, writhing, stiffening, and jerking of both arms.
These results and electroencephalography (EEG) findings were attributed as the diagnosis of infantile spasms (IS). Brain computed tomography (CT) and magnetic resonance imaging (MRI) revealed the presence of chronic subdural hematoma (SDH) mixed with acute ischemic injuries. Examination of the eye fundus confirmed the presence of retinal hemorrhage. Therefore, all evidence pointed to a diagnosis of shaken baby syndrome (SBS).
She was discharged on day 10 with complete recovery. After 3 months, follow-up brain MRI showed resorption of the bilateral subdural hematomas. Magnetic response spectroscopy sampling in bilateral hippocampi showed a similar appearance with decreased N-acetyl aspartate peak height. EEG showed better background organization and reduction in epileptiform activity, but persistent slow activity in the right parietal area. Based on this case, we suggest that physicians should consider a diagnosis of SBS for children with new-onset IS and that should be evaluated, diagnosed, and treated as promptly as possible.

G4-01

MUTATION SCREENING OF THE GABRG2 GENE IN KOREAN PATIENTS WITH CHILDHOOD ABSENCE EPILEPSY

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Objective: Since the γ-aminobutyric acid type-A receptor subunit γ2 gene (*GABRG2*) mutation was discovered in an Australian family with childhood absence epilepsy (CAE) and febrile convulsions, a few screening studies for the *GABRG2* mutation have been conducted in sporadic individuals with CAE from other ethnic groups. The aim of this study was to determine whether or not the previously reported genetic mutations and single-nucleotide polymorphisms (SNPs) of *GABRG2* can be reproduced in sporadic Korean individuals with CAE, compared to healthy Korean individuals.
Methods: Thirty-five children with CAE in Chonnam National University Hospital and healthy controls (n=207) were enrolled, and the medical records of patients with CAE were reviewed. CAE was diagnosed according to the Classification and Terminology of the International League Against Epilepsy. All nine exons of *GABRG2* were directly sequenced.
In addition, the two SNPs found in our CAE patients were analyzed: C315T in exon 3 (E3) and C588T in exon 5 (E5). The frequencies of the two SNPs in the CAE patients were compared with data from healthy controls (for E3 and E5) and from previously reported Korean population data (only for E3).
Results: No mutation of *GABRG2* was found in our CAE patients. In addition, the allele and genotype frequencies of the two polymorphisms did not differ significantly between CAE patients, healthy controls, and the Korean general population (P>0.05).
Conclusion: Our study of sporadic Korean individuals with CAE found no evidence that *GABRG2* contributes to the genetic basis of CAE.

G4-02

A NOVEL PCDH19 MUTATION IN A PATIENT WITH MICROCEPHALY, INFANTILE MYOCLONIC EPILEPSY AND PSYCHOMOTOR RETARDATION

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Introduction: Mutations in the protocadherin 19 gene (*PCDH19*) has been reported to cause female-restricted epilepsy with mental retardation. We report the identification of a novel missense mutation in *PCDH19* gene from a female patient presenting unusual phenotype of myoclonic epilepsy, microcephaly and severe psychomotor retardation.
Patient: The 2 years and 6 months old girl was the second-born of healthy, unrelated parents with a birth weight of 2,300 gm after gestation of 36 weeks. She had microcephaly and her development was severely delayed. Clusters of afebrile focal seizures developed when she was 4-month-old and was changed to myoclonic seizures a year later. Neurological examination showed muscle hypotonus and autistic behavior. Brain MRI revealed diffuse cerebral atrophy and corpus callosum hypoplasia. Mutational analysis on *PCDH19* disclosed a missense mutation of c.2797 C>T (p.R933C) localized to a conserved region (CM1) within the cytoplasmic domain.
Discussion: The phenotypes attributed to mutations in *PCDH 19* were variable. Early seizure-onset, intractability of seizures, intellectual disabilities and behavioral problems were commonly observed. However, myoclonic seizure was extremely rare and microcephaly with brain atrophy has never been reported previously. Our case report further extended the mutational spectrum and clinical presentations related to *PCDH19*-associated epilepsy in females.

