International Symposium on Epilepsy in Autism Spectrum Disorders and Related Conditions (ISEASD)

The 12th Annual Meeting of the Infantile Seizure Society

Program & Abstracts

May 9 -10, 2009
Kurume University Chikusui Hall
Kurume City, Fukuoka, Japan

Sponsored by Infantile Seizure Society (ISS), Japan
(Co-sponsored by Japan Foundation for Neuroscience and Mental Health)

Supported by
Japan Epilepsy Society
Japan Pediatric Society
Japanese Society of Child Neurology

Contents

Welcome Messages

Dear colleagues,

I am greatly pleased to be hosting the 12th Annual Meeting of the Infantile Seizure Society (ISS), focusing on epilepsy in autism spectrum disorders (ASDs). The International Symposium on Epilepsy in Autism Spectrum Disorders (ISEASD) will be held at Chikusui Hall in Kurume City, Fukuoka prefecture, Japan on May 9-10, 2009.

ASDs are a behaviorally defined set of developmental disorders that result from diverse biologic, genetic, and epigenetic factors. Autistic disorder, Asperger syndrome, Rett syndrome, and childhood disintegrative disorder are subsumed under the category of ASDs.

Epilepsy occurs frequently in children with autistic disorder; approximately one-third develop epilepsy by adulthood, and all seizure types occur. There is a bimodal distribution of age of onset, with peaks at age < 5 years and adolescence. However, many questions about epilepsy in both the basic and the clinical science fields still remain to be answered. The ISEASD aims to present a comprehensive update of the topic and discussions on epilepsy in ASDs, such issues as genetics, epidemiology, pathophysiology, neuro-imaging, and treatment.

I believe that this symposium will be inspiring and fruitful for all participants, as many distinguished scientists and physicians in this field have been invited from all over the world. It will certainly be a great opportunity for you to exchange recent views and engage in discussions of all aspects of epilepsy in ASDs and related conditions.

Kurume City is located in the southwest of Fukuoka prefecture on Kyushu, the southern island of Japan's four main islands, close to various Asian countries. Kurume is endowed with a rich cultural heritage and beautiful natural resources. All participants of ISEASD can easily enjoy a trip to Mt. Aso (world's largest volcano) and the famous Beppu hot spa resort. We hope you will stay in Kurume City, giving you plenty of opportunity to share memorable times with invited speakers, other participants, your friends and family.

We are looking forward to seeing you in Kurume, this spring.

All the best

Toyojiro Matsuishi, MD, PhD
President of ISEASD
Professor & Chairman
Department of Pediatrics and Child Health
Kurume University School of Medicine

Dear ISS members and colleagues

It is our great pleasure to announce that the 12th Annual Meeting of the Infantile Seizure Society (ISS) will be held in Kurume City, Japan, on May 9-10, 2009. "Epilepsy in Autism Spectrum Disorders" was chosen as the main theme of the Meeting, and as usual for our Society, the Meeting will be organized as an international symposium. Thus, the next Meeting will be called as ISEASD, abbreviated from the formal name of International Symposium on Epilepsy in Autism Spectrum Disorders and Related Conditions.

Problems of epilepsy seen in autism spectrum disorders (ASDs) can be approached in two ways. The one is to scrutinize the implication of comorbidity of epilepsy in autistic disorders. Whether co-existing epilepsy will influence or modify somehow by itself on clinical features and outcomes of ASDs proper ? Or, are there any differences between the group of ASD patients with epilepsy and those without, in terms of etiology, clinical characteristics and prognosis? Is it meaningful or not to control seizures successfully for improving autism ?

Another approach will be to analyse the issue from the viewpoint of epileptology. Globally specking, the ASDs patients do not present gross structural abnormalities of brain. The disturbances in ASDs are typically at the level of higher brain function such as cognition, behavior, and emotion. This background stands in sharp contrast with infants and young children who suffer from symptomatic epilepsy. Then, the problems will emerge whether epilepsy in ASDs has any specificities in comparison to conventional epileptic disorders in infants and young children. Are different epileptogenetic mechanisms operating in the two groups (ADSs vs common epilepsies)?

It has been known that babies with the West syndrome often evolve to autism later. The same is recognized in cases of tuberous sclerosis. How relate autism in later stage to epilepsies in earlier age in these conditions? On the other hand, it is also well known that chronic suffering from epilepsy will not rarely lead to cognitive decline and mood changes. The mechanism is unknown, but if we could better understand the interrelation between autism and epilepsy, it may give us a significant clue to better understand the mechanism of mental deterioration in chronic epilepsy, in general.

Thus, the main theme of ISEASD contains significant practical and theoretical implications, and more importantly, the issues have been scarcely explored by modern medicine with advanced technology in the past. The ISEASD is going to challenge boldly to this wild area for the first time in the world.

Everyone, who might have interests in these issues, will be welcomed. Please come from all corners of the world and join us at this ISEASD, Kurume, Japan, May 9-10, 2009. Spring is the best season in Japan, and you will be able to enjoy world-famous traditional entertainments to a full extent.

Looking forward to welcoming all of you to Kurume, Japan.

Yukio Fukuyama

Yukio Fukuyama, MD
Chairperson, Board of Councilors,
Infantile Seizure Society

Organization

ORGANIZING COMMITTEE

[A] GENERAL

Supreme Advisor Yukio FUKUYAMA (Tokyo, Japan)

Chairperson (President) Toyojiro MATSUISHI (Kurume, Japan)

Vice-chairperson Kenji SUGAI (Kodaira, Japan)
Yoshihiro TAKEUCHI (Otsu, Japan)

Advisors
Sunao KANEKO (Hirosaki, Japan)
Shunsuke OHTAHARA (Okayama, Japan)
Tatsuya TANAKA (Asahikawa, Japan)
Kazuyoshi WATANABE (Nagoya, Japan)

Committee Members
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Tateki FUJIWARA (Shizuoka, Japan)
Mitsumasa FUKUDA (Matsuyama, Japan)
Shin-ichirou HAMANO (Saitama, Japan)
Shinichi HIROSE (Fukuoka, Japan)
Yong-Seung HWANG (Seoul, Korea)
Kazuie IINUMA (Ishinomaki, Japan)
Akio IKEDA (Kyoto, Japan)
Yushi INOUE (Shizuoka, Japan)
Tatsuro IZUMI (Oita, Japan)
Akemi KAKIDA (Niigata, Japan)
Shigeki KAMEYAMA (Niigata, Japan)
Osamu KANAZAWA (Saitama, Japan)
Mitsuhiro KATO (Yamagata, Japan)
Kensuke KAWAI (Tokyo, Japan)
Ryutaro KIRA (Fukuoka, Japan)
Jun KOHYAMA (Tokyo, Japan)
Yoshiya MURASHIMA (Tokyo, Japan)
Toshisaburo NAGAI (Osaka, Japan)
Shin-ichi NIIJIMA (Tokyo, Japan)
Hirokazu OGUNI (Tokyo, Japan)
Akihisa OKUMURA (Tokyo, Japan)
Makiko OSAWA (Tokyo, Japan)
Yoko OHTSUKA (Okayama, Japan)
Taisuke OTSUKI (Kodaira, Japan)
Shinji SAITO (Sapporo, Japan)
Yasuhiro SUZUKI (Izumi, Japan)
Satoshi TAKADA (Kobe, Japan)
Takao TAKAHASHI (Tokyo, Japan)
Kazuhiro YAMAKAWA (Wako, Japan)
Tsunekazu YAMANO (Osaka, Japan)
Hideo YAMANOUCHI (Tochigi, Japan)
Hitoshi YAMAMOTO (Kawasaki, Japan)

[B] SCIENTIFIC PROGRAM COMMITTEE

Chairpersons Toyojiro MATSUISHI (Kurume, Japan), Yushiro YAMASHITA (Kurume, Japan)

Committee Members
Yukio FUKUYAMA (Tokyo, Japan)
Hitoshi HARA (Yokohama, Japan)
Shinichi HIROSE (Fukuoka, Japan)
Yushi INOUE (Shizuoka, Japan)
Tatsuro IZUMI (Oita, Japan)
Osamu KANAZAWA (Saitama, Japan)
Ryutaro KIRA (Fukuoka, Japan)
Shin-ichi NIIJIMA (Tokyo, Japan)
Yoshiko NOMURA (Tokyo, Japan)
Kenji SUGAI (Kodaira, Japan)
Hitoshi YAMAMOTO (Kawasaki, Japan)

[C] FUND COMMITTEE AND TREASURER

Chairperson & Treasurer	Toyojiro MATSUISHI (Kurume, Japan),	Shinichi NIIJIMA (Tokyo, Japan)
Committee Members	Yukio FUKUYAMA (Tokyo, Japan)	Satoshi TAKADA (Kobe, Japan)
	Takao TAKAHASHI (Tokyo, Japan)	Toshisaburo NAGAI (Osaka, Japan)
	Hitoshi YAMAMOTO (Kawasaki, Japan)	Kenji SUGAI (Kodaira, Japan)
	Tsunekazu YAMANO (Osaka, Japan)	Yasuhiro SUZUKI (Izumi, Japan)
	Hideo YAMANOUCHI (Tochigi, Japan)	

[D] SPONSORING ORGANIZATIONS

Sponsored: Infantile Seizure Society

Co-sponsored: Japan Foundation for Neuroscience and Mental Health

Supported: Japan Epilepsy Society
Japan Pediatric Society
Japanese Society of Child Neurology

Head Office

Yushiro YAMASHITA, MD, PhD
Department of Pediatrics & Child Health
Kurume University School of Medicine ,
67 Asahi-machi, Kurume City, Fukuoka, 830-0011, Japan
Tel: +81-942-31-7565 / Fax: +81-942-38-1792
Email: iss2009@med.kurume-u.ac.jp
URL: http://www.med.kurume-u.ac.jp/med/ped/iss2009/index.html

Registration

Desk for Registration and General Information, located at the 1st floor of Kurume University Chikusui Hall, will be opened for the following periods:

May 9 th (Saturday) 08:00-19:00
May 10 th (Sunday) 07:30-19:00

Pre-Registration

Those who completed the registration before January 31, 2009, should go to the Pre-registrant Reception Desk, present his/her Registration Confirmation Sheet to the receptionist and then receive his/her ready-prepared bag.

On-site Registration

Registration Form should be presented to the reception desk, after filling out its upper part on ly, together with the fee payment in Japanese yen (cash) of appropriate amount.
The fee rates are defined variably as shown below according to the participant's category.

	2 days participation May 9 & 10
Symposium	
Japanese colleagues	
ISS* member	21,000 JPY
Non-ISS member	24,000 JPY
Non-Japanese colleagues	
AOCNA** members	18,000 JPY
Non- AOCNA members	24,000 JPY
Junior physicians***	18,000 JPY
Grand Social Party (May 9)	
Japanese colleagues	5,000 JPY
Non-Japanese colleagues	Free

*= Infantile Seizure Society
**= Asian & Oceanian Child Neurology Association
***= Young physicians graduated from medical school after January 2003.
Students of post-graduate course are also applicable to this category.
Copy of official document such as a student's identification or a certificate may be required.

To ISS members:

The members of the Infantile Seizures Society (ISS) are requested to pay his/her annual fees (\3,000) at the registration desk. Anyone who wish to become a member of ISS is requested to fill out the membership application form and pay for the 2009 annual fee (\3,000).

To AOCNA members:

The members of the Asian & Oceanian Child Neurology Association (AOCNA) are requested to contact the AOCNA reception desk, and to confirm his/her correspondence address in the membership roster and status of his/her dues payment. To become a member of AOCNA, please fill out the application form and pay the fee for the coming 4years (2009-2012)(40.00US$) or the lifetime membership fee (100.00US$) in cash.

Don't miss the Grand Social Party

Please get together everybody in the Grand Social Party at the Suikoyen Hotel. All non-Japanese colleagues are free of charge. Japanese colleagues are requested to register. Please refer to the "General Information (p.7)".

General Information

Date

May 9th (Sat) - May 10th (Sun), 2009

Venue

Kurume University, Chikusui Hall
67 Asahi-machi, Kurume City, Fukuoka, 830-0011 Japan
Phone: +81-942-31-7565

0fficial Language

English. Simultaneous translation (English to Japanese) will be available on Lunch Time and Evening Seminars. (May 9, 11:30-12:20, 17:40-18:30; May 10, 12:20-13:10)

Social Function

1) Presidential Welcome Reception
Date & Time : Friday, May 8th, 19:00-21:30
Place : Suikoyen Hotel (see page 12)
Attire : Casual
Attendance : Limited to the invitees

2) Grand Social Party
Date & Time : Saturday, May 9th, 19:15-21:45
Place : Suikoyen Hotel (see page 12)
Attire : Casual
Attendance : Free (Non Japanese colleagues)/5,000 JPY per person (Japanese colleagues)

3) Free Farewell Party
Date & Time : Sunday, May 10th, 19:00-21:30
Place : Koyanagi (Yakitori Restaurant; see page 12)
Attire : Casual
Attendance : Free

Official Certificate for Attendance and CME Points

An official certificate for attendance at the ISEASD will be delivered to all participants. To Japanese colleagues, a certificate for authorized CME units will be rewarded by two societies as follows:

Society	Attendance	Authorship	
		Presenter	Co-author
Japan Pediatric Society	5 u	0 u	0 u
Japanese Society of Child Neurology	2 u	2 u	0 u

u=unit

Lunch (Days 1 and 2)

Lunch will be served for participants during the time of Lunch Time Seminars.

Coffee

Coffee will be served at the 1st floor.

Internet

We will provide a PC for Internet access at the 1st floor. Wireless Internet is not available.

ISS Council Meeting

ISS council meeting will be held at the "Conference Room B", (1st floor) from 17:30 to 18:40, May 10th after the scientific program is over. The ISS councilors are requested to attend this meeting.

To Make Your Stay Comfortable

Climate

May is the most comfortable season of the year in Kurume with high temperature being around 22 degree Celsius (72 degree Fahrenheit) and low around 14 degree Celsius (58 degree Fahrenheit).

Currency Exchange

We strongly recommend purchasing Japanese currency, yen at Kansai International Airport or Tokyo Narita International Airport. This is because you need cash in yen to purchase tickets for ground transportation from the airport to wherever your first destination in Kurume may be.

Voltage and Frequency of Electricity

The voltage is 100 V throughout the country. Frequency is 60 Hz in Kurume area and 50 Hz in the Eastern Japan such as Tokyo.

Shopping

Five percent sales tax is applicable to personal purchases.

Gratuity / Tip

Gratuity or "service fee" is included in a bill at most of the decent restaurants. Otherwise they usually do not accept any tips.

Sightseeing Tours

If you wish to have a sightseeing tour during your stay in Japan, we recommend making a reservation in advance through travel agency such as Nishitetsu Travel Co. **If you have any questions, please contact to ISEASD secretariat.**

Secretariat

Inquiries on ISEASD 2009

ISEASD Secretariat
Yushiro YAMASHITA, MD, PhD
Department of Pediatrics & Child Health, Kurume University School of Medicine
67 Asahi-machi, Kurume City, Fukuoka, 830-0011, Japan
Phone: +81-942-31-7565 / Fax: +81-942-38-1792
Email: iss2008@med.kurume-u.ac.jp

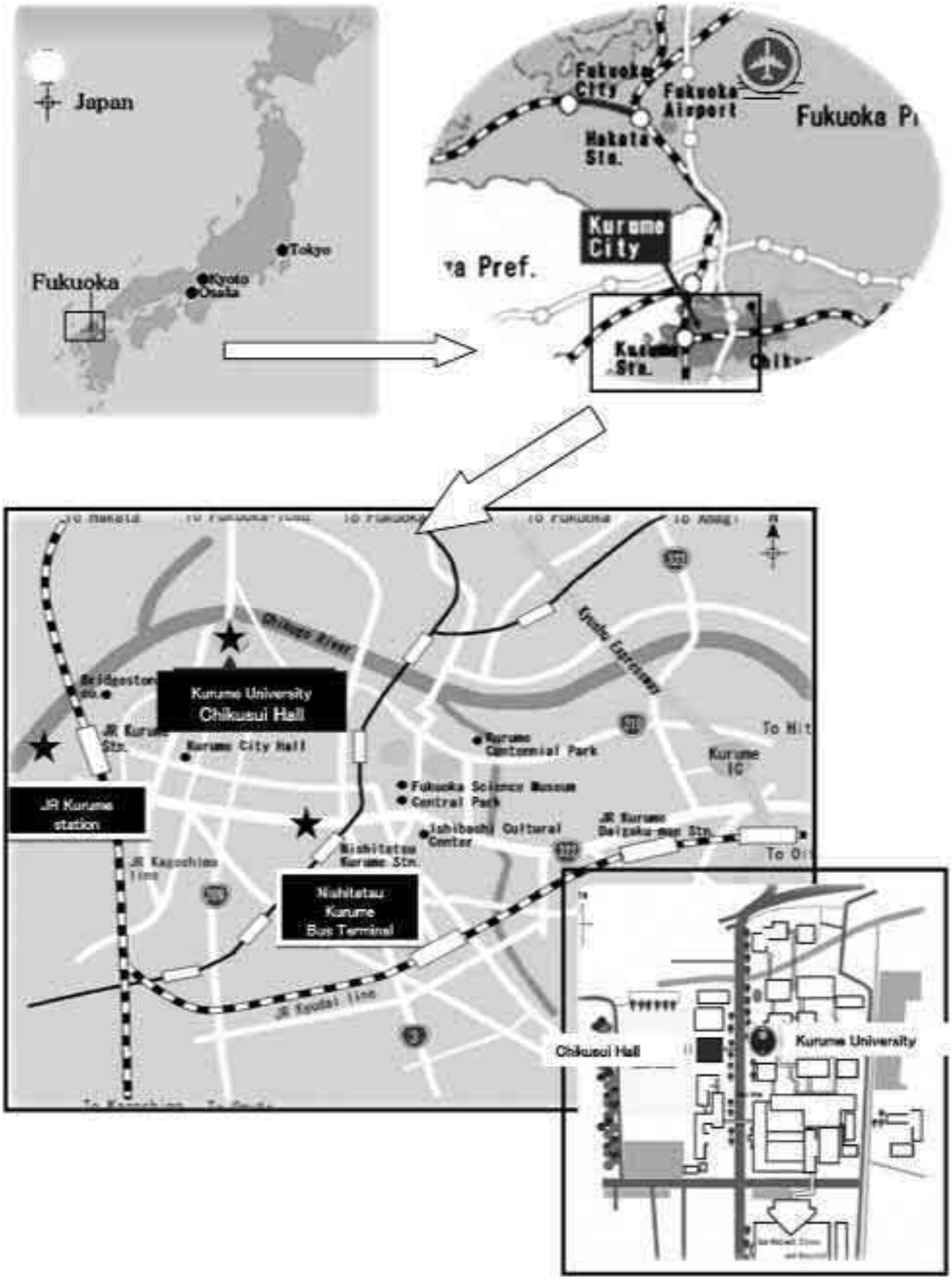
Inquiries on Infantile Seizure Society in general:

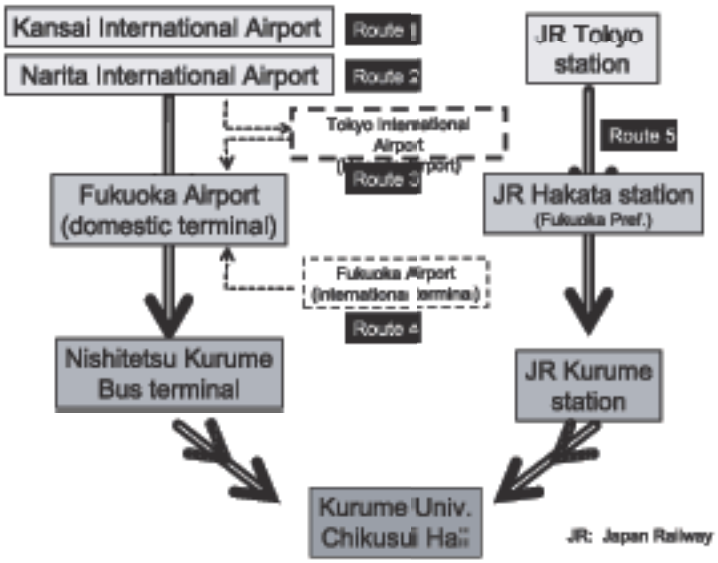
ISS Secretariat
Yukio FUKUYAMA, MD, PhD
c/o Child Neurology Institute
6-12-17-201 Minami-Shinagawa, Shinagawa-ku, Tokyo
140-0004, Japan
Phone: +81-3-5781-7680 / Fax: +81-3-3740-0874
Email: yfukuyam@sc4.so-net.ne.jp

ISS/ISEASD Websites

ISS website: http://www.iss-jpn.info/
ISEASD website: http://www.med.kurume-u.ac.jp/med/ped/iss2009/index.html

Venue Access





I. From Kansai Internatinal Airport

Route 1

Kansai International Airport → Fukuoka Airport (Domestic Terminal)

Departure	Destination	Movement method	Travel Time
Kansai International Airport	Fukuoka Airport (Domestic Terminal)	AirPlane	105min.