G4-03

CONTINOUS EEG MONITORING IN PEDIATRIC INTENSIVE CARE UNIT IN TAIWAN

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Objective: Continuous electroencephalogram monitoring is a powerful tool for evaluating cerebral function in many neurologic diseases, which is especially useful when the clinical examination is limited. The study presents a description of children and adolescents with acute neurological disorders monitored by continuous electroencephalogram.
Method: Retrospective review of 73 children who underwent continuous electroencephalogram monitoring in pediatric intensive care unit in Chang Gung Children's Hospital between October 2007 and November 2010. Baseline demographic, indication, outcome and electroencephalogram findings were recorded.
Results: 60 patients were monitored for detection of nonconvulsive seizures and characterization of spells in patients with altered mental status, including a history of epilepsy (n=17), fluctuating level of consciousness (n=10), acute brain injury (n=17), recent convulsive status epilepticus (n=11) and stereotyped activity (n=5). 13 patients were monitored for ongoing therapy of refractory status epilepticus. In altered mental status group (n=60), the electroencephalogram finding showed negative (n= 9, 15%), cortical dysfunction (n=14, 23.3%), nonconvulsive seizure (n=19, 31.7%), nonconvulsive status epilepticus (n=12, 20%), electrocortical silence (n=6, 10%).
Conclusion: In the pediatric intensive care unit, the most important indication for continuous electroencephalogram monitoring is patients with altered mental status for detection of nonconvulsive seizures and characterization of spells. Recognizing altered mental status children with critical neurological disorders early is important for the prognosis and perhaps for initial treatment. Therefore, continuous electroencephalogram monitoring should be part of the pediatric intensive care among altered mental status children with critical neurological disorders.

G4-04

NEIGHBORHOOD, FAMILY AND RISK OF CHILDHOOD AND ADOLESCENT EPILEPSY: A NATIONWIDE EPIDEMIOLOGICAL STUDY FROM SWEDEN

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Objective: To examine whether neighbourhood deprivation increases the risk of hospitalisation for childhood and adolescent epilepsy, after accounting for family- and individual-level sociodemographic characteristics.
Design: An open cohort of all children aged 2 to 17 years was followed between January 1, 2000 and December 31, 2010. Children residential addresses were geocoded and classified according to neighbourhood deprivation. Data were analyzed by multilevel logistic regression, with family and individual-level characteristics at the first level and level of neighborhood deprivation at the second level.
Results: During the study period, among a total of 1,020,766 children, 9354 (0.9%) were hospitalized with childhood and adolescent epilepsy. Age-adjusted hospitalized rates for childhood and adolescent epilepsy increased with increasing level of neighbourhood deprivation. In the study population, 8.7 per 1000 and 10.0 per 1000 children in the least and most deprived neighbourhoods, respectively, were hospitalised with childhood and adolescent epilepsy. Incidence of hospitalisation for childhood and adolescent epilepsy increased with increasing neighbourhood-level deprivation across all family and individual-level sociodemographic categories. The odds ratio (OR) for hospitalisation for childhood and adolescent epilepsy for those living in high-deprivation neighbourhoods versus those living in low-deprivation neighbourhoods was 1.15 (95% confidence interval=1.07–1.23). High neighbourhood deprivation remained significantly associated with odds of childhood and adolescent epilepsy after adjustment for family- and individual-level sociodemographic characteristics (OR=1.13, 95% confidence interval=1.05–1.22, p=0.001).
Conclusions: This study is the largest so far on neighbourhood influences on childhood and adolescent epilepsy. Our results suggest that neighbourhood characteristics affect the risk of hospitalization for childhood and adolescent epilepsy independently of family- and individual-level sociodemographic characteristics.

G4-05

HYPERBARIC OXYGEN THERAPY IN CHILDREN WITH HYPOXIC ENCEPHALOPATHY USING VENTILATOR - A MEDICAL CENTER EXPERIENCE IN TAIWAN

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Objective: Hyperbaric-oxygen therapy (HBO) is recommended for patients with acute carbon monoxide (CO) intoxication, particularly if they have lost consciousness or have severe intoxication associated with vital organ insult. Some indications for neurological-related disorders are fairly well recognized such as brain abscess and sudden hearing loss. But many are supported only by experimental studies or rare case reports, e.g. post anoxic encephalopathy and Radio-induced CNS lesion. Nevertheless limited reports for pediatric patients suffered from hypoxic-ischemic encephalopathy (HIE) had been addressed.
Method: The subjects were 10 children range of age: 5M~17Y; gender, male: 6; female: 4) for outcome analysis in recent 3 years. All are undergone mechanical ventilator.
Results: We observed prolonged hypoxic course may lead to resistant to HBO therapy and grave outcome eventually. One whole recovery, 1 moderate neurologic sequela (tremor but ventilator weaning), 2 moderate handicap (home-care ventilator); 1 severe handicap (ventilator weaning) and grave consequences of remained 5 victims showing vegetative status and ventilator dependence. Four of eight patients' family (4/10) displayed satisfaction for HBO therapy.
Conclusion: We revealed prolong hypoxic course associated with resistant to HBO therapy and eventually grave outcome. Nevertheless, more scientific studies are needed to conduct the exact efficacy of HBO for pediatric HIE indications.

G4-06

AGE-DEPENDENT NEUROLOGIC VULNERABILITY TO CYCLOSPORINE AMONG CHILDREN

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Objective: Cyclosporine (CsA) is a potent immuno-suppressant known for its neurotoxicity. Neurologic complications of CsA include seizures and encephalopathy in the most severe form and are more severe in children than in adults. This study hypothesizes that younger children are more vulnerable to CsA-associated neurotoxicity than older ones in terms of occurrence rate, acute presentations, and long-term outcomes.
Methods: Pediatric patients who received CsA in the hospital between 1988 and 2011 were retrospectively reviewed. The clinical presentations, demographic data, and laboratory examinations were analyzed through t-test for numerical and Fisher's exact test for categorical variables. Exact logistic regression was used to examine the effect of each variable.
Results: Twelve (8.2%) of the enrolled 146 patients developed CsA neurotoxicity. Compared to the non-neurotoxicity group, the neurotoxicity group was significantly younger upon onset of CsA (p=0.008) and had significantly higher percentages of hypertension after CsA treatment (p=0.01). Regression analysis showed that age <6 years (odds ratio 7.62, 95% confidence interval 1.58-51.46; p=0.007) and hypertension after CsA (6.26, 1.35-35.40; p=0.016) were significantly associated with CsA neurotoxicity. Younger children were prone to have more severe seizures in the acute stage and worse neuro-behavioral outcome than older children. Follow-up showed frontal/parietal cerebral atrophy in all examined children <6 years of age.
Conclusions: Children <6 years old have higher risk of CsA neurotoxicity, more severe acute presentations, worse long-term neurologic outcomes, and persistent frontal/parietal atrophy compared to older children. These findings suggest an age-dependent susceptibility of CsA neurotoxicity in the pediatric population.

G4-07

CORRELATION BETWEEN DIFFUSION TENSOR IMAGING AND DEVELOPMENTAL PROGNOSIS IN CRYPTOGENIC WEST SYNDROME

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Objectives: To elucidate the correlation between diffusion tensor imaging and the developmental prognosis in cryptogenic West syndrome.
Methods: We studied eight patients referred to our hospital in whom cryptogenic West syndrome had developed between June 2006 and September 2008. We classified them into two groups: Group MR included two patients whose developmental quotients (DQs) were under 70 at the age of 1 year, and Group N included six patients whose DQs were 70 or higher. We also studied eight age-matched controls (Group C). Patients underwent 3T-MRI before ACTH therapy and at the age of 1 year. We visualized the corticospinal tract (CST) and inferior longitudinal fasciculus (ILF) bilaterally and commissural fibers passing through the corpus callosum (CF) using tractography. We set regions of interest (ROIs) on these fibers and calculated the fractional anisotropy (FA) for each ROI.
Results: The FA values in all of the fibers before ACTH therapy were similar among the three groups. At the age of 1 year, for all of the fibers except the left CST, the FA values were lower in Group MR than in Groups N and C. The FA values were similar in Groups N and C at the age of 1 year.
Conclusions: The FA values in the CST, ILF, and CF were similar at the onset of cryptogenic West syndrome, independently of the developmental prognosis. At the age of 1 year, however, all the FA values were lower in the patients with poor developmental prognoses, except for the left CST.