Fukuoka Airport (Domestic Terminal) → Nishitetsu Kurume Bus Terminal

Departure	Destination	Bus Stop No.	Travel Time	Fare
Fukuoka Airport (Domestic Terminal)	Nishitetsu Kurume Bus Terminal	Terminal 2/No.3 Terminal 3/No.2	45min.	1,200yen

II . From Narita International Airport

Route 2

Narita International Airport → Fukuoka Airport (Domestic Terminal)

Departure	Destination	Movement method	Travel Time
Narita Airport	Fukuoka Airport (Domestic Terminal)	AirPlane	120min.

Fukuoka Airport (Domestic Terminal) → Nishitetsu Kurume Bus Terminal

same as route 1

Route 3

Narita International Airport → Tokyo International Airport (Haneda Airport)

●Rail and Limousine bus provide convenient connections to and from Haneda Airport.

Departure	Destination	Travel Time		Fare
Narita Airport	Haneda Airport	Rail	110min.	1,560yen
		Limousine bus	75min.	3,000yen

Tokyo International Airport (Haneda Airport) → Fukuoka Airport (Domestic Terminal)

Departure	Destination	Movement method	Travel Time
Haneda Airport	Fukuoka Airport (Domestic Terminal)	AirPlane	110min.

Fukuoka Airport (Domestic Terminal) → Nishitetsu Kurume Bus Terminal

same as route 1

III.From Fukuoka Airport (International Terminal)

Route 4

Fukuoka Airport (International Terminal) → Fukuoka Airport (Domestic Terminal)

Departure	Destination	Bus Stop	Travel Time	Fare
Fukuoka Airport (International Terminal)	Fukuoka Airport (Domestic Terminal)	Passenger Terminal First Floor	10min.	Free(Shuttle Bus)

Fukuoka Airport (Domestic Terminal) → Nishitetsu Kurume Bus Terminal
same as route 1

IV.From J R, Tokyo Station

Route 5

JR Tokyo Station → JR Hakata Station

Departure	Destination	Platform	Travel Time	Fare
JR Tokyo Station	JR Hakata Station	No.14 ~ 19	about 300min.	about 22,320yen

JR Hakata Station → JR Kurume Station

Departure	Destination	Route	Travel Time	Fare
JR Hakata Station	JR Kurume	Kagoshima-Honsen Rapid train	40min.	720yen

Ground Transportation

By Taxi

Taking taxi is a convenient way to get to the venue from Nishitetsu Kurume Bus terminal and JR Kurume station. The ride normally takes 10 min (costs 1000 yen) from Nishutetsu Kurume Bus terminal and 5 min (costs 650 yen) from JR Kurume Station.

By Bus

Taking bus is other way to get to the venue.

Nishirtetsu Kurume Bus Terminal → Kurume Univ.Hospital

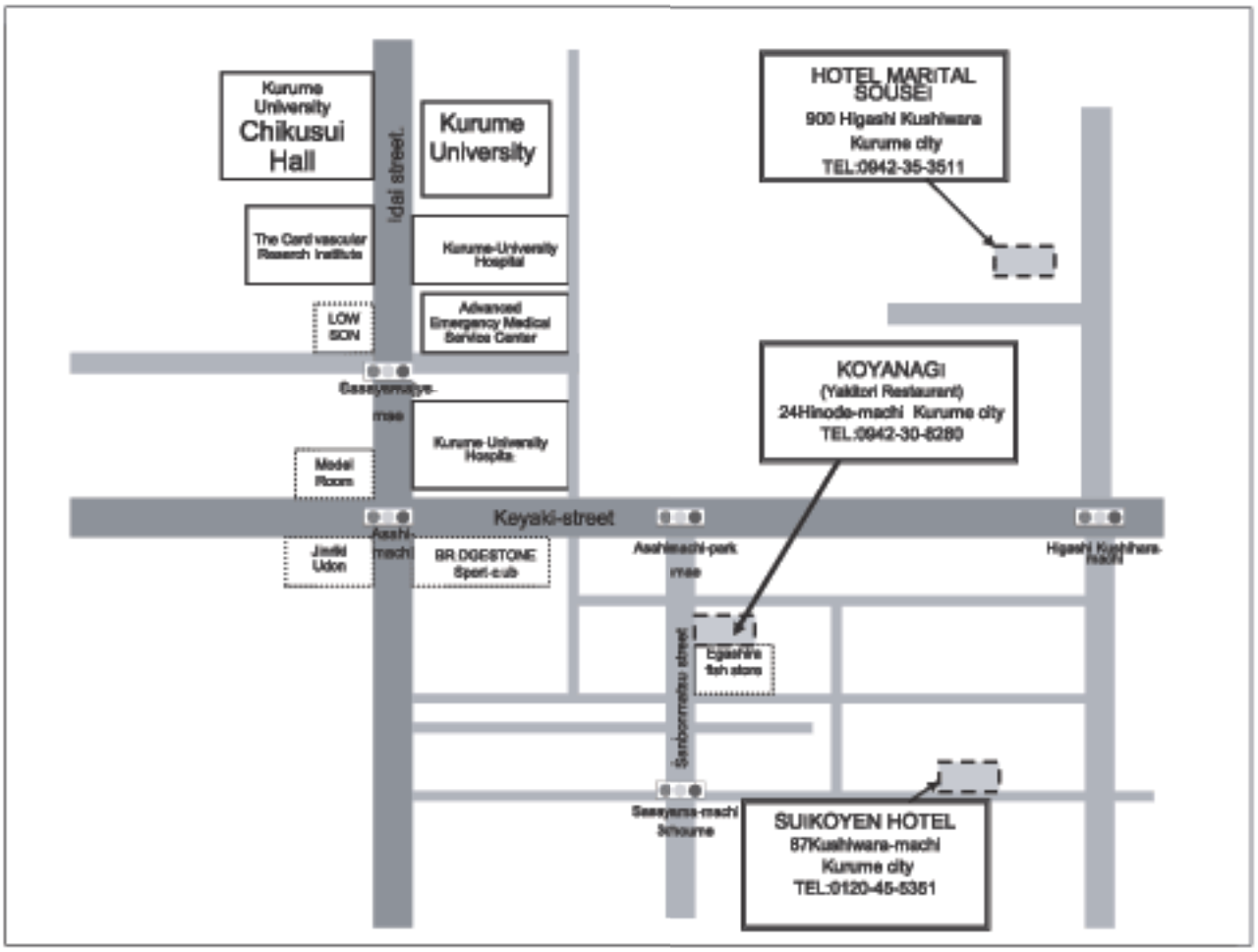
Departure	Destination	Bus No.	Travel Time	Fare
Nishitetsu Kurume Bus Terminal	Kurume Univ. Hospital Mae	No.6、No.8、No.24、No.52	15min.	220yen

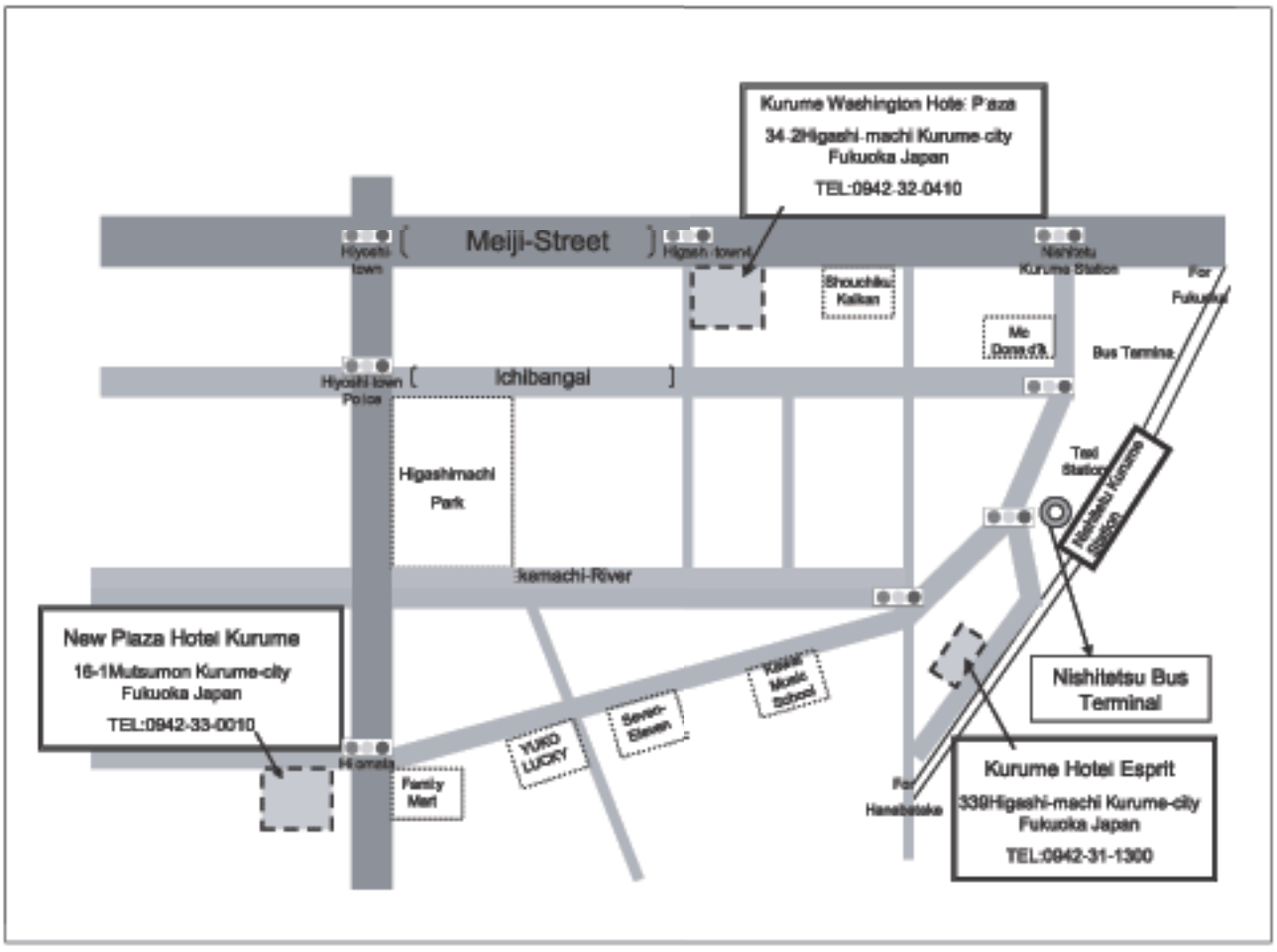
JR Kurume Station → Kurume Univ. Hospital

Departure	Destination	Bus No.	Travel Time	Fare
JR Kurume Station	Kurume Univ. Hospital Mae	No.8	8min.	160yen

Usefuel Website

Fukuoka Airport	http://www.fuk-ab.co.jp/english/frame_index.html
JR Kyushu	http://www.jrkyushu.co.jp/english/index.html
Nishitetsu	http://jik.nnr.co.jp/cgi-bin/Tschedule/menu.exe?pwd=gb/menu.pwd&mod=F&menu=F
Kurume Univ. Hospital	http://www.kurume-u.ac.jp/english/index.html
Narita International Airport	http://www.narita-airport.jp/en/index.html
Kansai International Airport	http://www.kansai-airport.or.jp/en/index.asp
Rail(Airport Limited Express)	http://www.narita-airport.jp/en/access/train/index.html
Limousine Bus	http://www.limousinebus.co.jp/en/ (Narita International Airport→Haneda Airport)


Kurume University Chikusui Hall
Kurume University
Idai street
The Cardiovascular Research Institute
Kurume University Hospital
Advanced Emergency Medical Service Center
LOW SION
Kurume University Hospital
Model Room
Keyaki-street
Asahi-machi
Jindai Udon
BRIDGESTONE Sport-club
Asahimachi-park mae
Sanbonmatsu street
Egashira fish store
Sasayama-machi 3chome
HOTEL MARITAL SOUSEI
900 Higashi Kushiwara
Kurume city
TEL:0942-35-3511
KOYANAGI
(Yakitori Restaurant)
24Hinode-machi Kurume city
TEL:0942-30-6280
SUIKOYEN HOTEL
87Kushiwara-machi
Kurume city
TEL:0120-45-5351
Higashi Kushiwara-machi


Kurume Washington Hotel Plaza
34-2Higashi-machi Kurume-city
Fukuoka Japan
TEL:0942-32-0410
Meiji-Street
Higashi-town
Nishitetsu Kurume Station
For Fukuoka
Shouchiku Kaikan
Mc Donald's
Bus Termina
Hiyoshi-town
Hiyoshi town Police
Ichibangai
Higashimachi Park
Taxi Station
Nishitetsu Kurume Station
kamachi-River
New Plaza Hotel Kurume
16-1Mutsumon Kurume-city
Fukuoka Japan
TEL:0942-33-0010
Hiyoshi
Family Mart
YUKO LUCKY
Seven-Eleven
Kawai Music School
Nishitetsu Bus Terminal
For Hanabatake
Kurume Hotel Esprit
339Higashi-machi Kurume-city
Fukuoka Japan
TEL:0942-31-1300

Instructions for Oral Presentations

1. All speakers are requested to strictly observe the allotted presentation time. Please note the time allocated for each presentation is as follows; 40 minutes for presentation followed by 10 minutes of discussion for most of the invited speakers and 25 minutes for presentation followed by 5 minutes of discussion for some of the invited speakers. The green light on the desk will be turned on one minute before the end and the red light will inform the time over.
2. Only one single LCD projection will be available.
3. Every speaker is requested to finish up an arrangement necessary for data projection two hours before the respective presentation at the latest, by contacting the staff of the PC Preview Center, located at the 2F.
4. The data has to be presented with USB flash memory or CD-R. Or individual laptop computer are permitted for presentations.
5. Facilities to preview are available at the PC Preview Center.
6. All presentations should be prepared by "PowerPoint, after ver. 2002" on Windows system. If your data was prepared by Mac system, the data may deform after its transfer to the Windows system. In this case, please check and correct this possible deformation at the Preview Center. If your PowerPoint is before ver. 2002, please inform it to the ISEASD Secretariat by mail or to the staff of Preview Center at the venue, as early as possible.
7. Each presenter is requested to manipulate the computer placed at the platform during the presentation. If you need help for manipulation, please inform to the ISEASD Secretariat at the Preview Center. Your own personal computer is also available to use for presentation.
8. Video tape presentation is not available. If you need to use video records, please transfer them to the computer in a digital form. In this case, it is advised to bring your own personal computer, since the software does not fit often for your record. If you bring your own computer, Mac system is also available.
9. Please remind that the details of the oral presentations will be delivered over a network by audio/video streaming, thereby enabling closed-users to see and hear the audio and video files. In this data streaming, the majority of PC slides will be shown with synchronized oral presentations. It will contain almost all lectures and discussions presented at the ISEASD meeting. In this regard, if you have any problems, or any PC slides that you want to delete from this data streaming, please contact the staff of PC Preview Center.

Instructions for Discussion

1. Active discussion from the floor is encouraged as far as the time is available.
2. All aspects of discussion session shall be ordered by due consideration of chairpersons.
3. Anyone who wishes to raise a question/discussion is urged to line up before the microphone stands to save time, and wait for an order of chairpersons. To begin your discussion, please identify yourself first.

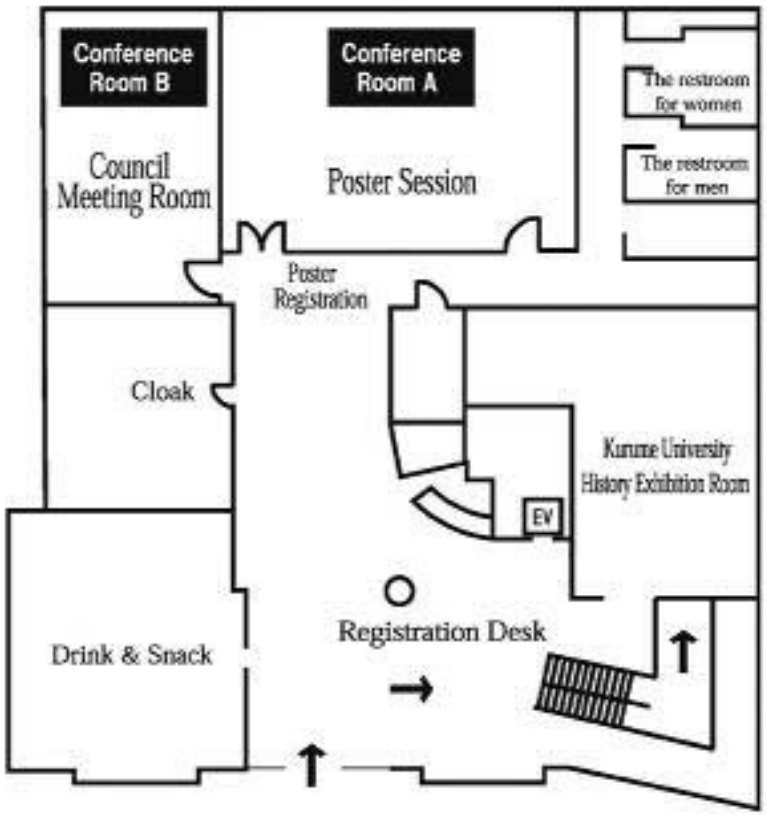
Instructions for Poster Presentations

1. Place: "Conference Room A" on the 1st floor.
2. Registration: The presenter is requested to register at the "Poster Reception Desk" on the 1st floor.
3. Pushpins will be provided.
4. All presentations should be posted on the pre-assigned panel by 9:00 am, May 9th.
5. A poster panel has a surface of 90 cm wide and 180 cm high. Top space will be used to place the poster number in a size of 20 cm x 20 cm, pre-fixed by the Secretariat. The title, author's names, and affiliations should be prepared by the presenters in the top space of 70 cm wide and 20 cm high. The other main space of 90 cm wide and 160 cm high is available for the body of poster presentation.
6. Poster round. Presenters are requested to be present at the site of respective posters for discussion during the time of Coffee Break & Poster Presentation (1) or (2). Each presenter is also requested to make a brief oral presentation for 3 minutes, and another 3 minutes will be allowed for questions from the participants. The rooms, the coordinators, and the detailed time schedule for each presentation are as follows.