G5-01

A SURVEY ON THE USE OF NEWER ANTIEPILEPTIC DRUGS FOR NEONATAL SEIZURES AMONG FILIPINO PEDIATRIC NEUROLOGISTS

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Background: Neonatal seizures remain a major clinical problem wherein conventional antiepileptic drugs (AEDs) show limited efficacy. Previous studies showed high incidence of off-label drug therapy in neonates.
Objectives: We sought to establish if local pediatric neurologists were recommending and using newer antiepileptic drugs in the management of neonatal seizures.
Methods: Surveys were distributed at the 2008 Biennial Convention of the Child Neurology Society of the Philippines (CNSP).
Results: Among the listed 77 members of the society, 63 participated in the survey. They comprised 82% of all the Filipino pediatric neurologists. Seventy-three percent (46/63) recommended treatment of neonatal seizures with the newer AEDs as add-on therapy. Majority (42/63; 67%) of the respondents considered the newer AEDs to be effective. Among the new AEDs, topiramate (40/46; 87%) and levetiracetam (10/46; 22%) were the most commonly used. Even with paucity of data on neonatal pharmacokinetics of these drugs, the Filipino pediatric neurologists made different dosing recommendations. Adverse effects were recognized only with topiramate.
Conclusion: These results were concurrent with the published data abroad and underscore the need for thorough and meticulous studies of the newer AEDs in neonates to understand the risks and benefits of new drug therapies for neonatal seizures.

G5-02

NEONATAL STATUS EPILEPTICUS CONTROLLED WITH LEVETIRACETAM

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Objective: Sturge-Weber syndrome is a rare, sporadic, congenital neurocutaneous syndrome characterized by facial cutaneous vascular malformation, leptomeningeal angioma and eye abnormalities.
Method: Seizures develop during the first year of life, may become refractory to multiple anticonvulsants and status epilepticus may develop.
Result: A rare subtype of Sturge-Weber syndrome with bilateral facial vascular malformation, unilateral cerebral involvement and neonatal status epilepticus is reported here.
Conclusion: Neonatal status epilepticus was successfully controlled with intravenous levetiracetam infusion.

G5-03

HIGH DOSE INTRAVENOSIS LEVATIRACETAM IN EARLY MYOKLONIC ENCEPHALOPATHY DUE TO NON KETOTIC HIPERGLYCINEMIA

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Non ketotic hyperglycinemia is a rare inborn error of glycine metabolism due to deficient activity of glycine cleavage system, a multienzymatic complex consisting of four protein subunits: the P-protein, the H-protein, the T-protein and the L-protein. The neonatal form of non ketotic hyperglycinemia presents in the first days of life with encephalopathy, seizures, multifocal myoclonus and characteristic "hiccups".The suppression-burst pattern is EEG characteristic. Rapid progression may lead to intractable seizures, coma and respiratory failure requiring mechanical ventilation.
Clinical trial with scavenges drugs decreasing glycine levels such as sodium benzoate, and with drugs reducing NMDA receptors excitatory properti es, such as ketamine and dextromethorphan, have been tried but the outcome is usually poor.Intravenosis levetiracetam has been successfully tried for refractory epilepsy in pediatric patients.
We report two cases affected by neonatal non ketotic hyperglycinemia and early myoclonic encephalopathy who have suppression-burst pattern at EEG treated with high dose intravenosis levatiracetam . In our patients determined dramatic reduction of seizures and improved EEG.

G5-04

VARIATION OF THERAPEUTIC HYPOTHERMIA PRACTICES IN U.S. NEONATAL INTENSIVE CARE UNITS: A NATIONAL SURVEY

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Objective: In 2005, a study from this group indicated that just 6% of institutions used therapeutic hypothermia (TH) for the treatment of hypoxic-ischemic encephalopathy (HIE) in neonates. Among those institutions that used TH there were wide variations among protocols for its use. The objectives of this study were to determine the extent to which TH has been incorporated into practice since our previous survey and to identify whether TH practices more closely and consistently reflect those shown to be effective in the medical literature.
Methods: In July 2011, a survey was sent to the directors of the same U.S. neonatal intensive care units (NICUs) queried in our 2005 study. These responses were compared to those of 2005 using appropriate statistical analysis.
Results: Completed surveys were received from 330 NICUs (41%) with similar demographics to the 2005 survey. Fifty percent of respondents use TH compared to the 6% reported previously, with the majority of centers not using TH transferring their neonates to centers that do. Of interest was the extent to which certain TH practices (imaging, EEG monitoring, transport, and gestational age) varied among the respondents' centers and deviated from the evidence published in the medical literature.
Conclusions: TH has become standard of care, with the majority of institutions either offering the therapy themselves or transferring neonates to an institution that does.
Variation does still exist in areas lacking sufficient research, particularly in regards to imaging, EEG monitoring, transport and gestational age. Further research into these areas is needed.