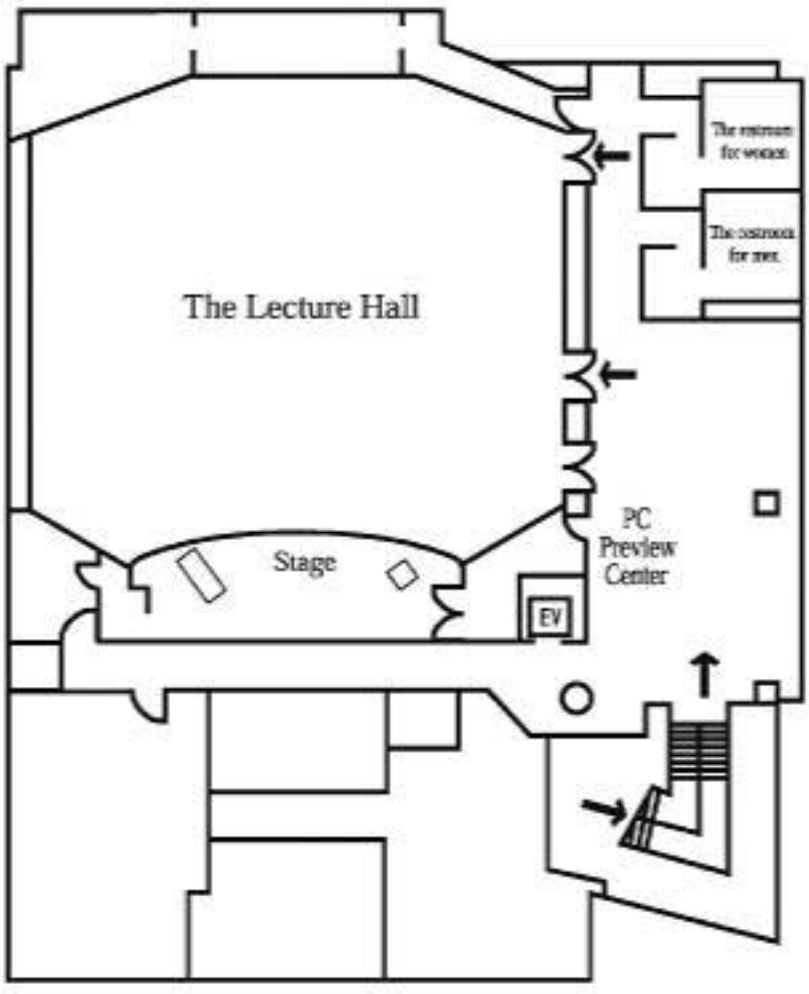
Coffee Break & Poster Presentation (1) May 9 (Saturday), 15:35-16:35			Coffee Break & Poster Presentation (2) May 10 (Sunday), 11:15-12:15		
Room	Conference Room A		Room	Conference Room A	
Coordinators	Ohtsuka Y Sugai K	Niijima S Yamanouchi H	Coordinators	Izumi T Nagai T	Hayashi M Kira R
15:35-15:41	Poster No.1	Poster No.11	11:15-11:21	Poster No.20	Poster No.30
15:41-15:47	2	12	11:21-11:27	21	31
15:47-15:53	3	13	11:27-11:33	22	32
15:53-15:59	4	14	11:33-11:39	23	33
15:59-16:05	5	15	11:39-11:45	24	34
16:05-16:11	6	16	11:45-11:51	25	35
16:11-16:17	7	17	11:51-11:57	26	
16:17-16:23	8	18	11:57-12:03	27	
16:23-16:29	9	19	12:03-12:09	28	
16:29-16:35	10		12:09-12:15	29	

Floor Plan

Chikusui Hall (1F)



Chikusui Hall (2F)



Overview of Daily Program

Friday, May 8

19:00-21:30
Welcome Reception (Invitees only)

Saturday, May 9

8:50-9:00
Registration
Opening Addresses

9:00-11:20
Epidemiology
- Matsuo M
- Kurokawa T
- Saemundsen E
- Wong V

11:30-12:20
Lunch Time Seminar
- Tuchman R

12:20-13:10
Coffee Break and Poster Round

13:10-15:30
Electrophysioligy
- Kawasaki Y
- Parmeggiani A
- Yasuhara A
- Sasaki M

15:30-16:40
Coffee Break and Poster Presentation (1)

16:40-17:40
Pathophysiology
- Nomura Y
- Toichi M

17:40-18:30
Evening Seminar
- Brooks-Kayal A

19:15-20:45
Grand Social Party

Sunday, May 10

8:30-9:50
Genetics
- Dibbens L
- Takumi T

9:50-11:10
Related Conditions
- Deonna T
- Chae JH

11:10-12:20
Coffee Break and Poster Presentation (2)

12:20-13:10
Lunch Time Seminar
- Zappella M

13:10-15:20
Treatment and Follow-up
- Deonna T
- Hara H
- Tuchman R

15:20-15:40
Coffee Break

15:40-17:00
Discussion Summary, Synthesis and Consensus Development

17:00-17:10
Closing Address

17:30-18:40
ISS Council Meeting

19:00-21:30
Free Farewell Party

PROGRAM

PROGRAM - ORAL PRESENTATIONS

Day 1, MAY 9 (SATURDAY)

Opening Addresses 8:50-9:00

Inanaga K (Professor Emeritus, Kurume University)
Matsuishi T (President of ISEASD)

Epidemiology 9:00-11:20

Chairpersons: Chang KP (Taipei, Taiwan)
Saemundsen E (Kopavogur, Iceland)

O 01 9:00-9:20

FREQUENT ASSOCIATION OF AUTISM SPECTRUM DISORDERS IN CHILDHOOD ONSET COMPLEX PARTIAL SEIZURE

Matsuo M[1)], Maeda T[1)], Sasaki K[2)], Ishii K[3)], (Saga[1) 2) 3)], Japan)

O 02 9:20-9:40

CLINICAL FEATURES OF EPILEPSY WITH PERVASIVE DEVELOPMENTAL DISORDER

Kurokawa T[1)], Yokomizo Y[1)], Lee S[2)], Kusuda T[2)] (Onojou[1)] and Fukuoka[2)] , Japan)

O 03 9:40-10:30

EPIDEMIOLOGY OF ASD AND EPILEPSY

Saemundsen E (Kopavogur, Iceland)

O 04 10:30-11:20

AUTISM SPECTRUM DISORDER (ASD) AND EPILEPSY- HONG KONG PERSPECTIVE

Wong V (Hong Kong)

Lunch Time Seminar (1) 11:30-12:20

Chairperson: Hirose S (Fukuoka, Japan)
Sponsored by JANSSEN PHARMACEUTICAL K.K., Japan

O 05 11:30-12:20

AUTISM AND EPILEPSY: HISTORICAL PERSPECTIVE

Tuchman R (Miami, USA)

Coffee Break and Poster Round 12:20-13:10

Electrophysioligy 13:10-15:30

Chairpersons: Deonna T (Lausanne, Switzerland)
Inoue Y (Shizuoka, Japan)

O 06 13:10-13:40

PATHOPHYSIOLOGY OF PERVASIVE DEVELOPMENTAL DISORDERS AS REVEALED BY EEG, MEG, WITH PARTICULAR REFERENCE OF EPILEPSY

Kawasaki Y[1)], Shinomia M[1)], Niwa S[1)] (Tokyo[1)] and Fukushima[2)], Japan)

O 07 13:40-14:30

AUTISM SPECTRUM DISORDERS (ASD) AND EEG PAROXYSMAL ABNORMALITIES: HOW ARE THEY LINKED?

Parmeggiani A, Barcia G, Posar A, Raimondi E, Santucci M, Scaduto MC (Bologna, Italy)

O 08 14:30-15:00

CORRELATION OF EEG ABNORMALITIES IN ASD WITH SYMPTOMS AND TREATMENT

Yasuhara A (Osaka, Japan)

O 09 15:00-15:30

BRAIN PERFUSION SPECT AND EEG FINDINGS IN AUTISM WITH EPILEPSY

Sasaki M (Kodaira, Japan)

Coffee Break and Poster Presentation (1) 15:30-16:40

Coordinators: Posters 01-10 Ohtsuka Y (Okayama, Japan), Sugaki K (Kodaira, Japan)
Posters 11-19 Niijima S (Tokyo, Japan), Yamanouchi H (Tochigi, Japan)

Pathophysiology 16:40-17:40

Chairpersons: Brooks-Kayal A (Denver, USA)
Kanazawa O (Saitama, Japan)

O 10 16:40-17:10

EPILEPSY IN AUTISM; A PATHOPHYSIOLOGICAL CONSIDERATION

Nomura Y (Tokyo, Japan)

O 11 17:10-17:40

CLINICAL AND COGNITIVE FEATURES AND POSSIBLE NEURAL BASES OF PERVASIVE DEVELOPMENTAL DISORDER

Toichi M (Kyoto, Japan)

Evening Seminar 17:40-18:30

Chairperson: Takeuchi Y (Otsu, Japan)
Sponsored by Kyowa Hakko Kirin Co., Ltd, Japan

O 12 17:40-18:30

EPILEPSY AND ASD: ARE THERE COMMON DEVELOPMENTAL MECHANISMS?

Brooks-Kayal A (Denver, USA)

Day 2, MAY 10 (SUNDAY)

Genetics

8:30-9:50

Chairpersons: Takahashi T (Tokyo, Japan)
Dibbens L (North Adelaide, Australia)

O 13 8:30-9:20

X-LINKED FEMALE-LIMITED EPILEPSY AND COGNITIVE IMPAIRMENT CAUSED BY PROTOCADHERIN 19 MUTATIONS

Dibbens LM[1)], Tarpey PS[2)], Hynes K[1)], Bayly MA[1)], Scheffer IE[3)], McKee S[4)], Berkovic SF[3)], Stratton MR[2)], Mulley JC[1)], Gecz J[1)] (North Adelaide[1)] and Victoria[3)], Australia; Hinxton[2)] and Northern Ireland[4)], UK)

O 14 9:20-9:50

A CHROMOSOME- ENGINEERED MOUSE FOR HUMAN 15q11-13 DUPLICATION AS AN AUTISM MODEL

Takumi T (Hiroshima, Japan)

Related Conditions

9:50-11:10

Chairpersons: Kaga M (Kodaira, Japan)
Wong V (Hong Kong)

O 15 9:50-10:40

OVERLAP-RELATIONSHIP BETWEEN EARLY ACQUIRED EPILEPTIC APHASIA (LANDAU- KLEFFNER SYNDROME, LKS) AND PERVASIVE DEVELOPMENTAL DISORDER (PDD)

Deonna T (Lausanne, Switzerland)

O 16 10:40-11:10

RETT SYNDROME AND EPILEPSY: EEG AND ITS TREATMENT

Chae JH (Seoul, Korea)

Coffee Break and Poster Presentation (2)

11:10-12:20

Coordinators: Posters 20-29 Izumi T (Oita, Japan), Nagai T (Osaka, Japan)
Posters 30-35 Hayashi M (Tokyo, Japan), Kira R (Fukuoka, Japan)

Lunch Time Seminar (2)

12:20-13:10

Chairperson: Segawa M (Tokyo, Japan)
Sponsored by Eli Lilly, K.K. Japan

O 17 12:20-13:10

AUTISTIC REGRESSION WITH AND WITHOUT EEG ABNORMALITIES FOLLOWED BY FAVOURABLE OUTCOME

Zappella M (Viareggio, Italy)

Treatment and Follow-up

13:10-15:20

Chairpersons: Yamamoto H (Kawasaki, Japan)
Parmeggiani A (Bologna, Italy)

O 18 13:10-14:00

LONG-TERM FOLLOW-UP OF CHILDREN WITH EARLY EPILEPTIC REGRESSION AND AUTISTIC FEATURES

Deonna T (Lausanne, Switzerland)

O 19 14:00-14:30

AUTISTIC INDIVIDUALS DEVELOPING EPILEPSY IN ADOLESCENCE: A FOLLOW-UP STUDY

Hara H (Yokohama, Japan)

O 20 14:30-15:20

AUTISM AND EPILEPSY: MOVING TOWARDS A COMPREHENSIVE APPROACH TO TREATMENT

Tuchman R (Miami, USA)

Coffee Break 15:20-15:40

Discussion Summary, Synthesis and Consensus Development

15:40-17:00

Chairpersons: Nomura Y (Tokyo, Japan)
Brooks-Kayal A (Denver, USA)
Tuchman R (Miami, USA)

Closing Address 17:00-17:10

Matsuishi T (President, 12th Annual Meeting of ISS, 2009)
Chang KP (Secretary General, 13th Annual Meeting of ISS, 2010, Taipei, Taiwan)

PROGRAM - POSTER PRESENTATIONS

Exhibition time: May 9, 8:30 - May 10, 17:00

P 01 **A CASE OF CRYPTOGENIC LOCALIZATION RELATED EPILEPSY WITH ASPERGER SYNDROME**
Iwasaki T, Nonoda Y, Hosoda N, Ishii M (Kanagawa, Japan)

P 02 **ASPERGER SYNDROME IN ASSOCIATION WITH BENIGN FAMILIAL INFANTILE CONVULSION: A NEW SYNDROME?**
Hirose M[1)], Haginoya K[1) 2)], Yokoyama H[3)], Nara C[1)], Uematsu M[1)], Tsuchiya S[1)] (Sendai[1) 2)] and Yamagata[3)], Japan)

P 03 **A CASE OF SYMPTOMATIC GENERALIZED EPILEPSY WITH AUTISM SHOWING MARKED BEHAVIORAL IMPROVEMENT WITH VPA**
Tanabe T[1)], Shimakawa S[2)], Fukui M[2)], Hara K[1)], Wakamiya E[3)], Tamai.H[2)] (Hirakata[1)], Takatsuki[2)] and Ibaraki[3)], Japan)

P 04 **OPTIMAL APPROACH FOR THE MANAGEMENT OF PSEUDO EPILEPTIC SEIZURES IN CHILDREN WITH DEVELOPMENTAL DISORDER**
Yasumoto S, Tomonoh Y, Ihara Y, Inoue T, Hirose S (Fukuoka, Japan)

P 05 **ALTERATION OF AUTISTIC CONDITIONS AFTER EPILEPSY SURGERY IN TWO CASES OF TEMPORAL LOBE EPILEPSY**
Hattori A[1)], Watanabe S[2)], Sugai K[1)], Sasaki M[1)], Takahashi A[3)], Otsuki T[3)], (Tokyo[1) 2) 3)], Japan)

P 06 **AUTISTIC LIKE BEHAVIORAL DISORDERS AND EPILEPSY**
Keihanidoust Z (Tehran, Iran)

P 07 **CLINICAL ANALYSIS OF CHILDREN WITH AUTISM SPECTRUM DISORDERS AND EPILEPSY**
Fujii A, Oguni H, Kodaira K, Inoko K, Hirano Y, Osawa M (Tokyo, Japan)

P 08 **SEIZURE CHARACTERISTICS OF EPILEPSY PATIENTS WITH AUTISM SPECTRUM DISORDERS**
Shimakawa S[1)], Tanabe T[2)], Wakamiya E[3)], Tamai H[1)] (Osaka[1) 2) 3)], Japan)

P 09 **CLINICAL CHARACTERISTICS OF EPILEPSIES IN PATIENTS WITH AUTISM**
Suzuki M, Maruyama K, Hayakawa C, Mizuno S, Nakamura M, Matsumoto A, Kumagai T, Miyazaki S (Kasugai, Japan)

P 10 **CHARACTERIZATION OF THE EPILEPSY ASSOCIATED WITH AUTISM SPECTRUM DISORDERS**
Maeda T[1)], Matsuo M[1)], Sasaki K[2)], Ishii K[3)] (Saga[1) 2) 3)], Japan)

P 11 **EPILEPSY IN CHILDREN WITH ASD AND OTHER NEURO-PSYCHIATRIC DISORDERS AT THE MENTAL HEALTH CLINIC IN BANGLADESH**
Banu SH[1)], Islam F[2)], Parveen M[2)], Dilara B[2)], Khan NZ[2)] (Dhaka[1) 2)], Bangladesh)

P 12 **BACKGROUND ACTIVITIES OF EEG IN THE CHILDREN WITH AUTISM SPECTRUM DIRORDERS**
Sawai C, Yoshioka S, Sakaue Y, Iwami M, Okada M, Takano T, Ohno M, Takeuchi Y (Otsu, Japan)

P 13 **SLEEP SPINDLES IN CHILDREN WITH PERVASIVE DEVELOPMENTAL DISORDERS**
Kimura I[1)], Kubota M[2)], Miyao M[3)], Komori T[4)] (Tokyo[1) 2) 3) 4)] , Japan)

P 14 **EEG FINDINGS IN AUTISM SPECTRUM DISORDERS WITH EPILEPSY**
Kanemura H, Goto Y, Aihara M (Yamanashi, Japan)

P 15 **INCIDENCE AND CHARACTERISTICS OF ELECTROCEPHALOGRAPHIC ABNORMALITIES AT INITIAL DIAGNOSIS IN CHILDREN WITH AUTISM SPECTRUM DISORDER**
Kim JY, Kim YO, Kim CJ, Woo YJ (Gwangju, Korea)

P 16 **ROLE OF EEG IN THE EVALUATION OF AUTISTIC DISORDERS**
Hung KL[1)], Lim AT[1)], Liaw HT[1)], Lu HH[2)], Li TC[3)] (Taipei[1) 2) 3)], Taiwan)

P 17 **EVIDENCE OF GABAERGIC DYSFUNCTION IN AUTISTIC BRAIN: A PROTON MAGNETIC RESONANCE SPECTROSCOPY STUDY**
Mori K, Toda Y, Fujii E, Kagami S (Tokushima, Japan)

P 18 **TWO CASES OF ASPERGER SYNDROME THAT WERE DIAGNOSED AT 2 YEARS OF AGE**
Imataka G), Yamanouchi H, Arisaka O (Tochigi, Japan)

P 19 **EPILEPTIC VISUAL AURA**
Alecu TR (Tirgu Mures, Romania)

P 20 **AUTISM RELATED 593-KB MICRODELETION OF 16P11.2 IN A MOTHER AND SON WITHOUT AUTISM BUT MR**
Yamamoto T[1)], Shimojima K[1)], Inoue T[2)], Fujii Y[2)], Ohno K[2)] (Tokyo[1)] and Yonago[2)], Japan)

P 21 **A FAMILIAL CASE OF LEOPARD SYNDROME ASSOCIATED WITH HIGH-FUNCTIONING AUTISM SPECTRUM DISORDER**
Watanabe Y[1)], Yano S[2)], Yoshino M[1)], Niihori T[3)], Matsubara Y[3)], Aoki Y[3)], Matsuishi T[1)] (Kurume[1)] and Sendai[3)], Japan); Los Angeles[2)], USA)

P 22 **SODIUM CHANNELS OF SCN1A GENE MUTATIONS IN GENERALIZED EPILEPSY WITH FEBRILE SEIZURE PLUS (GEFS+) SPECTRUM RELATED TO AUTISM**
Herini ES[1)], Patria SY[1)], Gunadi[2)], Yusoff S[2)], Sunartini[1)], Sutaryo[1)], Takada S[3)], Nishio H[2)] (Yogyakarta[1)], Indonesia; Kobe[2) 3)] , Japan)

P 23 **CDKL5 MUTATIONS IN PATIENTS WITH EARLY-ONSET INTRACTABLE EPILEPSY AND AUTISTIC BEHAVIOR**
Liang J-S[1)], Shimojima K[1)], Natsume J[2)], Fukazawa T[2)], Okumura A[3)], Hirasawa K[4)], Oguni H[4)], Osawa M[4)], Yamamoto T[1)] (Tokyo[1) 3) 4)], and Nagoya[2)], Japan)

P 24 **EFFECTS OF BETA-HYDROXYBUTYRATE ON NEUROGENESIS AFTER PILOCARPINE-INDUCED SEIZURES IN YOUNG MICE**
Kim DW[1)], Lee KS[2)] (Goyang[1)] and Daejeon[2)], Korea)

P 25 **CHARACTERISTICS OF INTELLIGENCE & LANGUAGE ABILITY IN CHILDREN WITH EPILEPSY**
Okazaki S[1)], Kuki I[1)], Kawawaki H[1)], Inoue T[1)], Kimura S[1)], Okada M[1)], Kusama Y[2)], Katada T[2)], Nagayasu K[2)], Manabe T[3)], Togawa M[4)], Shiomi M[5)], Tomiwa K[6)], (Osaka[1) 2) 3) 4) 6)] and Kyoto[5)], Japan)

P 26 **A STUDY ON BEHAVIORAL PROBLEMS IN CHILDREN WITH EPILEPSY**
Endoh F[1)], Kobayashi K[1)], Ogino T[2)], Ohtsuka Y[1)] (Okayama[1) 2)], Japan)

P 27 **CASE REPORT: STEREOTYPIC SELF-HITTING IN TWO PATIENTS WITH SYMPTOMATIC LOCALIZATION-RELATED EPILEPSY AND MENTAL RETARDATION**
Izumi T, Shimizu M, Okanari K, Korematsu S, Kiyota A (Oita, Japan)

P 28 **EATING PROBLEM IN A 31-MONTH-OLD GIRL WITH AUTISM SPECTRUM DISORDER**
Yamasaki Y[1)], Ogawa A[1)], Moriyasu Y[1)], Akiyoshi H[1)], Fukamachi S[1)], Yokoyama T[2)], Hirose S[3)] (Chikushino[1)], Kasuga[2)] and Fukuoka[3)], Japan)

P 29 **THE RELATIONSHIP BETWEEN SLEEP DISORDER AND SEIZURE DISORDER IN ANGELMAN SYNDROME: A CASE REPORT**
Ohya T, Nagamitsu S, Hara M, Yamashita Y, Matsuishi T (Kurume, Japan)

P 30 **THE CORRELATION BETWEEN 1H-MR SPECTROSCOPY (1H-MRS) AND CLINICAL MANIFESTATION WITH TUBEROUS SCLEROSIS COMPLEX (TSC)**
Imamura A[1)], Iwai A[1)], Terasawa A[1)], Miura R[1)], Ito R[1)], Orii KO[1)], Takahashi Y[2)] (Gifu[1)] and Shizuoka[2)], Japan)

P 31 **MR SPECTROSCOPY ANALYSES OF THE TWO CASES WITH SYMPTOMATIC WEST SYNDROME**
Iwai A[1)], Miura R[1)], Terazawa A[1)], Ito R[1)], Orii KO[1)], Imamura A[1)], Takahashi Y[2)], Kimata K[3)] (Gifu[1) 3)] and Shizuoka[2)], Japan)

P 32 **FDG-PET AFTER INITIAL TREATMENTS PREDICTS 10-YEAR DEVELOPMENTAL OUTCOME IN CRYPTOGENIC WEST SYNDROME**
Natsume J, Maeda N, Negoro T, Itomi K, Okumura A, Maruyama K, Kubota T, Kato K, Watanabe K (Nagoya, Japan)

P 33 **LONG-TERM FOLLOW-UP CASES OF INFANTILE SPASMS AFTER LESIONECTOMY**
Ko T-S[1)], Yum M-S[1)], Lee JK[2)], Kim DS[3)] (Seoul[1) 2) 3)], Korea)

P 34 **EPILEPSY IN MECP2 DUPLICATION SYNDROME**
Yanagihara K[1)], Okamoto N[1)], Yamada K[1)], Mogami Y[1)], Toribe Y[1)], Mano T[1)], Suzuki Y[1)], Nakagawa E[2)], Goto Y[2)], Honda S[3)], Inazawa J[3)] (Izumi[1)], Kodaira[2)] and Tokyo[3)], Japan)

P 35 **BRAIN WAVE RESULTS IN CHILDREN WITH ATTENTION DEFICIT HYPERACTIVE DISORDER AND TREATMENT RESULT WITH CENTRAL NERVOUS SYSTEM STIMULANTS**
Kim WS[1)], Park HJ[2)], Lee KS[3)] (Cheongju[1)] and Daejeon[2) 3)], Korea)

Abstracts - Oral Presentations

FREQUENT ASSOCIATION OF AUTISM SPECTRUM DISORDERS IN CHILDHOOD ONSET COMPLEX PARTIAL SEIZURE

1

12th ISS

Matsuo M [1)], Maeda T [1)], Sasaki K [2)], Ishii K [3)]
[1)] Department of Pediatrics, Faculty of Medicine, Saga University, Saga, Japan
[2)] Department of Pediatrics, Saga Prefectural Hospital, Saga, Japan
[3)] Department of Pediatrics, Saga Handicapped Children's Hospital, Saga, Japan

Objectives: Autism spectrum disorders (ASD) have a close relationship with epilepsy. A previous study showed complex partial seizure (CPS) to be the most frequent type of seizure in epilepsy with ASD. Patients with childhood onset CPS were retrospectively studied to investigate the prevalence of ASD in childhood onset CPS and characterize CPS associated with ASD.
Subjects: The study included 86 patients with CPS which manifested from one to nine years of age. Symptomatic CPS and Panayiotopoulos syndrome were excluded. The CPS with ASD (ASD group) was compared to those without ASD (non-ASD group).
Results: Thirty-five patients (40%) with childhood onset CPS were associated with ASD. In the ASD group, there was a male predominance (68.6%) and frequent seizures (60% were monthly or more). CPS without secondary generalization was more common in the ASD group (71.4%) than in the non-ASD group (35.3%). Frontal paroxysms on EEG were more common in the ASD group (56%) than in the non-ASD group (29%).
Conclusions: ASD is frequently associated with childhood onset CPS. Male, frequent seizure, and frontal paroxysms are risk factors for ASD association.

CLINICAL FEATURES OF EPILEPSY WITH PERVASIVE DEVELOPMENTAL DISORDER

2

12th ISS

Kurokawa T [1)], Yokomizo Y [1)], Lee S [2)], Kusuda T [2)]
[1)] Seiai Rehabilitation Hospital, Onojou, Japan
[2)] Department of Pediatrics, Kyushu University School of Medicine, Fukuoka, Japan

Purpose: To clarify the clinical features of patients with epilepsy and pervasive developmental disorder (PDD).
Methods: We examined twelve out-patients with epilepsy as well as PDD who visited Seiai Rehabilitation Hospital.
Results: Genders were 7 males and 5 females. The ages at the onset of seizures ranged from 10 to 19 years in 8 out of 12 cases. The types of seizures were generalized in all cases, they include the follows: astatic-drop 2 cases; tonic-to-astatic one; absence (decreased consciousness) + generalized tonic clonic seizures (GTCS) 3; GTCS 4; and myoclonic + psychomotor 2. The mental development distributed from normal to extremely severe retardation. Paroxysmal abnormalities on EEGs were focal at the frontal area in 6 cases (50%) and other findings in 6. Presumptive etiologies were prenatal 6 (family history for PDD one case, and for epilepsy one, twin pregnancy 2, and others 2), perinatal 2, postnatal one, and unknown 3.
Conclusions: The seizures occurred mostly after the onset of developmental disorders. The etiologies were prenatal in 50% of the cases, and frontal lobe lesions were associated in 50% in terms of EEG findings.

3

12th ISS

EPIDEMIOLOGY OF ASD AND EPILEPSY

Saemundsen E
Director of Services, Division of Autism and Communication Disorders
State Diagnostic and Counseling Center, Kopavogur, Iceland

Objectives: a) to describe the epidemiology of ASD, and b) to investigate the association between ASD and epilepsy when seizures precede the diagnosis of ASD.
Methods: Recent epidemiological literature on ASD was reviewed. The prevalence of ASD was studied in a cohort of children with a history of unprovoked seizures in the first year of life. Further, it was investigated in a case-control design whether infantile spasms were an independent risk factor for ASD.
Results: Epidemiological studies of ASD appear to concur that the prevalence of ICD-10/DSM-IV ASD has stabilized at approximately 6 per 1,000. The incidence of unprovoked seizures in the first year of life was 163.4 per 100,000 person years (95% CI 135.6-195.3). The prevalence of ASD in children with unprovoked seizures in the first year of life was 13.7% (95% CI 7.5-22.3). The adjusted odds ratios were 1.55 (95% CI 0.33-7.37) for children with infantile spasms and 8.73 (95% CI 1.88-40.54) for children with symptomatic origin of seizures.
Conclusions: High prevalence of ASD was found in children with a history of unprovoked seizures in the first year of life compared to estimates from the general population. Symptomatic origin of seizures increased the risk of ASD, rather than type of epilepsy.

4

12th ISS

AUTISM SPECTRUM DISORDER (ASD) AND EPILEPSY- HONG KONG PERSPECTIVE

Wong V
Division of Child Neurology/Developmental Paediatrics/Neurohabilitation
Department of Paediatrics & Adolescent Medicine, the University of Hong Kong, Hong Kong

Objectives: To study the relationship between epilepsy and ASD.
Methods: Children with ASD diagnosed with DSM IV criteria and/or ADIR/ADOS and followed up during 1984-2008 were recruited. A comparison was made between those with Idiopathic or Secondary Autism. Possible risk factors were compared for those with (E+) and without epilepsy (E-).
Results: Of 1066 children (933 male; 133 female) with ASD, 94/1066 (8.9%) had epilepsy, of which 59/94 (62.8%) and 35/94 (37.2%) were idiopathic and secondary ASD respectively. Secondary ASD included Dravet disease (12), Tuberous Sclerosis (7), Rett Syndrome (6); Fragile X Syndrome (1), other syndromes (11). The median age of onset of seizures was 18 months. 12/41 (29%) had "age of onset of first seizure" earlier than "age at first symptom of autism" and 54/81 (67%) had "age of onset of first seizure" earlier than "age at initial diagnosis of autism". Of types of seizures, 18 % had partial seizures only, 56 % had generalized seizure only, and 20 % had both. 8 (8.5%) had infantile spasm, 2 (2.1%) had photosensitivity, and 10 (10.6%) had status epilepticus. Comparison of E+ and E- groups showed that febrile seizures occurred in 29/94 (30.9%) and 20/972 (2.1%) respectively. The age of occurrence of FS was earlier for secondary versus idiopathic ASD. Analysis of risk factors between E+ and E- group showed that the following were significantly related to developing epilepsy: gender, abnormal perinatal history, family history of epilepsy, age of autistic diagnosis, dysmorphic features, history of febrile convulsion and lower intelligence. Those with secondary ASD (83%) had poorer control of seizures than idiopathic (58%)(p=0.013). Four children died and there was a significant relationship between those in secondary (3) than idiopathic (1) ASD (p=0.000) with mortality.
Conclusions: Children with secondary ASD had a higher incidence and poorer prognosis with epilepsy.

AUTISM AND EPILEPSY: HISTORICAL PERSPECTIVE

5

12th ISS

Tuchman R

Miami Children's Hospital Dan Marino Center, Center for Autism and Related Disorders, University of Miami, Department of Neurology, University of Miami Miller School of Medicine at Affiliated Institution, Miami, USA

Epilepsy has been associated with autism since the initial description of the disorder by Leo Kanner in 1943. In the 1960's the first studies on the relationship of autism to epilepsy and to electroencephalogram (EEG) abnormalities emerged. These studies were among the first to suggest that autism was a disorder of brain function and helped pave the road to the inquiry into the neurobiological basis of autism. In the early1970s a relationship between infantile spasms and autism was first described. By the late 1970s and early 1980s there was a growing interest in specific aspects of the relationship of autism and epilepsy and of epilepsy and EEG abnormalities to language disorders. With the question posed being what is the contribution of epilepsy and epileptiform activity to the behavioral phenotype of autism?

In the 1980s studies on autism-epilepsy started to investigate the differences in rates of epilepsy in different groups of children with autism. During this period of time the increased risk of seizures in autism at puberty was identified and the relationship of autism and epilepsy to tuberous sclerosis was described. Criteria for autism and advances in the classification of the epilepsies were refined during the 1980s and early 1990, allowing for a more scientific approach to the study of autism-epilepsy. By the 1990s the relationship between autism and epilepsy had been established but this relationship was still poorly understood. During the 90's studies focused on the specific risk factors for development of epilepsy in children with autism. In addition the relationship of epilepsy and EEG findings to autistic regression was investigated highlighting the relationship between epileptic encephalopathies and autism.

In this decade our understanding of autism and epilepsy as separate disorders continues to progress and offers important clues as to the common pathophysiological mechanisms that account for the co-existence of these disorders. The conceptualization of this co-existence has been labeled the autism-epilepsy phenotype and has shifted the focus of research interest to the genetics and the molecular biology common to both the autisms and the epilepsies. A review of the autism-epilepsy phenotype, while variable along multiple dimensions such as seizure type, reveals a consistent set of core features including intellectual disability, sleep related EEG changes, circadian rhythm abnormalities, an increased prevalence of developmental regression, and receptive language impairments. There is strong evidence for genetic and molecular contributions to the autism-epilepsy phenotype. Moving forward our understanding of the autism-epilepsy phenotype will require clinical and research efforts to address several key issues. The issues include the need for: a more detailed description of the autism-epilepsy phenotype, a greater understanding of its underlying pathophysiology common to both autism and epilepsy, predictive algorithms to identify individuals at greatest risk, and innovative ways to tailor interventions to the special features of the autism-epilepsy phenotype.

PATHOPHYSIOLOGY OF PERVASIVE DEVELOPMENTAL DISORDERS AS REVEALED BY EEG, MEG, WITH PARTICULAR REFERENCE OF EPILEPSY

6

12th ISS

Kawasaki Y [1)], Shinomiya M Y [1)], Niwa S [2)]

[1)] Musashino Child Development Clinic, Tokyo, Japan

[2)] Fukushima Medical College, Fukushima, Japan

In this presentation, we describe the neurophysiological bases of pervasive developmental disorders (PDD) in terms of clinical and electrophysiological features, namely epileptic seizures, paroxysmal electroencephalographic (EEG) abnormalities, and deviated sensory perceptions. PDD displays a high comorbid ratio of epilepsy with its peak onset time at the adolescent period. We observed electroencephalographic abnormalities frequently appear in the frontal regions in subjects with PDD, and we speculate that the EEG abnormalities underpin there epileptic seizures. We named the paroxysmal EEG activities as "Paroxysm at F" (PaF). PaF also appears in EEGs of PDD subjects who do not develop epileptic seizures. Therefore, we supposed PaF is a neurophysiological reflection of possible common brain dysfunction underlying PDD, rather than a simple reflection of epileptic seizures. Using the magneto-encephalography, we could identify the origin of PaF at the medial frontal cortex and anterior cingulated cortex.

In addition, it was characteristic of PDD subjects that they demonstrate faster basic activities as compared to healthy controls and non-autistic mentally retarded subjects.

PDD subjects also exhibit rhythmic fast activities in the frontal regions. These findings lend support to the notion that the anterior thalamocortical system is involved in the pathophysiology of PDD. Moreover, many PDD subjects are known as hypersensitive in sensation, which suggests thalamic commitment in PDD.

Based upon the findings cited above, the pathophysiology of PDD is thought to consist of systematic dysfunction in the medial frontal cortex, the anterior cingulate gyrus, and the thalamus.

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12th ISS

AUTISM SPECTRUM DISORDERS (ASD) AND EEG PAROXYSMAL ABNORMALITIES: HOW ARE THEY LINKED?

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Objectives: ASD are heterogeneous conditions. Although epilepsy and EEG abnormalities have been described in ASD, their recurrence is extremely variable.
Methods: Our sample includes 345 patients affected by ASD. We evaluated occurrence and features of epilepsy and EEG paroxysmal abnormalities in different age and diagnostic subgroups.
Results: 223 patients had autistic disorder, 94 atypical autism, 21 Asperger disorder, 7 childhood disintegrative disorder. Symptomatic ASD was present in 35.9% of cases, familial antecedents for epilepsy in 16.2%, epilepsy in 24.9% and EEG paroxysmal abnormalities without epilepsy in 23.5%. Focal EEG abnormalities prevailed in central and temporal regions (31.4%). Epileptic seizure onset was early or during adolescence. EEG abnormalities were more common during childhood. Partial and generalized epilepsies were both represented.
Conclusions: Epilepsy and EEG abnormalities prevailed in symptomatic cases. An underlying genetically determined pathological process could be the origin of different manifestations of cerebral dysfunction. The current understanding of the link between epilepsy and autism is still limited at the etiologic level, but from a clinical point of view this association should be routinely investigated.

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12th ISS

CORRELATION OF EEG ABNORMALITIES IN ASD WITH SYMPTOMS AND TREATMENT

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Objectives: It is often experienced that loci of seizure waves, brain parts where brain dysfunctions occur and symptoms of ASD correlate. In this study, we examined the connection between localization of EEG paroxysmal aberration and symptoms of ASD as well as the effects of the treatment for patients who have these problems.
Methods: The subjects were children with ASD visiting our clinic for the treatment. Consent to this study was obtained from the subjects. In the following cases, the subjects were treated with anticonvulsant agent: 1) epileptic seizures were observed and 2) the brain parts where brain dysfunctions occurred coincided with the localizations of seizure waves.
Results: Localizations of paroxysmal bursts were various from case to case, and paroxysmal bursts had developed from all loci of the brain. Those subjects with spikes in prefrontal area had tendency of having less self-control and more impulsive action, and had difficulty in language acquisition. Temporary atypical absence, autonomy neurological symptoms, and affective alteration were observed in those having sporadic seizure waves in bilateral forehead. Those having spikes in the right parietal area had difficulty in acquiring movement imitation. Psychic blindness, hyperorality, hypermetamorphosis, hypoemotionality, abnormality and aberration of alimentary flavor were observed in those having spikes in temporal region. The brain functions of those who took anticonvulsant had improved.
Conclusions: EEG abnormalities in people with ASD are not simplex and there is a wide variation in localization. When EEG paroxysmal aberration localizes in specific brain parts, the brain functions of these parts are disturbed.

BRAIN PERFUSION SPECT AND EEG FINDINGS IN AUTISM WITH EPILEPSY

9

12th ISS

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Objectives: We performed brain perfusion single-photon emission computed tomography (SPECT) to elucidate the abnormal brain region in children with autism spectrum disorders (ASD). Using SPECT, brain function of ASD children with intractable epilepsy was screened.
Methods: Fifteen patients, aged 4-16 years, underwent multimodal examinations (MRI, interictal ECD-SPECT, EEG, MEG, ictal ECD-SPECT in some patients) to investigate their suitability for surgical treatment. All children were diagnosed as ASD by DSM-IV and had no basic disorders. Despite medical treatment for more than one year, all experienced at least one seizure per month. Each SPECT result was statistically analyzed by comparison with standard SPECT images obtained from our institute (easy Z-score imaging system; eZIS). The relationship between eZIS pattern and EEG abnormalities or clinical symptoms was investigated.
Results: All children showed normal MRI, focal abnormal pattern in eZIS, and focal spikes in EEG. eZIS revealed a mixed hypoperfusion pattern, especially in the front pole, medial frontal lobe, and/or temporal lobe. These patterns were not necessarily related to the focus observed on EEG. Some relationships were observed between the eZIS pattern and clinical symptoms.
Conclusions: eZIS is useful not only for the search of the epileptic focus but also for examination of the low functional brain region.

EPILEPSY IN AUTISM; A PATHOPHYSIOLOGICAL CONSIDERATION

10

12th ISS

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The frequency of epilepsy is common in idiopathic autism (IA), and two peaks of occurrence in early childhood and adolescence are known. However, the underlying pathophysiologies of the epilepsy in IA remain to be understood. We reported that the abnormality of locomotion is significantly high in IA, and that it is the important factor to discuss the pathophysiology of IA.

In this presentation we discuss our observations of epilepsy in IA. The subjects are 88 cases (69 male and 19 female) of IA with epilepsy who are currently followed in this clinic over 10 years. Their ages ranged from 15 to 42 years of age (10 cases in the teens, 24 cases in the twentieth, 49 cases in the thirtieth and 5 cases in the fortieth). The cases with the ages of onset of epilepsy below 10 years of age (group A) and above 10 years of age (group B) were 49 % and 51 % respectively. The oldest age of onset of epilepsy was 26 year-7 months-old, and five cases had onset in their 20th including this case. The early developments of locomotion and occurrences of EEG findings were compared in the two groups, A and B. As to the locomotion, the ages of starting to crawl and its pattern were checked. The most of the group A crawled at normal age ranges. In contrast the majority of the group B had either never crawled or showed shuffling pattern. The neurological examination of most cases of group A and B at current ages showed the abnormal pattern of crawling with dorsi-flexed posture of the toes. The EEG findings of group A consisted with spike focus in central area, and of group B in mid-temporal and frontal foci developing after adolescence.

From these observations we speculate that the pathophysiologies of epilepsy of IA differ in younger and older onset groups. The ages of onset of epilepsy in group A are similar to the childhood onset epilepsy. The ages of onset of epilepsy in group B and their mid-temporal and frontal foci in EEG suggest the involvement of the specific tempo-frontal pathways developing after adolescence. The high frequency of abnormal locomotion during late infancy in group B suggest the involvement of early monoaminergic systems, particularly hypofunction of serotonergic system, which involves pedunculo-pontine nucleus, then hypofunction of striatal and limbic dopaminergic systems, and frontal cortex after adolescence and adulthood.

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12th ISS

CLINICAL AND COGNITIVE FEATURES AND POSSIBLE NEURAL BASES OF PERVASIVE DEVELOPMENTAL DISORDER

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Objective: The aim of the presentation is to review clinical and cognitive features of pervasive developmental disorder (PDD) in a recent neuroscientific perspective, and to discuss possible neural bases of PDD.
Methods: First, the unique clinical features and research findings on cognition of PDD (autistic disorder, Asperger's disorder, PDD-NOS) will be reviewed. Then, possible neural bases of PDD will be discussed based primarily on previous neurological findings concerning PDD and similarities to other neurological disorders such as mesial temporal lobe epilepsy and obsessive-compulsive disorder (OCD)
Results: Although clinical findings of PDD vary greatly across ages and subtypes, most features are considered to be the manifestation of impairment in social interaction (such as a lack of joint attention, imitation, and empathy) and a restricted range of interest and repetitive behavior. The neural base of the impairment of social interaction, a core clinical feature, may be dysfunction of amygdala and neocortices involved in social/emotional processing (such as fusiform face area, superior temporal sulcus, and orbito- and medial-frontal cortex), and that of the restricted range of interest and repetitive behaviors may be the neural structures related to OCD (such as caudate nucleus).
Conclusions: The neural bases of PDD may derive from the underdevelopment and/or dysfunction of medial temporal (limbic) structures and related cortices.

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12th ISS

EPILEPSY AND ASD: ARE THERE COMMON DEVELOPMENTAL MECHANISMS?

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Autism Spectrum Disorders (ASD) and epilepsies are heterogeneous disorders that have diverse etiologies and pathophysiologies. The high rate of co-occurrence of these disorders suggest potentially shared underlying mechanisms. A number of well-known genetic disorders share epilepsy and autism as prominent phenotypic features, including Tuberous Sclerosis, Rett's Syndrome, Fragile X, and Neurofibromatosis Type I. In addition, mutations of a series of genes involved in neurodevelopment, including ARX, DLX2, FOXP2, PTEN, UBE3A, Neuroligins and Semaphorins, have been identified in children with epilepsy, ASD or often both. Finally, in animal models, early life seizures can result in cellular and molecular changes also seen in models of ASD. Increased understanding of the common genetic, molecular and cellular mechanisms of ASD and epilepsy may provide insight into their underlying pathophysiology and elucidate new therapeutic approaches of both conditions.