G5-05

EFFECTS OF LAMOTRIGINE ON A PATIENT WITH EARLY MYOCLONIC ENCEPHALOPATHY

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Background: Early myoclonic encephalopathy (EME) is a rare and severe childhood epileptic syndrome characterized by intractable seizures, onset during the first 3 months of life, and a suppression-burst EEG pattern. Most patients are given an unfavorable prognosis, and neither conventional antiepileptic drugs (AEDs) nor adrenocorticotrophic hormone (ACTH) are effective. We report an EME patient who experienced significant reduction in seizure frequency with lamotrigine (LTG), one of a group of newer AEDs. In our case, the use of LTG showed severely abnormal EEGs return to nearly normal.
Case: The patient was a two-year-old boy delivered without accident at 39 weeks of gestation. His weight was 3,306 g. On his first day of life, he showed transient tonic spasms, and myoclonic and partial seizures. On his second day, his EEG showed a suppression-burst pattern and we diagnosed EME. He was given multi-conventional AEDs, but the seizures continued, and at four months he began having frequent tonic spasms. ACTH therapy reduced seizure frequency for 3-4 months, but the effect was temporary, and at age 10 months, seizure frequently increased, and the boy developed severe retardation and a bad temper. LTG was initiated at this time and seizure frequency decreased and EEG findings improved when he was given LTG in addition to other AEDs. His bad temper disappeared and his appetite increased.
Conclusions: LTG, by providing an efficient and tolerated option for EME treatment, demonstrates a high efficacy against seizures and contributes significantly to the EME patient's quality of life.

G6-01

THE EFFICACY OF TOPIRAMATE IN TREATMENT OF INFANTILE SPASMS

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Objective: West syndrome and Aicardi syndrome are the most refractory epileptic syndromes in infancy, and many researchers have made great effort to find optimal treatment modalities for these syndromes. We investigated the topiramate (TPM) efficacy in complex treatment of infantile spasms in children with West and Aicardi syndromes.
Methods: 29 children in the age of from 1st month till 1 year with the diagnosis of West and Aicardi syndrome with infantile spasms have been surveyed. TPM was added to traditional treatment by AED and was started at a dose of 3-5 mg/kg/day (12.5 mg/day) with the minimum target dose of 50 mg/day and the maximum dose was 12 mg/kg/day.
Results: The etiology of these syndromes in 35 % of patients was cryptogenic, in 53% symptomatic, and in 12% idiopathic. The 30% of patients treated by TPM during the first month became seizure free, 40% had ≥ 50% reduction of seizures, and in 20% there weren't considerable changes and 10% had worsening of their spasms. The typical pattern on EEG as hypsarrhythmia normalized in 21% of cases, improved in 13%, persistent hypsarrhythmia in 34% and modified hypsarrhythmia was determined in 32% of patients.
Conclusion: Topiramate has a good effect on the clinical features of West and Aicardi syndromes, but clinical features do not correlate with EEG finding.

G6-02

EFFECT OF ANTI EPILEPTIC THERAPY ON SERUM HOMOCYSTEINE IN CHILDREN

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Background: Elevated plasma Homocysteine concentration is associated with increased risk for vaso-occlusive disease like cerebrovascular stroke, coronary artery disease, and also the risk of resistance to anti-epileptics and refractory epilepsy. Hyperhomocysteinemia has been frequently associated with the administration of Anti epileptic drugs (AED). This study aims at evaluating the effect of anti-epileptic therapy on serum Homocysteine levels in children.
Methods: 53 children (Males-32,Females-21) presenting to the Pediatric Outpatient and Inpatient department of Holy Family Hospital, New Delhi with Seizures in age group of 6 months-14 years were included in the study. Serum Homocysteine (Hcy) levels of Children already on AEDs for > 6 months (Group A) were compared with Children before Initiation of Anti epileptic drugs (Group B). These children were followed up after 6 months of Anti epileptic therapy and Serum Homocysteine was compared (Group C).
Results: Average Hcy levels in subjects who had already received >6 months of antiepileptic drug therapy were 12.58±2.68 µmol/l, compared to 8.83±2.82 µmol/l, at recruitment (*p=0.001*). Significant increased levels were also observed in children followed up after 6 months of AED - 10.27 ± 3.06 (µmol/l) compared to 8.63 ± 2.90(µmol/l) at initiation of AED. 9 children who received >1 AED had significantly higher levels-14.15 ± 2.56 (µmol/l) compared to children on monotherapy-10.22 ± 3.06 (µmol/l). Carbamazepine therapy for 6 months caused significant increase in Hcy 10.78 ± 2.82 (µmol/l) compared to baseline of 9.30 ± 2.70 (µmol/l) (*p=0.016*).
Conclusions: AEDs in children, especially those receiving multidrug or long duration treatment, cause Hyperhomocysteinemia.