X-LINKED FEMALE-LIMITED EPILEPSY AND COGNITIVE IMPAIRMENT CAUSED BY PROTOCADHERIN 19 MUTATIONS

13

12th ISS

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Objectives: To identify the causative gene for X-linked Epilepsy and Mental Retardation limited to Females (EFMR) which has an unusual pattern of sex-limited expression. EFMR spares transmitting males and affects only carrier females. EFMR is characterized by early onset seizures in previously normal infants, followed by developmental regression of varying severity.

Methods: Aided by systematic re-sequencing of 737 X chromosome genes we identified mutations in the Protocadherin 19 (PCDH19) gene in seven EFMR families.

Results: Five mutations result in the introduction of a premature termination codon. Study of two of these demonstrated nonsense mediated decay of mRNA. The two missense mutations are predicted to affect adhesiveness of PCDH19. Murine and human brain expression analyses of PCDH19 support an important role in brain function.

Conclusions: These results identify protocadherins as a new gene family directly associated with epilepsy and brain cognition. We are now investigating the role of PCDH19 in other related neurological conditions.

A CHROMOSOME- ENGINEERED MOUSE FOR HUMAN 15q11-13 DUPLICATION AS AN AUTISM MODEL

14

12th ISS

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Autism is a complex psychiatric illness that has received considerable attention as a developmental brain disorder. Substantial evidence suggests that chromosomal abnormalities contribute to the risk of autism. The duplication of human chromosome 15q11-13 is known to be the most frequent cytogenetic abnormality in autism. We have modeled this genetic change in mice using chromosome engineering to generate a 6.3-Mb duplication of the conserved linkage group on mouse chromosome 7. Mice with a paternal duplication display autistic behavioral features such as poor social interaction, behavioral inflexibility, abnormal ultrasonic vocalizations, and correlates of anxiety. An increased MBII52 snoRNA within the duplicated region, affecting the serotonin 2c receptor (5-HT2cR), correlates with altered intracellular Ca^{2+} responses elicited by a 5-HT2cR agonist in the neurons in mice with a paternal duplication. This first chromosome-engineered mouse model for autism replicates various aspects of human autistic phenotypes and validates the relevance of the human chromosome abnormality. This model will facilitate forward genetics of developmental brain disorders and serve as an invaluable tool for therapeutic development.

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12th ISS

OVERLAP-RELATIONSHIP BETWEEN EARLY ACQUIRED EPILEPTIC APHASIA (LANDAU-KLEFFNER SYNDROME, LKS) AND PERVASIVE DEVELOPMENTAL DISORDER (PDD)

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LKS can start very early in the 1st or 2nd year of life, so that no loss of language is suspected and only stagnation of emerging language or fluctuations in the dynamics of development is at best recognized. On the other hand, autistic regression (which always involves language as well) occurs in about a third of children later diagnosed as having idiopathic autism. These two sets of clinical facts that have emerged in the last 2o to 3o years have led to many EEG studies (and more recently MEG) in cohorts of children with either developmental language disorders or autism with/without autistic regression searching for focal/multifocal epileptic discharges and continuous spike-waves during sleep (CSWS), the hallmark of LKS. When present, various antiepileptic therapies were undertaken trying to suppress the discharges and see if a clinical benefit could be seen. On the whole, no firm evidence has come to implicate epilepsy (or epileptic EEG discharges) as a causal factor in most of the children with specific developmental language disorders or autism. Recently, a large study of children who presented with either early language regression alone or language regression associated with an autistic spectrum disorder found that a subgroup (mainly those with language regression alone, seizures and marked EEG abnormalities) did meet criteria for Landau-Kleffner syndrome.This suggests that there are some children in whom epilepsy could be the causal or at least a significant factor responsible for language(+/-) autistic regression. There are also important individual longitudinal case reports of children with epileptic EEG abnormalities as seen in LKS and a behaviour within the autism spectrum disorder in whom a specific verbal language disorder became obvious when normalization in one or all of the triad of behavioral impairments considered "necessary" to define autism (social difficulties, impaired communication rigid repetitive traits) occurred rapidly in correlation with successful antiepileptic treatment. This presentation will focus on such longitudinal case reports in whom co-occurrence, association-dissociation of the specific verbal language disorder on the one hand and the autistic features on the other evolved over time in an original and significantly different way (with a possible direct role of epilepsy) from other children with severe developmental language disorders (with often associated autistic features) but from presumably other causes.

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12th ISS

RETT SYNDROME AND EPILEPSY: EEG AND ITS TREATMENT

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Rett syndrome (RS) is a severe neurodevelopmental disorder almost exclusively affecting female patients and responsible for the mutations in methyl-CpG binding protein 2 gene (MECP2 gene). RS is also a second most common cause of autistic spectrum disorders in female followed by Down syndrome. Its clinical features are characterized by loss of acquired skills, such as hand purposeful movements and speech, hand stereotypic movements, deceleration of head growth, scoliosis, and epilepsy. Epilepsy is frequently associated with RS, which is known to be occurred in 50%-94% of all patients. The seizure pattern is often quite typical but sometimes various type of seizures from complex partial seizures, absence, to generalized tonic clonic seizures coexist in same patients, and resistance to antiepileptic drug (AED) treatment may not be rare, but which is known to be lower than general epilepsy population.
Electroencephalographic finding (EEG) abnormalities have also been proposed that typical EEG changes and a characteristic developmental pattern of EEG have been correlated with the four clinical stages of RS. According to several recent reports, there is lack of association between clinical severity of epilepsy and EEG appearance and 42% of the clinical events identified by parents were not to be associated with EEG seizure discharges under EEG monitoring.
Practically, in some clinical situations, some of the typical symptoms of RS such as autistic hand stereotypy, abnormal breathing, unresponsiveness and vacant spells can be difficult to differentiate from epileptic seizure activities. In other hands, we can mislead the true epileptic seizure activities as autistic abnormal behaviors. Here we present some of our experience of epilepsy in RS and RS - like Angelman syndrome through video EEG monitoring.
Conclusively EEG features in RS are not diagnostic, however, EEG patterns, associated epilepsy and response to AED treatment seems to be changed according to clinical stages. Therefore AED therapy should be decided considering the patient's clinical stages and if needed long-term video monitoring might be useful for appropriate treatment decision in patients with autistic spectrum disorders like Rett syndrome.

AUTISTIC REGRESSION WITH AND WITHOUT EEG ABNORMALITIES FOLLOWED BY FAVOURABLE OUTCOME

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12th ISS

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Objectives: To explore the relationship between autistic regression with and without EEG abnormalities and favourable outcome.
Methods: Follow up data on 534 children aged below 5 years and diagnosed as ASD.
Results: Cases with regression were close to 30% usually with persistent ASD, intellectual disabilities and EEG abnormalities. Children who went off autism and recovered entirely their intellectual and social abilities were close to 7%. Few of them included examples of pharmacologically treated Landau and Kleffner syndrome and other similar complex cases with abnormal EEG. The majority was represented by 36 (6.7%) children, mostly males, with a dysmaturational syndrome: their development was initially normal up to 18 months when an autistic regression occurred accompanied by the appearance of motor and vocal tics. Relational therapies were followed by rapid improvement. By 6 years all children had lost features of ASD and their I.Q. was in most cases between 90 and 110. Their EEG was normal, convulsions absent. In a few of them recovery was spontaneous. 17 children were followed after 5 years 6 months: 12 (70%) had ADHD, 10 (56%) persistent tics. Tics were often present in parents and relatives, ASD absent. No children off autism were noticed among those subjects with ASD and without regression.
Conclusions: In this series children off autism were either early onset epilepsies or cases of dysmaturational syndrome: autistic regression was present in all.

LONG-TERM FOLLOW-UP OF CHILDREN WITH EARLY EPILEPTIC REGRESSION AND AUTISTIC FEATURES

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12th ISS

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Among children whose epilepsy has a major direct impact on cognition or behaviour , especially those with documented regression and who improve with permanent control or remission of their epilepsy (medical or surgical) , the changes observed in the autistic symptoms are the most difficult to evaluate formally (as opposed to measures of IQ or language functions) . When marked improvement is seen in this domain, the question is whether it is the natural history of the autism spectrum disorder (ASD) or if it is a direct but partly reversible manifestation of the epilepsy. Retrospective cohort studies can not answer this question, but prospective individual case studies followed over a number of years may give some hints, especially those in whom the autistic regression and its direct link with epilepsy was documented early on. The question is not whether the children "loose" a formal diagnosis of ASD, but if they significantly improved (or not) in one of the several classically affected domains (socialization, communication, rigid/stereotypic behaviour) , if this led to major changes in their life, and how it evolved over the years. We draw form our personal longitudinal studies in various epileptic syndromes or specific diseases (late infantile spasms,tuberous sclerosis, surgical cases, etc) and other sources from the literature which can contribute to answer these unresolved questions.

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12th ISS

AUTISTIC INDIVIDUALS DEVELOPING EPILEPSY IN ADOLESCENCE: A FOLLOW-UP STUDY

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Several studies have pointed out that there are two unique "peak-ages" of epilepsy onset in autism; the first one is before 3 years of age, and the second is during adolescence. The former peak is comprised of epilepsies in symptomatic autism. For instance, an infant affected by West syndrome was diagnosed having an autistic disorder after 3 years of age. The next peak is observed in epilepsies that occur in so-called idiopathic autism with no major complications or preexisting diseases before the diagnosis of autistic disorder.

Similar conclusions from the studies dealing with epilepsies in idiopathic autism are; (1) lower cognitive development is higher risk of epilepsy onset, (2) epileptiform EEG abnormalities recognized before adolescence correlate to development of epilepsy during adolescence, and (3) the main seizure type is complex partial seizures evolving to secondarily generalized seizures, although other types of seizures are also seen.

Controversy exists as to whether or not (1) there is any sex difference, (2) laterality and/or specific area of epileptiform EEG abnormalities are related to developing epilepsy, and (3) a refracted course or speech loss during infancy is one of the risk factors.

From the prognostic point of view, developing epilepsy is an unfavorable factor for idiopathic autism.

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12th ISS

AUTISM AND EPILEPSY: MOVING TOWARDS A COMPREHENSIVE APPROACH TO TREATMENT

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We have effective interventions for epilepsy and for autism but interventions unique to the autism-epilepsy phenotype have not been formally developed. Despite our present understanding that cellular/molecular abnormalities of neurons or network abnormalities of the brain lead to epilepsy and to the phenotypic cognitive and socio-communicative behaviors characteristic of autism, there is at most limited success, with therapeutic pharmacological agents that target abnormalities in these systems. This lack of specificity in treatment may be partly responsible for the relatively poor outcomes reported for individuals with both epilepsy and autism.

The autism-epilepsy phenotype represents a new way of conceptualizing the co-existence of epilepsy in autism. The logic for a unified approach that includes specific pharmacological agents and educational-behavior interventions in treating children with the autism-epilepsy phenotype is that eliminating seizures early will enhance the capacity of intensive behaviorally-based educational interventions to remediate the associated neurodevelopmental deficits. Regardless of etiology this approach takes advantage of the neural plasticity of the developing brain once the brain is unburdened of the seizures.

The clinical and genetic disorders associated with the autism-epilepsy phenotype provide an important clinical source of information that should allow the translation of molecular information to rational and specific intervention. Current thinking is that genetic and molecular mechanisms play a major contributing role to both the pathophysiology of the autism phenotype and the epileptic disorder. For instance, 15q13.3 deletions appear to contribute to risk for both autism and idiopathic generalized epilepsy. GABAergic mechanisms also play a role in seizure development in autism. Specifically, neuronal system level dysfunction in GABAergic interneurons at the level of the minicolumns and alterations in GABA (B) brain receptors are both likely contributors to the increased seizure susceptibility in autism.

There are also several clinical syndromes such as tuberous sclerosis, fragile X and Rett syndrome in which the autism-epilepsy phenotype is commonly present. These clinical models suggest that epilepsy in autism is both a byproduct of the underlying network dysfunction and to a lesser degree a contributor to further disruption of this network (e.g., exacerbating cognitive and socio-communicative impairments). The autism-epilepsy phenotype most likely represents the confluence of multiple genes across a variety of pathways. In addition abnormalities of synaptic structure and function are central to the brain basis of autism and of epilepsy. Translation of basic science and mouse model data will be crucial to the development of clinical interventions for the autism-epilepsy phenotype. One area of focus is the genes controlling circadian rhythms that modulate protein complexes important in synaptic development and in the normal balance between excitation and inhibition in brain circuits. Understanding the processes these specific genes regulate will help in further defining the autism-epilepsy phenotype and in devising intervention strategies specific to this phenotype.

This discussion will focus on how to recognize, assess and treat the early signs of autism and epilepsy. Advances in our understanding of the molecular biology of autism and of epilepsy now allow us to conceptualize intervention strategies that may positively change the developmental trajectory of both autism and epilepsy. The autism-epilepsy phenotype is a potentially distinct entity resulting from diverse etiologic mechanisms thus requiring comprehensive intervention efforts. We propose a combined pharmacological-educational-behavioral protocol developed for managing the complex autism-epilepsy phenotype.

Abstracts - Posters

A CASE OF CRYPTOGENIC LOCALIZATION RELATED EPILEPSY WITH ASPERGER SYNDROME

POSTER 1 12th ISS

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Background: We report on the possible link between localization-related epilepsy of origin around frontal lobe and Asperger syndrome.
Clinical Details: A 13-year-old boy born through normal delivery was diagnosed with Asperger syndrome at 7 years old based on the criteria laid out by the DSM -IV, due to the presentation of characteristic clinical symptoms and the results of a WISC-III assessment. Four months later, he experienced complex partial seizures, and was prescribed sodium valproate for the first time. Carbamazepine was supplementarily prescribed for status epilepticus at 8 years old, after which, his convulsions disappeared. Presently, he attends a special support class at a public junior high school due to his Asperger syndrome. In a cranial CT scan and cranial MRI scans, marginal irregularity of the lateral ventricle circumference could be seen. However, these scans did not show hypoplasia of the cerebellar vermis. The patient also has presented no neurological symptoms, such as paralysis. EEG at 9 years old showed unilateral and frontal paroxysmal discharges.
Conclusions: Abnormality in the frontal area, which was strongly indicated by the EEG findings, could be a cause of epilepsy and pervasive developmental disorder, including Asperger syndrome.

ASPERGER SYNDROME IN ASSOCIATION WITH BENIGN FAMILIAL INFANTILE CONVULSION: A NEW SYNDROME?

POSTER 2 12th ISS

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Background: Benign familial infantile convulsion (BFIC) is an autosomal dominant epileptic disorder. A part of patients later develops paroxysmal kinesigenic choreoathetosis, which is now recognized as ICCA syndrome (IC with choreoathetosis); OMIM602066. Asperger syndrome is a common neurological condition, while the causative genes are still under investigation. Here we report sisters with BFIC who were later diagnosed as having Asperger syndrome.
Case Report: Family history revealed that father, paternal grandfather, one paternal cousin as well as these sisters had seizures in infancy. Sisters were born normally. Episodes of eye deviation, loss of consciousness and generalized tonic seizure developed at age of 5 and 6 months old, respectively. EEG and brain MRI were normal. They were diagnosed as BFIC and seizures were easily controlled. Sisters were thereafter diagnosed as Asperger syndrome at 9 and 4 year-old. Both have qualitative social interaction and communication impairments, and restricted interests with speech fluency.
Conclusions: An association of Asperger syndrome with BFIC implies a new clinical entity with genetic linkage of both disorders as seen in ICCA syndrome, although genetic study is needed for further understanding.

POSTER 3
12th ISS

A CASE OF SYMPTOMATIC GENERALIZED EPILEPSY WITH AUTISM SHOWING MARKED BEHAVIORAL IMPROVEMENT WITH VPA

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Objectives: We report a 3-year old girl who presented with autistic regression and symptomatic generalized epilepsy. The CARS (childhood autism rating scale) improved in response to medication with valproic acid (VPA).
Cases: She had started to speak words at age 2 years. Her parents subsequently noticed frequent daily involuntary movements including myoclonic jerking of the upper extremities with upward-gazing deviation of the eyes with arrest of behavior. She was brought to our clinic at age 3 years. EEG monitoring revealed the movements to be associated with generalized spike-wave complexes, confirming a diagnosis of myoclonic epilepsy. Eye contact was extremely poor. Both verbal and non-verbal communications were almost impossible. Tool operation was inadequate and peculiar. The CARS showed severe autism; 38.5 points. Epileptic seizures and EEG discharge ceased immediately after starting VPA. We noticed that communication improved. Social smiling and sympathetic emotions also gradually improved. The CARS score decreased to 26 points. Marked improvement was shown in 'relationships to others'.
Conclusions: VPA treatment can affect behavior. The CARS score was useful for objectively assessing treatment efficacy.

POSTER 4
12th ISS

OPTIMAL APPROACH FOR THE MANAGEMENT OF PSEUDO EPILEPTIC SEIZURES IN CHILDREN WITH DEVELOPMENTAL DISORDER

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Objectives: To consider optimal management of pseudo epileptic seizures (PSE) in children with developmental disorders, we herein show our approaches to 3 children with such conditions.
Methods: Case 1 was a 13-year-old girl, Case 2 an 11-year-old boy and Case 3 a 6-year-old girl. All had been treated for refractory epilepsy while their seizures had been suspected to contain PSE. Simultaneous ictal video EEG monitoring was performed several times. Life histories were taken in detail. We performed WISC III for all the patients.
Results: The diagnoses given to Case 1, 2 and 3 were an autism spectrum disorder with mild mental retardation, mild mental retardation and Asperger syndrome, respectively. We verified that some of their seizures were PSE based on both the symptoms and lack of ictal spikes on the EEG. They could not adapt to their environments very well because of their developmental disorder. Having frequent interviews, we cooperated with their families and teachers, and gave our best support to the patients and their families to improve the patients' environments.
Conclusions: PSE of the patients severely impaired their quality of life. Therefore, PSE should be accurately diagnosed as well as the underlying developmental disorders should be treated accordingly.

ALTERATION OF AUTISTIC CONDITIONS AFTER EPILEPSY SURGERY IN TWO CASES OF TEMPORAL LOBE EPILEPSY

POSTER 5 12th ISS

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Objectives: The pathogenesis of autistic condition is not elucidated, but involvement of limbic system is postulated. Autopsy studies have reported increased neuron density and decreased neuron size in the hippocampus and amygdala and neuroimaging studies have showed decreased volume of the hippocampus and amygdala. We report alteration of autistic conditions after lesionectomy in two patients with temporal lobe epilepsy and autistic disorder.
Case reports: A 13 year-old boy with Asperger syndrome had febrile seizure status at 11 months of age and complex partial seizures since 10 years. He had behavioral problems since 8 years. MR imaging showed right mesial temporal lobe sclerosis. He underwent amygdalo-hippocampectomy at 13 years, and showed dramatic improvement of the behavioral problems as well as seizure freedom. A 10 year-old girl with autism had secondarily generalized seizures since 2 months of age, and mental retardation and autistic tendency since 2 years. Neuroimaging studies showed cortical dysplasia in the left lateral temporal lobe. She underwent lateral temporal corticectomy at 4 years, and autistic symptoms exacerbated despite seizure freedom.
Conclusions: Laterality or lateral or mesial structures of the temporal lobe might involve autistic conditions.

AUTISTIC LIKE BEHAVIORAL DISORDERS AND EPILEPSY

POSTER 6 12th ISS

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Objectives: Autistic like behaviors are common in pediatric population and are one of the common referrals to pediatric psychiatry, psychology and neurology but this could be a feature of non convulsive epilepsy.
Methods & Materials: We have done EEG for 250 patients admitted to pediatric department. Among out patients in Neurology division, Imam Khomeini Hospital Tehran- Iran during 2006-2008 due to behavioral disorders (ADHD or autism diagnosed by psychiatrics), 100 patients had autistic behavior; 40 girls and 60 boys aged 4 and 8 yrs. We also have evaluated serum ferritin level, T4,TSH, and hearing and visual abilities.
Results: 95 of 100 patients had abnormal EEG (spike slow 3HZ, diffusely abnormal), 2 of patients had moderately sensorineural deafness, and 3 had visual acuity defects.
80% of 100 had serum ferritin below 15mg, 15% of them had serum ferritin between 15-25mg , 2 had TSH more than 10 IU.
Discussion: Epilepsy and iron deficiency syndrome have been suggested to be ruled in behavioral disorders in many published data and also this work can confirm them.
Conclusions: It is better to rule out organic disorders in behavioral problems.

POSTER 7 12th ISS

CLINICAL ANALYSIS OF CHILDREN WITH AUTISM SPECTRUM DISORDERS AND EPILEPSY

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Purpose: This study was conducted to evaluate the seizure types and epileptic syndromes prevalent in children with autism spectrum disorders (ASD).
Subjects and Methods: The subjects were 24 children with ASD followed up in both epilepsy and child neuropsychiatric clinics. Patients with tuberous sclerosis or a history of West syndrome were excluded from this study. The medical chart, EEG files and neuroimagings were retrospectively analyzed to classify seizure types as well as epileptic syndromes.
Results: ASD were classified into pervasive developmental disorders not specified (PDD NOS) in 17 children, Asperger's syndrome in 5 children and autism in 2 patients. They had complex partial seizures (n=12), (secondarily) generalized tonic-clonic seizures (n=17), myoclonic or astatic seizures (n=4) and simple partial seizures (n=1). The age at onset of epilepsy ranged from 3 months to 11 years of age (median = 2 years). The epilepsy onset was so early during infancy period that the onset of ASD symptoms was difficult to assess either before or after the seizures had become frequent in most subjects. The epileptic syndromic classification was made as follows: symptomatic focal epilepsy (n=12), benign focal epilepsy (BFE) in 6, Dravet syndrome spectrum in 3, and cryptogenic generalized epilepsy in 3.
Conclusions: This study revealed the presence of specific epileptic syndromes among children with ASD. Although the combination of ASD symptoms and the relatively resistant nature of seizures obscured the diagnosis of BFE syndromes, they comprised one fourth. As BFE predisposition appears to be prevalent, its coincidence with ASD would occur frequently.

POSTER 8 12th ISS

SEIZURE CHARACTERISTICS OF EPILEPSY PATIENTS WITH AUTISM SPECTRUM DISORDERS

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Objectives: We aimed to assess clinical characteristics of epilepsy patients with autism spectrum disorders (ASD).
Methods: Our series includes 31 patients, diagnosed with both ASD and epilepsy who were followed for more than one year at Osaka Medical College and its affiliates. Age distribution at epilepsy onset was bimodal, with one peak in early childhood and another in adolescence. We divided the patients by age; (1) onset age 5 years or less (younger group, n=14), (2) onset age 6 years or more (older group, n=17). Clinical characteristics were compared between the groups.
Results: As to sex ratios, there were more females than males in the younger group, more males than females in the older group. On electroencephalograms, epileptiform activity site tended to be in the frontal or anterior temporal area in the older group, while no specific localization was recognizable in the younger group. As for seizure type, neither partial seizures with secondarily generalization nor generalized seizures were seen in the older group, while the younger group had all types of seizures.
Conclusions: There are characteristic differences in clinical symptoms and sex ratio between younger and older epilepsy patients with ASD.

CLINICAL CHARACTERISTICS OF EPILEPSIES IN PATIENTS WITH AUTISM

POSTER 9 12th ISS

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Objectives: The aim of this study is to elucidate the clinical characteristics of epilepsies in patients with autism.
Methods: Subjects included were patients with both autism diagnosed based on DSM-**IV** and epilepsy followed by pediatric neurologists in our hospital. We retrospectively investigated epilepsy-onset age, seizure type, seizure frequency, electroencephalogram (EEG) findings and seizure prognosis.
Results: Sixty-seven subjects were included. The average epilepsy-onset age was 11± (0-21) years. Seizure types were partial in 54 patients, absence in 3, tonic in 2, myoclonic in 1, spasms in 1 and unclassified in 8. As for seizure frequency, 8 subjects had seizures on a daily basis, 5 weekly, 19 monthly and 28 yearly. Six subjects had less than one seizure in a year. The EEGs were recorded in 58 patients. The EEG findings were normal in 13 patients, focal paroxysmal discharges in 34 and diffuse paroxysmal discharges in 13. Seizures disappeared in 38 patients, whereas seizures were intractable in 29. There were no characteristic differences between patients who responded to anti-epileptic drugs and patients with intractable epilepsy.
Conclusions: Epilepsies in patients with autism were mainly childhood-onset, partial seizures, although clinical characteristics were various and heterogeneous.

CHARACTERIZATION OF THE EPILEPSY ASSOCIATED WITH AUTISM SPECTRUM DISORDERS

POSTER 10 12th ISS

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Objectives: Autism spectrum disorders (ASD) and epilepsy have a close relationship. To characterize epilepsy associated with ASD, we retrospectively studied the patients with epilepsy associated with ASD.
Subjects: The study included 61 patients with epilepsy and ASD without any causative disease such as tuberous sclerosis. Patients with ASD secondary to infantile spasms were excluded.
Results: There were 46 males and 15 females, aged 2 to 43 years (median 11 years). The onset of seizures was most frequent at 4 years, and 85% was occurred before 10. The most frequent type of seizure was complex partial seizure (CPS; 70%). Paroxysmal activities on EEG were localized in the frontal area in about half of the cases. Multiple anti-convulsants were used in 32.8% cases (two in 16.4%, three in 16.4%), and 45.9% of the patients were seizure-free for more than two years. An amelioration of the autistic symptoms occurred after epilepsy treatment in five cases (8%).
Conclusions: CPS with frontal paroxysms occurring from one to ten years of age seems to be characteristic of epilepsy associated with ASD.

POSTER 11
12th ISS

EPILEPSY IN CHILDREN WITH ASD AND OTHER NEURO-PSYCHIATRIC DISORDERS AT THE MENTAL HEALTH CLINIC IN BANGLADESH

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Objectives: Autism is a complex neurodevelopmental disorder and there is a variable association between autism and epilepsy. Recently the number of children with autism spectrum disorder (ASD) is about six fold increased at the child development and neurology unit (CDNU) of the National Children's Hospital in Bangladesh.
Objective of this paper was to look at the frequency and types of seizure disorders as comorbidity among the children with ASD and other pervasive mental disorders among the children attending the mental health clinic.
Methods: The medical records of the children attending the mental health clinic of the CDNU were studied retrospectively. Autism and ASDs were diagnosed by multiaxial technique according to DSM IV classification. A routine EEG was performed in a proportion of children suspected with seizure disorder.
Results: Total 491 children were diagnosed with ASD and other neuropsychiatric disorders. Among them 181 (36%) were diagnosed with ASD. Mean age at diagnosis was 4.4 years, and the male female ration was 2.6:1. About 40% among the total population and 15% in 181 children with ASD had history of definite unprovoked seizures before the first assessment at our clinic.
Conclusions: Diagnosis of epilepsy and its treatment may reduce the family burden and improve the communication skill in children with ASD. Appropriate history taking and rational use of the investigation tool such as EEG, particularly after sleep deprivation, is helpful to explore treatable cerebral dysfunction. In some selective cases EEG telemetry would be very helpful to differentiate from LKS. EEG may also help to classify the autism.

POSTER 12
12th ISS

BACKGROUND ACTIVITIES OF EEG IN THE CHILDREN WITH AUTISM SPECTRUM DIRORDERS

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Objectives: To investigate the relationship between the cognitive functions and electroencephalogram (EEG) findings, the background activities (BGA) of EEG are analyzed in the patients with autism spectrum disorders (ASD).
Methods: The subject was 59 cases (average 7.7±2.9 years), which were divided into two groups: the mental retardation positive (intelligence quotient≦70, following MR+) and the mental retardation negative (IQ>70, following MR-). All EEGs were recorded from awakening state to the natural sleep stage without any sleep inducing drugs. The frequency, amplitude and reactivity of the BGA and distribution of the paroxysmal discharges were examined.
Results: The case number of MR+group was 26 and MR-group was 33. Age distribution did not show the significant difference in two groups. Epilepsy was found in 5 cases (MR+3, MR-2). In MR+, mean frequency of BGA was 6.8±1.59Hz in infancy (2-6 years), 9.3±0.50Hz in school children (7-12 years) , and 10.1±0.38Hz in adolescence (above 13 years) . In MR-, it was 8.6±0.84Hz, 9.2±0.62Hz and 9.5±0.60Hz, respectively. Alpha attenuation was not recognized in 10 cases of MR+ (38.5%) and 4cases of MR- (12.1%). Paroxysmal discharges were found in 8 cases of MR+ (31.0%) and 5 cases of MR- (15.2%).
Conclusions: Our results showed that the mean frequency of BGA was significantly low in MR+infancy comparing with that of MR-infancy. The incidence of paroxysmal discharges with ASD children seems to be higher than that of general population.

SLEEP SPINDLES IN CHILDREN WITH PERVASIVE DEVELOPMENTAL DISORDERS

POSTER 13 12th ISS

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Objectives: In clinical observations, sleep disturbance in children with pervasive developmental disorders (PDD) have been often pointed out. We tried to clarify whether those children have some abnormalities or not, in sleep electrophysiological patterns.
Methods: We analyzed total 360 natural sleep electroencephalograms recorded in our institution (Tama-Ryoikuen) or in National Center for Child Health and Development. 240 were recorded from PDD children, 80 were from children with diagnosis of pure mental retardation (MR), and 40 were recorded from children with cerebral palsy (CP), from 2 to 20 years old. We specifically analyzed sleep spindles as to their shapes, localizations, and cycles.
Results: We found that there often exist distinct 10-12Hz frontal spindles with poor 14Hz central spindles in PDD children. On the contrary, we generally observed rather stable 14Hz central spindles in children with MR or CP. In PDD children, 14Hz central spindles become stable with ages from around 6-years-old.
Conclusions: Maturational process of sleep structure may be aberrant in PDD children.

EEG FINDINGS IN AUTISM SPECTRUM DISORDERS WITH EPILEPSY

POSTER 14 12th ISS

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Objectives: Epilepsy is quite common in autism spectrum disorders (ASD), and it is increasingly recognized as an additional clinical problem that must be dealt with. We investigated the epileptiform EEG abnormalities in children with ASD and incidence of later development of epilepsy.
Methods: In a prospective study, 15 children, ranging in age from 3 to 6 years, were enrolled and followed. EEG recordings and clinical evaluation were performed every 6 months for at least 6 years. Epileptiform abnormalities, including focal spikes, multifocal spikes, and generalized spike and wave complexes were each coded separately. We scored the occurrence of the localization of spikes and evaluated the relation with later development of epilepsy.
Results: Of the 15 subjects, 4 (26.7%) had later development of epilepsy. The focus of paroxysmal discharge with later development of epilepsy was frontal regions in 3 and multifocal in one. The focus in the patients with EEG abnormalities was frontal regions in 4, centrotemporal in 1, and multifocal in 1.
Conclusions: The incidence of later development of epilepsy in ASD may depend on the localization of epileptiform EEG abnormalities. The presence of frontal paroxysms may be a higher risk of epilepsy in ASD.

POSTER 15 12th ISS

INCIDENCE AND CHARACTERISTICS OF ELECTROCEPHALOGRAPHIC ABNORMALITIES AT INITIAL DIAGNOSIS IN CHILDREN WITH AUTISM SPECTRUM DISORDERS

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Objectives: The incidence of electroencephalography (EEG) abnormalities has been reported higher in children with autism spectrum disorder (ASD) than in normal children. The aims of this study are to know the incidence and characteristics of EEG abnormalities at initial diagnosis in children with ASD.
Methods: We retrospectively reviewed the medical records of children with ASD who diagnosed at Chonnam National University Hospital in from 1996 to 2003. Clinical characteristics, EEG, brain image and the duration of follow-up were reviewed.
Results: The incidence of abnormal EEG at initial diagnosis of autism was 25.8 % (25 out of 97 cases, epileptiform abnormalities in 12 (12.4%), non-epileptiform in 13 (13.4%). Epileptiform EEG abnormalities (spikes, spike and waves or sharps) were mainly located in frontal areas (7/12, 58.3%), followed by the temporal areas (2/15, 16.7%) or generalized (2/15, 16.7%). Most of non-epileptiform abnormality was focal slowing. 16 cases revealed as epileptics during observation periods, among which 7 who showed no abnormality at initial EEG showed the epileptiform EEG abnormalities during serial follow- up.
Conclusions: In this study, the incidence of EEG abnormalities in children with ASD seemed relatively high. Even though initial EEG was normal, in epileptics follow-up EEG showed abnormalities.

POSTER 16 12th ISS

ROLE OF EEG IN THE EVALUATION OF AUTISTIC DISORDERS

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Objectives: To evaluate the role of EEG in autistic children with/without clinical epilepsies.
Methods: A retrospective chart review of autistic children with an ICD diagnostic code of 299.00 or 299.01 was conducted from special outpatient clinics during the period of March 2007 to December 2008. The demography of the patients, EEG findings, and co-morbidities including epilepsies were reviewed.
Results: Totally 215 cases were enrolled. Male was 183 and female 32. The age ranged from 1y10m to 18 years (means: 6.9 years). Among them, 7 attributed to Asperger syndrome, 6 was considered as high function autism. There were 100 patients received EEG studies consecutively, which revealed negative findings in 68, focal paroxysmal discharges in 17, multifocal epileptiform discharges in 9, hypsarrthythmia in 1 and slow background activities in 5. The focal paroxysms originated from frontal or frontopolar regions in 12 patients (71%). Twenty-three patients (11%) developed clinical seizures and 10 (43%) of them were difficult-to-control ones.
Conclusions: EEG abnormality occurs in a certain number of autistic children. The frontal predominance of EEG paroxysms might implicate its major pathophysiological basis in autistic patients. Multifocal discharges have the clinical significance indicating the refractability of underlining seizure activities.

EVIDENCE OF GABAERGIC DYSFUNCTION IN AUTISTIC BRAIN: A PROTON MAGNETIC RESONANCE SPECTROSCOPY STUDY

POSTER 17 12th ISS

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Objectives: GABA is the major inhibitory neurotransmitter in the central nervous system and is integral to managing seizure activity. We performed in vivo 1H-MRS study to evaluate GABA concentration in autistic brain.
Methods: The subjects consisted of 32 individuals who met DSM-**IV** criteria for autistic disorder (2 to 14 years old). The control subjects were 21 children (2 to 14 years old). The study was performed on a clinical 3 Tesla MRI. The sequence parameters of a conventional STEAM method were following: TR=5000 ms, TE=15 ms. We analyzed the metabolites, N-acetylaspartate, Choline, Creatine, Glutamate using LCModel. MEGA-PRESS was created to evaluate GABA. The sequence parameter was following: TR=1500 ms, TE=68 ms. The concentration of GABA was also quantified using LCModel. We placed a single volume of interest in the anterior cingulate gyrus.
Results: The concentration of GABA in the anterior cingulate gyrus of autism was significantly decreased compared with controls (autism; 0.55 ± 0.21 mM, controls; 0.73 ± 0.31 mM, $p<0.05$). There was no significant difference between the concentration of other metabolites in autism and control group.
Conclusions: GABAergic dysfunction may be involved in seizure susceptibility in autism.

TWO CASES OF ASPERGER SYNDROME THAT WERE DIAGNOSED AT 2 YEARS OF AGE

POSTER 18 12th ISS

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Introduction: Asperger syndrome (AS) is not accompanied by any obvious delay in language development, so it may often missed in infancy. We report 2 cases of AS that were diagnosed at early phase.
Case 1: She began refusing a nursing bottle from 6 months, did not eat baby food thereafter. She sat at 10 months, started walking at 20 months. She began speaking single words at 14 months, two-word sentences at 24 months. She began sleeping in the morning and arising at noon from 18 months. She did not play with friends. At 24 months, was diagnosed AS under DSM-IV TR.
Case 2: From infancy, he was extremely picky and only ate same food. He began walking at 24 months but was obsessive strollers. Before 12 months, the cycle of sleeping in the morning and awakening in the evening had become established. He began speaking two-word sentences at 24 months, so diagnosed AS with DSM-IV TR.
Results: In these 2 cases, we observed 5 typical findings.
1. Deficient flexibility in eating habits and compulsive obsession with favorite foods
2. Disorder in mutual social relationships and interpersonal relationships
3. Normal language development
4. Disorder in sleeping cycle
5. Delay in motion development after 6 months of age

Conclusions: No diagnostic criteria have yet been established for infantile AS. Our findings may important key for early diagnosis of infancy onset AS.

POSTER 19 12th ISS

EPILEPTIC VISUAL AURA

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Objectives: The patients with refractory epilepsy have many types of auras. Auras may be classified by symptom type: the visual types, special sensory, autonomic, or psychic symptoms. The most important are the visual auras which occur in the great part of temporal lobe and precede about 50% of epilepsy seizures.
Methods: A patient aged 28 years, with secondary generalized temporal epilepsy, has been stimulated with different visual stimuli, high intensity, especially: stroboscopic light, white light, brightness light, approximately during 10 seconds.
Results: After 10 seconds come into sight visual auras, time is 5 seconds. In this time vision was concentrated in the area occupied by the visual aura. Immediately starting the epilepsy temporal seizure tonic-clonic time is approximately 1- 3 minutes.
Conclusions: My investigation aims to show you inside visual aura and how it is, what colours appear. For each patient with visual auras, the visual stimulus represents the first and the most excitatory stimuli for the patient. Another stimulus: physical and psychical effort, tiredness, caffeine, stress, drugs, they are less important, with effect minutes later just hours face to the visual stimuli.

POSTER 20 12th ISS

AUTISM RELATED 593-KB MICRODELETION OF 16P11.2 IN A MOTHER AND SON WITHOUT AUTISM BUT MR

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Objectives: Several large-scale screening studies for autism spectrum disorders have identified a common 593-kb interstitial deletion of 16p11.2 flanked by 147-kb segmental duplications as one of the most common genomic disorders associated with autism. Here we report the second case of the familial 16p11.2 deletion in a 3-year 2-month-old boy with developmental delay, hyperactive but not autistic behavior, and dysmorphic features, including small stature, curly brown hair, rib anomalies and inguinal hernia.
Methods: Oligoarray comparative genomic hybridization analysis was performed according to the manufacture's protocol under the permission of ethical committee of the institution.
Results: A common 16p11.2 microdeletion was identified in the patient and his mother with suspected borderline mental retardation.
Conclusions: Deletion of 16p11.2 is the only chromosomal aberration common among the patients with autism. It is widely identified from all over the world with the incidence of 1% in autistic patients. The complicated phenotypes associated with the presenting patient might be a consequent of unmasking of a hemiallelic mutation on the homologous allele by the deletion of 16p11.2, or might be coincidental. More information is needed to establish the clinical characters of 16p11.2 microdeletion syndrome.

A FAMILIAL CASE OF LEOPARD SYNDROME ASSOCIATED WITH HIGH-FUNCTIONING AUTISM SPECTRUM DISORDER

POSTER 21 12th ISS

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Objectives: LEOPARD syndrome is an autosomal dominant disorder and shares many clinical features with Noonan syndrome and NF type I. Common molecular pathology is believed to be involved in these conditions. Association of autism spectrum disorder (ASD) in LEOPARD syndrome was studied.
Methods: Neuropsychological evaluation by the high-functioning Autism Spectrum Screening Questionnaire (ASSQ) and DSM IV were performed in a family with four individuals with LEOPARD syndrome (heterozygous for Thr468Met in PTPN11.
Results: The second and the third son were diagnosed with high-functioning autism spectrum disorder (HFASD), and the father had a borderline-score on the ASSQ. However, the first son marked a low score on the ASSQ.
Discussion: To our knowledge, this is the first report to demonstrate the association of LEOPARD syndrome and HFASD. LEOPARD syndrome is primarily due to mutations in PTPN11 resulting in interfering with RAS/ERK2/MAPK pathway. It is unclear how this pathway is involved in the CNS functions causing HFASD. Further studies will be needed to clarify the association of LEOPARD syndrome and HFASD.