G6-03

VAGUS NERVE STIMULATION IN PEDIATRIC REFRACTORY EPILEPSY MANAGEMENT (EXPERIENCE OF A METROPOLITAN TEACHING HOSPITAL IN TAIWAN)

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Purpose: Vagus Nerve Stimulation (VNS) is approved by FDA in 1997 for adjunctive therapy for intractable epilepsy. In Taiwan, the first Model102 VNS was implanted since August, 2007 under informed consent and IRB certification. To investigate clinical outcome of VNS implantation effect for Pediatric patient, we collected clinical data before & after VNS insertion for refractory epileptic children.
Method: There are 18 refractory epilepsy children implanted with vagus nerve stimulation therapy were enrolled in this research. 17 of these patients were implanted with vagus nerve stimulation more than 12 months. The mean of patients' age is 11.93 (range from 4 to 21 years old). There are 9 girls and 6 boys included in this research. 3 of 18 patients were explanted, and 2 of 3 were loss of follow-up. The mean implantation period was 31.85 months. The seizure pattern, seizure frequency and quality of life were recorded monthly in OPD and the psychological test was performed before and 1 year after the implantation. The etiology included neuronal migration disorder, encephalitis, and unknown origin. Epilepsy classifications included Lennox-Gastaut syndrome, congenital brain malformation, epilepsy related to encephalitis, generalized symptomatic epilepsy, localized symptomatic epilepsy, generalized cryptogenic epilepsy, and localized cryptogenic epilepsy (including temporal lobe epilepsy). The seizure types included atypical absence, simple partial seizure, complex partial seizure, myoclonic seizure, primary GTC, secondary GTC, and tonic seizure. The seizure reduction rate, EEG, brain MRI and intelligence test were done before or after performance of VNS for 1/2~1 year.
Results: The seizure free rate in these types is 33% in complex partial seizure, 50% in primary GTC and secondary GTC respectively. The overall seizure reduction rates were more than 50% in most patients, 23% seizure free among these seizure pattern, 43% has reduction rate more than 50%. 2 of these patients (11%) are seizure free after 6 months of implantation.
Conclusions: (1) Seizure reduction rates were more than 50% after implantation for half year in 43% of all patients; (2) The quality of life such as alertness, mood, and language function improved markedly and psychological test were both significantly improved.

G6-04

SIDE EFFECTS OF CHRONIC VAGUS NERVE STIMULATION FOR EPILEPSY IN CHILDREN

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Background: The aim of this study was to define better the incidence of surgical complications and untoward side effects of chronic vagus nerve stimulation (VNS) in a population of children with medically refractory epilepsy.
Methods: The authors retrospectively reviewed the cases of 50 connective patients (25 male and 25 female) 18 years of age or younger (mean age 8.8 years, range 11 month -18 years) who had undergone implantation of vagal stimulator between 2010 and 2011 with a minimum follow up of 1 year (mean 2.2 years). Of the 50 patients treated, seven (9.4%) had a complication ultimately resulting in removing the stimulator. The rate of deep infections necessitating device removal was 3.5% (three of 50 patients who had undergone 85 implantation and/or revision procedures). An additional three superficial infections occurred in patients in whom the stimulators were not removed: one was treated with superficial operative debridement and antibiotic agents and the other two with oral antibiotics only. Another four stimulators (5.4%) were removed because of the absence of clinical benefit and device intolerance.
Results: Two devices were revised because of lead fracture (2.7%). Among the cohort, 11 battery changes have been performed thus far, although none less than 33 months after initial implantation. Several patients experienced stimulation-induced symptoms (hoarseness, cough, drooling, outburst of laughter, shoulder abduction, dysphagia, or urinary retention) that did not require device removal. Ispilateral vocal code paralysis was identified in one patient. One patient died of aspiration pneumonia more than 30 days after device implantation.
Conclusions: Vagus nerve stimulation remains a viable option for improving seizure control in difficult to treat pediatric patients with epilepsy. Surgical complications such as hardware failure (2.7%) or deep infection (3.5%) occurred, resulting in device removal or revision. Occasional stimulation-induced symptoms such as hoarseness, dysphagia, or torticollis may be expected (5.4%).