SODIUM CHANNELS OF SCN1A GENE MUTATIONS IN GENERALIZED EPILEPSY WITH FEBRILE SEIZURE PLUS (GEFS+) SPECTRUM RELATED TO AUTISM

POSTER 22 12th ISS

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Objectives: To identify the mutations of SCN1A gene in patients with GEFS+ spectrum related to autism.
Methods: We examined four patients with autism and GEFS+ spectrum who were admitted to the Department of Pediatrics, Dr Sardjito hospital, Yogyakarta, Indonesia. Diagnosis of autism was based on DSM-IV criteria. Mutations in SCN1A were identified by PCR amplification and denaturing high-performance liquid chromatography analysis, with subsequent sequencing.
Results: There were 4 patients, all boys, aged 1.8 year to 7 years. The phenotypes of epilepsy were GEFS+ in 1 patient, SMEB in 1 patient and SMEI in 2 patients. Sequencing analysis revealed a G-to-A heterozygous transition which was detected at nucleotide c.4834G>A (p.V1612I) in exon 25. Other single nucleotid polymorphisms (SNP) were c.392+52T>C in intron 2 and c.1028+21T>C in intron 7.
Conclusions: In this study, we reported the first cases with mutation in SCN1A gen in GEFS+ spectrum related to autistic patients in Indonesian population, which showed a novel missense mutation p.V1612I

POSTER 23
12th ISS

CDKL5 MUTATIONS IN PATIENTS WITH EARLY-ONSET INTRACTABLE EPILEPSY AND AUTISTIC BEHAVIOR

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Objectives: Mutations in X-linked cyclin-dependent kinase-like 5 gene (CDKL5) are responsible for a severe encephalopathy with X-linked early-onset seizures and atypical Rett syndrome. Until now, many CDKL5 gene mutations have been reported to date. To reveal underlying etiology for severe infantile epilepsies, we analyzed CDKL5 mutations.
Methods: At least 50 patients with different types of cryptogenic infantile epileptic encephalopathy were examined by array-CGH to screen genomic copy number aberrations. The samples, which did not show any genomic copy number aberrations, were conducted to analyze CDKL5 mutations by using PCR and direct sequencing.
Results: Array-CGH identified a loss of genomic copy number of CDKL5 in a male patient whoseintractable epilepsies started when he was 2-month old. He showed extremely severe developmental delay. Two new CDKL5 mutations were identified in 2 unrelated female patients. One patient showed profound psychomotor delay. Her epileptic seizures started from 4 days after delivery.
The other presented atypical Rett features and early on-set seizures which started at 2-month old.
Conclusions: We identified three genomic mutations of CDKL5 in patients with early onset epilepsy and severe psychomotor developmental delay in both male and females. The female patient with a missense mutation showed severer phenotype compared to the patient with nonsense mutation, who showed atypical Rett phenotype.

POSTER 24
12th ISS

EFFECTS OF BETA-HYDROXYBUTYRATE ON NEUROGENESIS AFTER PILOCARPINE-INDUCED SEIZURES IN YOUNG MICE

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Objectives: Ketogenic diet (KD) was originally devised to mimic biochemical changes seen upon fasting, specifically the formation of ketone bodies: beta-hydroxybutyrate (BHB), acetoactate, and acetone. Recent data suggest that the anticonvulsant efficacy of KD may be due in part to direct actions of ketone bodies. This study was designed to investigate effects of BHB on neurogenesis after seizures in mice.
Methods: Young (P21) mice were used. Experimental mice (n = 5) were injected intraperitoneally with BHB (20 mmol/kg), while control mice (n = 6) with normal saline. Fifteen minutes later, seizures were induced by pilocarpine (300 mg/kg. i.p.) in both groups. Then, bromodeoxyuridine (BrdU, 50 mg/kg) was subsequently administered once a day for 6 consecutive days, starting at 24 hours after pilocarpine injection. Thereafter, BrdU-positive cells in the hippocampus were counted.
Results: In BHB-treated mice, BrdU-positive cells of the hippocampal dentate granule cell layer increased significantly compared to control mice (377.57 ± 150.40 vs. 230.55 ± 59.50, $p<0.001$).
Conclusions: In this study, we found a significant increase in the proliferation rate of neuronal progenitor cells after pilocarpine-induced seizures in BHB-treated mice. These results suggest that BHB enhances neurogenesis after seizures.

CHARACTERISTICS OF INTELLIGENCE & LANGUAGE ABILITY IN CHILDREN WITH EPILEPSY

POSTER 25 12th ISS

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Objectives: Epileptic patients have sometimes suffered from mental retardation or regression because of back ground pathology and side effects of the treatment as well as seizure discharges per se. We analyzed intelligence and language ability in epileptic children who had showed normal development at the diagnosis of epilepsy.
Materials: Subjects are twenty seven patients aged 6 to 16 years, 13 boys and 9 girls, with idiopathic partial epilepsy and cryptogenic partial epilepsy. All the subjects were considered to have developed normally by careful examination and history taking by the authors at the diagnosis of epilepsy.
Methods: For intelligence and language evaluation, Wechsler intelligence scale for children (WISC III) and Illinois test of psycholinguistic abilities (ITPA) were tested. EEG, MRI, SPECT with HM-PAO and IMZ were performed for localization of seizure foci.
Results: IQ ranged from 48 to 104; 12 below 69, and 15 patients above 80. In 6 patients, Performance IQ was significantly higher than Verbal IQ, and Verbal Comprehension Index was relatively higher than Perceptual Reasoning Index. One girl was diagnosed as having Pervasive Developmental Disorder (PDD), one male patient as Attention-Deficit Hyperactivity Disorder (ADHD). In cases with electrical status epileptics during sleep (ESES), IQ profiles depend on discharge foci.
Conclusions: Mental retardation and/or PDD were observed in the epileptic children who had normal development at the diagnosis. The developmental profiles differed among patients, but these seemed partly related to the epileptic foci. It is essential to check IQ tests in the management of epilepsy.

A STUDY ON BEHAVIORAL PROBLEMS IN CHILDREN WITH EPILEPSY

POSTER 26 12th ISS

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Purpose: We investigated behavioral problems in children with epilepsy using the Child Behavior Checklist (CBCL).
Methods: The subjects were 139 epileptic patients 6-18 years of age who could speak and walk by themselves. Localization-related epilepsy (LRE) was diagnosed in 87 patients, generalized epilepsy (GE) in 38, and undetermined focal or generalized epilepsies in 14. LRE and GE were sub-classified according to etiology as idiopathic, cryptogenic or symptomatic. In evaluation using the CBCL, Superordinate scales (Internalizing and Externalizing scores) and Total score were assumed to be abnormal when their T score was≧60.
Results: The abnormality rates of Internalizing, Externalizing and Total scores of CBCL were 25.9%, 27.3% and 36.7%, respectively, in all patients, and varied depending on diagnosis. Regarding the children with LRE, the abnormality rates were lowest in idiopathic cases, higher in cryptogenic cases, and highest in symptomatic cases. By contrast, in the patients with GE, the abnormality rates were largely the same irrespective of etiology.
Conclusions: The CBCL was demonstrated to be effective for evaluation of behavioral problems in children with epilepsy.

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CASE REPORT: STEREOTYPIC SELF-HITTING IN TWO PATIENTS WITH SYMPTOMATIC LOCALIZATION-RELATED EPILEPSY AND MENTAL RETARDATION

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Self-injurious behaviors (SIBs) are multiple and diverse in their presentation and often bewildering in the pathology that driven them, their blatant self-destructiveness, and the call for intervention and basic pathogenesis.
Now, we present two patients with symptomatic epilepsy, mental retardation and stereotypic SIBs, face hitting, and consider the basic pathogenesis and efficacious intervention.
Case I. The 18 years old male patient has been given VP shunt for Arnold-Chiari type hydrocephalus since neonate, and nasal desmopressin for diabetes insipidus since 12 years old. He has been also suffered from symptomatic fronto-temporal lobes epilepsy; versive sz. with 2 ° GTS and its cluster formations, and CPS with/without 2 ° GTS. Around the onset of DI and epilepsy, since 12 years old, he has shown the stereotypic self face-hitting by his lt-hand during day and night times, except sleeping. EEG showed Sp, Sp & W complex on rt-F, -C & mT, and lt-F & -C. MRI revealed the dilatation of lt-anterior horn and colpocephaly.
Case 2. The 25 years old female patient was suffered from purulent meningitis on 9 months old. After this episode, she has been treated VP shunt for the hydrocephalus and symptomatic fronto - temporal lobes epilepsy. Her seizures were CPS with/without 2 ° GTS, which were still noticed about once a month. She has shown the stereotypic self-face hitting by her rt-hand since around 7 years old. EEG shows Sp, Sp & W complex on lt-F, Fp,- aT, and lt-P &-O, independently. MRI shows diffuse ventricular dilatation with rt-dominancy.
Discussion : In spite of the different etiology, these two patients have shown the similar clinical symptoms, such as prolonged, refractory stereotypic SIBs, self-face hitting, fronto-temporal lobes seizures, ventricular dilatation-VP shunt and severe mental retardation.
SIBs are classified to four categories of stereotypic, major, compulsive and impulsive (Favazza and Simeon 1995), which are based phenomenologically and largely disregarded basic etiology. Stereotypic SIBs are common in patients with mental retardation. Various specific syndrome, such as Lesch-Nyhan syndrome, Prader-Willi syndrome and autism may also exhibit. These two patients could not be accounted for pervasive developmental disorders, such as autism or obsessive-compulsive disorder under the criteria of DSM-IV-TR. The SIBs may be not related to their epilepsy and its seizure frequency, but may suggest the involved lesions. The SIBs are among the troubling manifestations, requiring behavioral, pharmacological managements, and etiological pathogenetic evaluations

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EATING PROBLEM IN A 31-MONTH-OLD GIRL WITH AUTISM SPECTRUM DISORDER

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[2)] Yokoyama Pediatric Clinic, Kasuga, Fukuoka, Japan
[3)] Department of Pediatrics, Faculty of Medicine, Fukuoka University, Fukuoka, Japan

Objectives: We present the case of a 31-month-old girl with an eating problem. She was admitted to our hospital for dehydration and 12% weight loss. She stopped eating when a dog at her relative's house interrupted her meal. She had eaten nothing except her mother's breast milk for 17 days.
Methods: We administered a drip infusion for seven days, and performed chest and abdominal X-rays, head magnetic resonance imaging and electroencephalogram, but found no organic abnormality. Metabolic disorders were excluded. Seven days after admission, she started drinking, so we stopped infusion. Eight days after admission, she suddenly started eating and recovered in a couple of days.
Results: She has mild speech delay. She has autistic features, such as hypersensitivity to touch, she cannot walk on sand without shoes, and she has been wearing the same shirt for a month. We therefore diagnosed her with autism spectrum disorder.
Conclusions: Early diagnosis of autism spectrum disorder is important for early training. Eating problems are one of the cardinal manifestations of autism spectrum disorder. We should be aware of the eating habits of infants for the early diagnosis of autism spectrum disorder.

THE RELATIONSHIP BETWEEN SLEEP DISORDER AND SEIZURE DISORDER IN ANGELMAN SYNDROME: A CASE REPORT.

POSTER 29 12th ISS

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Objectives: Angelman syndrome (AS) is a neurodevelopmental disorder caused by various abnormalities of maternally inherited chromosome 15q11-q13. AS had deficit of the gammaaminobutyric acid type A ($GABA_A$) receptor that is related to neurodevelopmental impairment, sleep disorder, and seizure disorder. Although GABA is the main inhibitory transmitter in the adult brain, $GABA_A$-receptor-mediated responses are depolarizing in early developmental period. During maturing the brain, these receptors shift from depolarization to hyperpolarization.We hypothesized that in the period of the shift to hyperpolarization, deficit of $GABA_A$ receptor may block the GABAergic pathway, and then sleep disorder and seizure disorder occur.
Methods: Subject is a boy with AS who was referred to our hospital at 11 months old. We asked his family to record the sleep diary log and to count seizure frequency.
Results: His seizure onset was at 1.5 years old. Although he had regular sleep cycle before seizure onset, his sleep was disturbed after seizure onset. The sleep diary log demonstrated reduction of total sleep time, sleep period time at night, and aggravation of sleep efficiency. Clobazam, that regulates chloride channels and enhances the GABAergic pathway, was effective in both disorders.
Conclusions: We surmise that the functional deficit of GABAA receptor in the period of the shift from depolarization to hyperpolarization may be related to the co-occurrence of sleep disorder and seizures in this patient.

THE CORRELATION BETWEEN ^{1}H-MR SPECTROSCOPY (^{1}H-MRS) AND CLINICAL MANIFESTATION WITH TUBEROUS SCLEROSIS COMPLEX (TSC)

POSTER 30 12th ISS

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[2)] Department of Pediatrics, National Epilepsy Center, Shizuoka Institute of Epilepsy and Neurological Disorders, Shizuoka, Japan

Objectives: To examine the correlation between the severity of clinical manifestation and neuro-radiological features of TSC
Methods: We examined 7 patients aged 2 to 15 years (mean 6.3 years) old with TSC and 6 normal controls aged 2 to 20 years (mean 10 years). Clinical features including developmental quotient (DQ) were examined in TSC patients. The number of hypomelanotic maculae and that of cortical tubers in MRI with TSC patients were calculated. The ^{1}H-MRS was performed in patients and controls, and the data were statistical analyzed using the Mann-Whitney U-test.
Results: The numbers of hypomelanotic maculae and that of cortical tubers were correlated with the clinical severance of TSC. The patients with severe mental retardation and intractable epilepsy had greater numbers of these lesions. Both decreased ratio of N-acetylaspartate (NAA)/creatine (Cr) and increased that of myoinositol (mI)/Cr in tubers were statistical significant compared with those in normal control subjects ($P<0.05$).
Conclusions: Decreased NAA/Cr in brain ^{1}H-MRS is closely associated with the clinical severity of TSC.

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MR SPECTROSCOPY ANALYSES OF THE TWO CASES WITH SYMPTOMATIC WEST SYNDROME

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[1)] Department of Pediatrics, Gifu Prefectural General Medical Center, Gifu, Japan
[2)] Shizuoka Institute of Epilepsy and Neurological Disorders, Shizuoka, Japan
[3)] Department of Pediatrics, Kizawa Hospital, Gifu, Japan

Case presentation: Case 1: Spasms appeared 1 year and 7 months old. He was diagnosed as West syndrome. Development delay started after seizure had appeared. ACTH therapy was not remarkably effective, but addition of nitrazepam resulted in complete remission. Cortical dysplasia was implied by brain MRI. In MR spectroscopy, decreased NAA/Cr and increased Cho/Cr were indicated, in the region.
Case 2: He had myoclonic seizures and tonic-clonic convulsions since 2 months old. He had developmental delay. Brain MRI showed lissencephaly and there had chromosomal abnormality, i.e., ish, del(17p13.3p13.3)(LIS1-). He was considered isolated lissencephaly sequence with no remarkable malformation. Infantile spasms came out 4 months old, so ACTH therapy was started and was significantly effective. Nitrazepam was added on after ACTH therapy. In SPECT, left hemisphere had hypoperfusion. MR spectroscopy besides showed decreased NAA/Cr and increased Cho/Cr especially in left hemisphere.
Conclusions: We speculated that in symptomatic West syndrome MR spectroscopy may detect lesions possibly related to epileptic activities.

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12th ISS

FDG-PET AFTER INITIAL TREATMENTS PREDICTS 10-YEAR DEVELOPMENTAL OUTCOME IN CRYPTOGENIC WEST SYNDROME

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Objectives: To examine the relation between cortical hypometabolism on FDG-PET and long-term outcome in cryptogenic West syndrome.
Methods: In 1991-1997, we prospectively performed FDG-PET studies in 20 patients with cryptogenic West syndrome. PET was performed first at the onset and second after initial treatments at 10 months of age or more than 1 month after the ACTH therapy. We evaluated developmental and seizure outcome in 17 patients at 10-17 years of age. Correlation between first or second PET findings and long-term outcome was examined.
Results: At the onset, PET showed cortical hypometabolism in 11/17 patients. The second PET revealed cortical hypometabolism in 5/17 patients. Temporal lobes were the most frequent hypometabolic areas. At 10-17 years of age, 4/17 patients had persisting or recurrent partial seizures and 8/17 patients had mental retardation. In 12 patients with normal PET findings on the second scans, 10 were free of seizures without AEDs and 9 showed normal psychomotor development. In 5 patients with cortical hypometabolism on the second PET, 2 had seizures and all 5 had mental retardation.
Conclusions: The cortical hypometabolism changes with clinical symptoms. Persistent hypometabolism predicts poor long-term developmental outcome.

LONG-TERM FOLLOW-UP CASES OF INFANTILE SPASMS AFTER LESIONECTOMY

POSTER 33 12th ISS

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[3)] Department of Pediatrics, Kangbuk Samsung Hospital, School of Medicine Sungkyunkwan University, Seoul, Korea

Objectives: Some infants in the setting of a focal epileptogenic lesions manifest with infantile spasms, one of the catastrophic epilepsies. Early epilepsy surgery in catastrophic epilepsies has been emphasized for its positive effect on developmental outcome. But the long-term outcome is still vague. We present here long-term outcomes of lesionectomy cases of localization-related infantile spasms.
Cases: A 11 months old girl presented with asymmetric right side tonic spasms and her brain MRI revealed left temporal cystic lesions. Multiple antiepileptic drugs failed to cease her spasms, left temporal lobectomy was done at his age of 17 months and the biopsy revealed low grade astrocytoma. After surgery, she has been seizure free for 9 years of follow-up and has done well as a primary school student.
Second case was a two month old girl with asymmetric right side tonic spasms who showed left frontal cortical dysplasia on brain MRI. Her seizures were refractory to medical treatment and left frontal corticectomy was done at her age of 29 months. She has been seizure free for 7 years of follow-up after surgery with borderline IQ.
Conclusions: In symptomatic infantile spasms with focal brain lesion, the decision of surgical treatment should not be delayed for their neurodevelopmental outcomes.

EPILEPSY IN MECP2 DUPLICATION SYNDROME

POSTER 34 12th ISS

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[1)] Osaka Medical Center for Maternal and Child Health, Izumi, Japan
[2)] National Center of Neurology and Psychiatry, Kodaira, Japan
[3)] Tokyo Medical and Dental University, Tokyo, Japan

Objectives: Pathogenic mutations of the MECP2 gene are detected in 90-95% of female patients with Rett syndrome. We report two male siblings with MECP2 duplication detected by MCG X-tiling array.
Case Reports: A 19-year-old male (case 1) and his younger brother (case 2, 18 years old) with MECP2 duplication, demonstrated hypotonia in infancy, severe mental retardation, autism, recurrent respiratory infections, and dysmorphic face. The first seizure in case one showed eye fixation and loss of consciousness, evolving to sGTC at 17 years of age. EEG demonstrated frequent bilateral frontal or diffuse spike-wave complexes. During the course, CPS was replaced by AED-resistant drop attack. Case 2 developed seizures with eye deviation and tonic movements at 3 years of age. EEG showed repeated high voltage slow waves in the bilateral centro-parietal regions. Seizures were easily controlled with CBZ. MECP2 duplication was also identified in their healthy mother.
Discussion: Although these brothers shared some clinical features, epilepsy phenotypes were different. The lack of clinical symptoms in the mother suggests X chromosome inactivation.

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BRAIN WAVE RESULTS IN CHILDREN WITH ATTENTION DEFICIT HYPERACTIVE DISORDER AND TREATMENT RESULT WITH CENTRAL NERVOUS SYSTEM STIMULANTS

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[3] Department of Pediatrics, Chungnam University, Daejeon, Korea

Objectives: Attention deficit hyperactivity disorder (ADHD) is a syndrome which has occasionally seizures. This study examined the relationships among electroencephalographic (EEG) findings, stimulant use, and seizure occurrence in children with ADHD.
Methods: We retrospectively studied 308 children who visited our hospital because of ADHD since January 2001 to December 2005. We retrospectively analyzed age distribution, etiology, abnormalities of EEGs, and the use of CNS stimulants. Among those children, EEGs was recorded in 84 patients.
Results: 84 children (72 males, 85.7%, 9.3 years of mean age; 12 females, 14.3%, 8.0 years of mean age) with ADHD had EEGs performed in our institute. 19 patients (22.6%) demonstrated abnormalities, and 65 (77.4%) demonstrated normal EEGs. Stimulant therapy was applied to 59 of 84 patients (70.2%). Seizures occurred in 1 of 65 patients with a normal EEG (1.5%), and 3 of 19 treated patients with abnormal EEGs (15.7%).
Conclusions: These data suggest that patients with normal EEG have minor risk for seizure. In contrast, patients with abnormal EEG have higher risk for seizure than patients with normal EEG.

Profiles of Lecturers

Muneaki MATSUO

1
12th ISS

Present Position

IInstructor
Department of Pediatrics
Saga University, Faculty of Medicine, **Japan**

Appointments

1985-1986: Resident, Department of Pediatrics, Saga Medical School Hospital
1986-1987: Resident, Department of Pediatrics, Kagoshima City Hospital
1987-1988: Physician, Department of Pediatrics, Tara Town Hospital
1988-1989: Physician, Department of Pediatrics, Ohita Red-Cross Hospital
1989-1990: Clinical fellow, Department of Pediatrics, Saga Medical School
1990-1991: Physician, Department of Pediatrics, Saga Prefectural Hospital
1992-1994: Clinical fellow, Department of Pediatrics, Saga Medical School
1994-1996: Post-doctoral fellow, Department of Medicinal Chemistry, University of Kentucky
1997-2005: Clinical fellow, Department of Pediatrics, Saga University
2005- present position

Selected publications

1. Matsuo M, Sasaki K, Ichimaru T, Nakazato S, Hamasaki Y. Increased IL-1beta production from dsRNA-stimulated leukocytes in febrile seizures. Pediatr Neurol 2006;35:102-6.
2. Matsuo M, Tasaki R, Kodama H, Hamasaki Y. Screening for Menkes disease using the urine HVA/VMA ratio. J Inherit Metab Dis 2005;28:89-93.
3. Matsuo M, Tsuchiya K, Hamasaki Y, Snger HS. Restless legs syndrome: association with streptococcal and/or Mycoplasma infection. Pediatr Neurol 2004;31:119-21.
4. Matsuo M, Yoshida N, Zaitsu M, Ishii K, Hamasaki Y. Inhibition of human glioma cell growth by a PHS-2 inhibitor, NS398, and a prostaglandin E receptor subtype EP1-selective antagonist, SC51089. J Neuro-Oncol 2004;66:285-92.
5. Matsuo M, Hamasaki,Y., Masuyama,T., et al. Leukotriene B4 and C4 in cerebrospinal fluid from children with meningitis and febrile seizures. Pediatr Neurol 1996;14:121-4.