G6-05

EVALUATION OF DIETARY THERAPIES (CLASSIC KETOGENIC AND MODIFIED ATKINS DIET) IN VIGABATRIN-RESISTANT INFANTILE SPASMS

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Objective: We report our experience with the use of dietary treatments; Ketogenic Diet (KD) and modified Atkins diet (MAD) in patients with vigabatrin-resistant infantile spasms.

Methods: Children aged 6 months to 3 years experiencing infantile spasms in clusters with electroencephalographic evidence of hypsarrhythmia despite treatment with vigabatrin, and who underwent dietary intervention with either KD (3:1 ketogenic ratio in children < 18 mo, and 4:1 in older children) or MAD (carbohydrates restricted to 10 grams/day, with no calorie/protein restriction) were enrolled. Outcomes included spasm freedom, EEG resolution of hypsarrhythmia and adverse effects.

Results: Nineteen children (14 boys, 5 girls) were enrolled; 13(68 %) had received MAD and 6 (31 %) received KD. Of these, 16 children had also failed hormonal therapy (ACTH/ prednisone). After 3 months, 8 children, (42%, 6 in MAD group, 2 in KD group) were spasm free with electroencephalographic resolution of hypsarrhythmia. The interval to spasm freedom ranged from 2 to 21 days. At six months follow up, 11 (58 %) children (8 in MAD, 2 in KD group) continued the dietary intervention. Of these, 8 (42%) were seizure free, and three had more than 90% reduction in spasms. Adverse effects included constipation (n=12, 63%), anorexia (n=5, 26%), and lethargy (n =4, 21%).

Conclusion: Both MAD and KD were found to be effective and well tolerated in children with vigabatrin-resistant infantile spasms. MAD offers the benefits of outpatient initiation, and is easier for parents to administer. MAD is also ideal in the resource-constraint settings encountered in developing countries.

G6-06

FAVOURABLE SEIZURE CONTROL WITH KETOGENIC DIET FOR TWO CHILDREN WITH MIGRATING PARTIAL SEIZURES OF INFANCY

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Objective: The effectiveness of ketogenic diet for seizure control in migrating partial epilepsy of infancy (MPSI) is rarely reported. We report on two children with favourable seizure control following ketogenic diet therapy.

Method: Retrospective review of two children that fulfilled criteria for diagnosis of MPSI, with negative metabolic workup, nonspecific MRI findings with favourable response to ketogenic diet is reported.

Results: Case 1: 2 year old girl with seizures in first day of life, initially as epileptic apnea progressing to alternating hemiclonic & asymmetric tonic seizures which were refractory to 7 antiepileptic drugs (AEDs). Her seizures became more frequent at 11 months with weekly clusters of 20 seizures/day and prominent autonomic features. Video-EEG monitoring showed near continuous electrographic seizures with interhemispheric migratory and shifting onset pattern with an abnormal background and multifocal epileptiform discharges. Following addition of ketogenic diet (4:1 ratio) at 17 months, seizures reduced to 2-3 seizures/month. At 2 years, she remained severely delayed and microcephalic.

Case 2: 14 month old girl with seizures from day 9 of life consisting of tonic seizures and eye blinking followed by generalized tonic-clonic seizures associated with development arrest. EEG showed similar findings as above. She failed to respond to multiple AEDs and continued to have numerous daily seizures. At 3.5 months, ketogenic diet (4:1 ratio) was introduced, resulting in 90% seizure reduction within 5 days of dietary therapy. At 14 months, she has daily seizures and AEDs reduced from three to one.

Conclusion: Ketogenic diet should be considered early in MPSI when seizures remain refractory to anticonvulsants

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