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Toru KUROKAWA

Present position

Director Emeritus
Department of Neurology (Child Neurology)
Seiai Rehabilitation Hospital, Japan

Appointments

1967-1970： Associate assistant, Department of Pediatrics, Kyushu University. Japan
1970-1972： Fellow, Seizure Unit, Boston Children's Hospital, Harvard Medical School
1984-1987： Associate Professor, Department of Pediatrics, Kyushu University, Japan
1987-1990： Professor, Jouetsu University of Education, Japan
1990-1992： Chief, Department of Child Neurology, National Center of Neurology and Psychiatry, Tokyo
1992-2001： Director, National Nishibeppu Hospital, Japan
(2002-Present Director Emeritus)
2002-2008： Director, Seiai Rehabilitation Hospital, Japan
(2008-Preesnt Director Emeritus)

Selected publications

1. KurokawaT, Mitsudome A, Yokota K, Goya N. Epilepsy of children with centro-midtemporal foci. Acta Pediatr Jap 1975;17:30-6.
2. Kurokawa T, Yokota K, Mitsudome A, Takeshita K, Shibata R, Goya N. Diagnostic and prognostic value of electroencephalography with intravenous diazepam in epilepsy. Folia Psychiatrica Neurol Jap 1979;33:15-20.
3. Kurokawa T, Goya N, Fukuyama Y, Suzuki M, Seki T, Ohtahara S. West syndrome and Lennox-Gastaut syndrome; A survey of natural history. Pediatrics 1980;65:81-8.
4. Kurokawa T, Tomita S, Ueda K, Narazaki O, Hara T, et al. Prognosis of occlusive disease of the circle of Willis (Moyamoya disease) in children. Pediatr Neurol 1985;1:274-7.
5. Kurokawa T, Yokomizo Y, Kimura N. Etiology of developmental disorders: Prenatal factors. The 50th Annual Meeting of Japanese Society of Child Neurology, May 31, 2008, Tokyo.

Evald SAEMUNDSEN

Present Position

Director of Services, Division of Autism and
Communication Disorders,
State Diagnostic and Counseling Center,
affiliated with the University of Iceland

Education

1978: Maitrice de Psych (MA), Universit? de Provence (Aix-Marseille I) France
1991: Doctoraal (MS) in Developmental Neuropsychology, Vrije Universiteit Amsterdam, Holland
1993: Accreditation as Specialist in Disability and Handicap by the Ministry of Health
2008: Ph.D. in Bio-Medical Sciences, University of Iceland

Appointments

1979-2000: Lecturer in Developmental and Clinical Child Psychology, University of Iceland
1983-1997: Clinical Child Psychologist at the State Diagnostic and Counseling Center
1983-1995: Consulting Psychologist at the St. Joseph's Hospital, Reykjavik, Dept. of Pediatrics
1995-1998: Consulting Psychologist at the City Hospital, Reykjavik, Dept. of Pediatrics
1997: Present position
2001- Research Associate of the Icelandic Autism Project, A Genetic Study of Autism
2009- Member of the Icelandic Team in the PsychCNVs, An EU Funded Project on Copy Number Variations Conferring Risk of Psychiatric Disorders in Children

Selected publications

1. Saemundsen E, Ludvigsson P, Rafnsson V. Risk of autism after infantile spasms - A population based study nested in a cohort with seizures in the first year of life. Epilepsia 2008;49:1865-70.
2. Saemundsen E, Ludvigsson P, Hilmarsdottir I, Rafnsson V. Autism spectrum disorders in children with seizures in the first year of life - A population based study. Epilepsia 2007;48:1724-30.
3. Saemundsen E, Ludvigsson P, Rafnsson V. Autism spectrum disorders in children with a history of infantile spasms - A population based study. J Child Neurol 2007;22:1102-7.
4. Magnusson P, Saemundsen E. Prevalence of Autism in Iceland. J Aut Dev Disord 2001; 31: 153-63.

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12th ISS

Virginia Chun-Nei, WONG

Present Position

Division of Child Neurology/Developmental Paediatrics/NeuroHabilitation
Department of Paediatrics & Adolescent Medicine,
the University of Hong Kong, Hong Kong

Degree:

1979 MBBS (Distinction in Paediatrics), The University of Hong Kong

Professional Qualification:

1984 MRCP (UK) (Best Candidate)
1984 DCH (Glasgow)
1985 DCH (London)
1993 FRCP (Edinburgh)
1993 FHKAM (Paediatrics)
1993 FHKCPaed
1997 FRCPCH
2000 FRCP (London)

Positions Held:

1980 - 1991 Lecturer
1991 - 1996 Senior Lecturer
1996 - now Professor, Division of Child Neurology/Developmental Paediatrics/Neuroabilitation Department of Paediatrics and Adolescent Medicine
Li Ka Shing Faculty of Medicine The University of Hong Kong

Honorary Appointments

1985 - now Unit Head, Division of Child Neurology, Developmental Paediatrics & Neurorehabilitation, Department of Paediatrics & Adolescent Medicine
The Duchess of Kent Children's Hospital at Sandy Bay
1999 - now Guest Professor (Beijing Medical University, China)
1998 - now Honorary Consultant (Children's Habilitation Institute (CHI)
The Duchess of Kent Children's Hospital at Sandy Bay
1993 - now Honorary Consultant Paediatrician (Department of Paediatrics & Adolescent Medicine, Queen Mary Hospital, Hospital Authority)
1993 - now Honorary Consultant Paediatrician (Child Development Centre (CDC)
The Duchess of Kent Children's Hospital at Sandy Bay)
1985 - now Honorary Consultant Paediatrician (Child Assessment Centre (CAC)
The Duchess of Kent Children's Hospital at Sandy Bay
2009 - Guest Professor (Fudan University, China)

Selected publications

1. Wong VC. Epilepsy in children with Autistic Spectrum Disorder. J Child Neurol 1993;8:316-22.
2. Wong VC, Hui LH, Lee WC, Leung LS, Ho PK, et al. A Modified Screening Tool for Autism (Checklist for Autism in Toddlers - [CHAT-23]) for Chinese Children. Pediatrics 2004;114:e166-76.
3. Feuk L, Kalervo A, LiPsanen-Nyman M, Skaug J, Nakabayashi K, Finucane B, et al. Absence of a paternally inherited FOXP2 gene in developmental verbal dyspraxia. Am J Hum Genet 2006;79:965-72.
4. Wong VC, Li SY. Rett syndrome: prevalence among Chinese and a comparison of MECP2 mutations of classic Rett syndrome with other neurodevelopmental disorders. J Child Neurol 2008;22:1397-400.
5. Wong VC, Hui SL. Epidemiological study of autism spectrum disorder in China. J Child Neurol 2008;23:67-72.

Roberto TUCHMAN

5
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Present Position

2001-present Director, Autism Program Miami Children's Hospital
2004-present Associate Professor of Neurology, University of Miami Miller School of Medicine at Miami Children's Hospital
2008-present Consulting Medical Director Center for Autism and Related Disorders University of Miami, Fl

Professional Experience and Academic Appointments

1984- 1986: Assistant Professor of Clinical Pediatrics Columbia University, New York
1984- 1986: Assistant Attending Pediatrician Presbyterian Hospital, New York
1986- 1989: Resident and Fellow Department of Neurology Albert Einstein School of Medicine, NY
1989- 1990: Epilepsy Fellow Department of Neurology Albert Einstein School of Medicine, NY
1990- 1998: Co-Director, Developmental and Behavioral Neurology Miami Children's Hospital, Florida
1991- 1995: Co-Director, Broward Division of the Comprehensive Epilepsy Center, Miami Children's Hospital Miami Children's Hospital, Florida
1991- 1998: Director, NETT Program (Neurobehavioral Evaluation Treatment Team) Miami Children's Hospital, Florida
1998- 2001: Executive Medical Director, Dan Marino Center Miami Children's Hospital
1991- 2004: Clinical Assistant Professor of Neurology University of Miami School of Medicine, Florida
2000- 2005: Professor of Pediatrics Nova Southeastern University, Florida
2002- 2009: Clinical Assistant (Visiting) Professor of Neurology Albert Einstein School of Medicine, NY

Selected publications

1. Tuchman R, Moshe SL, Rapin I. Convulsing toward the pathophysiology of autism. Brain Dev 2009;31:95-103.
2. Tuchman R. AEDs and psychotropic drugs in children with autism and epilepsy. Ment Retard Dev Disabil Res Rev 2004;10:135.
3. Tuchman R, Rapin I. Epilepsy in Autism. Lancet Neurol 2002;1:352-8.
4. Tuchman R, Rapin I. Regression in pervasive developmental disorders: seizures and epilepti form electroencephalogram correlates. Pediatrics 1997;99:560-6.
5. Tuchman R, Rapin I, Shinnar S. Autistic and dysphasic children: II Epilepsy. Pediatrics 1991;8:1219-25.

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12th ISS

Yoko KAWASAKI

Present Position

Director, Musashino Child Development Clinic,
Koganei, Tokyo, Japan

Appointments

1977-1980： Resident, Department of Neuropsychiatry, Tokyo University Hospital
1985-2004： Chief, Department of Child Psychiatry, Tama Habilitation Clinic for Handicapped Children
2004 - ： Present position

Selected publications

1. Kawasaki Y. Neurophysiological bases of pervasive developmental disorders. Jap J Clini Neurophysiol. 2006;34:142-51.
2. Kawasaki Y, Shinomiya M, Yumoto M, Hiramatsu K, Niwa S. Magnetoencephalographic localization of 'Paroxysm at F' in autism-Localization of frontal EEG paroxysms first seen between middle childhood and adolescence in autism. Jap J Clin Neuro physiol. 2001;29:262-8.
3. Kawasaki Y, MishimaT, Tamura M, Sakai K, Ino T, Murakami K, et al. Aberrant sensation and perception in PDD. Jap J Dev Disabil 2003;25:31-8.
4. Kawasaki Y, Yokota K, Shinomiya M, Shimizu Y, Niwa S. Brief report: Electroencephalographic paroxysmal activities in the frontal area emerged in middle childhood and during adolescence in a follow-up study of autism. J Aut Dev Disord 1997; 27:.605-20.
5. Kawasaki Y, Shimizu Y, Ota M. Lose of speech in setback autistics. Jap J Child Adoles Psy chiatr 1985;26:201-12.

Antonia PARMEGGIANI

Present Position

Chief of: the "Autism Center: research, diagnosis and therapy", the "Laboratory of Childhood and Adolescence Neuropsychology" and the "Child Neurology and Psychiatry Unit" of the Department of Neurological Sciences of the University of Bologna, Italy

Appointments

1986-1987: Visitor in the Laboratory of Evoked Potential of the Child Neurology and Psychiatry Unit of the University of Perugia
1989: Specialization in Child Neurology and Psychiatry with summa cum laude
1991: Technical Collaborator in Child Neurology and Psychiatry
1995-1997: Regional Secretary of the Italian Society of Childhood Neuropsychiatry
1998-2000: National Adviser of the Italian Society of Childhood Neuropsychiatry
1999: Researcher in Child Neurology and Psychiatry
2002: Associated Professor in Child Neurology and Psychiatry
2004-2007: Member of the Dean Advice of the Faculty of Medicine and Surgery
2005- : Director of the Specialization School of Childhood Neurology and Psychiatry

- o Adviser of AdDU Board, of Fa.Ce, ANGSA, ARSINPI Scientific Boards. Member of the reviewer board of Journal of Pediatric Neurology.
- o Professor teaching in degree courses: Medicine and Surgery, Ophtalmology Assistant, Logotherapy and Professional Education, Technical Science of Preventive and Adapted Mo tor Activity.
- o Professor teaching in Specialization Schools: Childhood Neurology and Psychiatry, Neurology, Clinical Psychology, Health Psychology.
- o Professor teaching in PhD Odontology for handicapped subjects.
- o Student Tutor in degree courses: Medicine and Surgery, Professional Education, Specialization Schools of Childhood Neurology and Psychiatry and recipient of fellowship.
- o Proposer and collaborator in university research works (FATMA, 40%, 60%, CNR, etc.).

Selected publications

1. Rossi PG, Parmeggiani A, Bach V, Santucci M, Visconti P. EEG features and epilepsy in patients with autism. Brain Dev 1995;17:169-74.
2. Rossi PG, Posar A, Parmeggiani A. Epilepsy in adolescents and young adults with autistic disorder. Brain Dev 2000;22:102-6.
3. Parmeggiani A, Posar A, Giovanardi-Rossi P, Andermann F, Zifkin B. Autism, macrocrania and epilepsy: how are they linked? Brain Dev 2002;24:296-9.
4. Parmeggiani A, Posar A, Antolini C, Scaduto MC, Santucci M, Giovanardi-Rossi P. Epilepsy in patients with pervasive developmental disorder not otherwise specified. J Child Neurol 2007;22:1198-203.
5. Parmeggiani A, Posar A, Scaduto MC. Cerebellar hypoplasia, continuous spike-waves during sleep, and neuropsychological and behavioural disorders. J Chil Neurol 2008;23:1472-6.

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12th ISS

Akihiro YASUHARA

Present Position

Director, Department of Pediatrics,
Yasuhara Children's Clinic and YCC Education Center,
Osaka, Japan

Appointments

1977-1978 : Resident in Department of Pediatrics, Kansai Medical University
1982 : Ph.D. Kansai Medical University
1987-1988 : Research Fellow at Neurology, School of Medicine, University of Iowa, Iowa, USA
1988-1993 : Assistant, Department of Pediatrics, Kansai Medical University
1993- : Assistant Professor, Kansai Medical University
1997-2005 : Director, Department of Pediatrics, Kansai Medical University Kohri Hospital
2005- : Associate Professor, Kansai Medical University
2006- : Present position

Selected publications

1. Yasuhara A, Yoshizaki Y, Yasuhara Y. Electroencephalography applications to child development disorder and epilepsia. Clini Electroencephalography 2008;50:216-20 (in Japanese).
2. Hijikata Y, Yasuhara A, Yoshida Y, Sento S. Traditional Chinese medicine treatment of epilepsy. J Altern Complement Med 2006;12:673-7.
3. Okuda K, Yasuhara A, Kamei A, Araki A, Kitamura N, Kobayashi Y. Successful control with bromide of two patients with malignant migrating partial seizures in infancy. Brain Dev 2000;22:56-9.
4. Yasuhara A, Ochi A, Harada Y, Kobayashi Y. Infantile spasms associated with a histamine H1 antagonist. Neuropediatrics 1998;29:320-1.
5. Yasuhara A, Yoshida H, Hatanaka T, Sugimoto T, Kobayashi Y, Dyken E. Epilepsy with continuous spike-waves during slow sleep and its treatment. Epilepsia 1991;32:59-62.

Masayuki SASAKI

Present Position

Head, Department of Child Neurology, National Center of Neurology and Psychiatry (NCNP), Japan

Appointments

1983-1988： Junior resident in Department of Pediatrics, Niigata University School of Medicine.
1988-1992： Resident in Department of Child Neurology, NCNP.
1992-1994： Visiting fellow, Laboratory of Molecular and Cellular Neurobiology, National Institute of Neurological Disorders and Stroke (NINDS), National Institute of Health (NIH), USA.
1994： Staff doctor, Department of Child Neurology, NCNP.
1996： Section Chief, Department of Child Neurology, NCNP.
2002： Present position

Selected publications

1. Sasaki M, Takanashi J, Tada H, Sakuma H, Furushima W, Sato N. Diffuse cerebral hypomyelination with cerebellar atrophy and hypoplasia of the corpus callosum. Brain Dev (In press)
2. Sasaki M, Sakuma H, Fukushima A, Yamada KI, Ohnishi T, Matsuda H. Abnormal cerebral glucose metabolism in alternating hemiplegia of childhood. Brain Dev 2009:31:20-6.
3. Sasaki M, Hashimoto T, Furushima W, Okada M, Kinoshita S, Fujikawa Y, Sugai K. Clinical aspects of hemimegalencephaly by means of a nationwide survey. J Child Neurol 2005;20: 337-41.
4. Ohnishi T, Matsuda H, Hashimoto T, Kunihiro T, Nishikawa M, Uema T, Sasaki M. Abnormal regional cerebral blood flow in childhood autism. Brain 2000;123:1838-44.
5. Sasaki M, Hanaoka S, Takashima S, Sakuragawa N, Arima M. MRI and CT findings in Krabbe disease. Pediatr Neurol 1991;7:283-8.

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Yoshiko NOMURA

Present position

Assistant director,
Segawa Neurological Clinic for Children, Tokyo, Japan

Education

1966 Graduated from Yokohama City University, School of Medicine, Yokohama, Japan

Post Graduate Training

1968 -1970 Completed Residency in Pediatrics, Mayo Clinic, Rochester, MN, U.S.A.
1970 -1971 Completed Fellowship in Pediatric Neurology, Children's Hospital of Washington DC, Washington DC, U.S.A.
1973 -1975 Completed Residency in Neurology, Georgetown University, Washington DC, U.S.A.

Appointments

1971 - 1973 Staff in Department of Pediatrics, Yokohama City University, School of Medicine, Yokohama, Japan
1975 - present Assistant Director, Segawa Neurological Clinic for Children, Tokyo, Japan

Licenses

1974 Commission on Licensure to Practice the Healing Art, Medicine and Surgery, Government of the District of Columbia, U.S.A.

Academic Appointments

1977 - 1998 Visiting Lecturer, Department of Pediatrics, Yokohama City University, School of Medicine, Yokohama, Japan
2000 - present Visiting Lecturer, Department of Pediatrics, Toho University,Tokyo, Japan

Selected publications

1. Nomura Y, Hachimori K, Nagao Y, Segawa M, Kimura K, Segawa M. Childhood myasthenia gravis in Japan - clinical analysis of 184 cases at Segawa Neurological Clinic for Children for 30 years. Neuro-Ophthalmology 2007;31:201-5.
2. Segawa M, Nomura Y. Pathophysiology of Autism: Evaluation of sleep and locomotion. In: Tuchman R, Rapin I, eds. Autism: A neurological disorder of early brain development. Interna tional Review of Child Neurology Series (ICNA). Mac Keith Press, London. pp.248-264, 2006.
3. Nomura Y. Disease entities with a temporary autistic phases; Autistic features of Rett syn drome. In: Coleman M, ed. The neurology of autism. Oxford University Press, New York. pp.136-156, 2005.
4. Nomura Y, Segawa M. Natural history of Rett syndrome. J Child Neurol 2005;20:764-8.
5. Nomura Y, Segawa M. The monoamine hypothesis in Rett syndrome. In: Kerr A, Engerström IW, eds. Rett Disorder and the Developing Brain. Oxford University Press, New York. pp.205-225, 2001.

Motomi TOICHI

Present Position

Professor, Associate Dean, Faculty of Human Health Science,
Graduate School of Medicine, Kyoto University, Kyoto, Japan

Appointments

1989-1991: Resident in Department of Neuropsychiatry, Kyoto University School of Medicine.
1992-1993: Regular staff in Division of Psychiatry, Obama Hospital (Fukui).
1999-2003: Ph.D., Clinical Psychophysiology (Kyoto University), Associate Professor, Health and Medical Services Center, Shiga University
2000-2002: Principal Investigator, Division of Child & Adolescent.Psychiatry, Case Western Reserve University/University Hospitals of Cleveland
2004: Professor, Faculty of Health Sciences, School of Medicine, Kyoto University
2007: Present position

Selected publications

1. Toichi M. Episodic memory, semantic memory and self-awareness in high-functioning autism. In Memory in Autism. Cambridge University Press, 2008
2. Sato W, Okada T, Toichi M. Attentional shift by gaze is triggered without awareness. Exp Brain Res 2007;183:87-94.
3. Toichi M, Findling RL, Kubota Y, Calabrese JR, Wiznitzer M, McNamara NK, et al. Hemodynamic differences in the activation of the prefrontal cortex. Neuropsychologia 2004;42:698-706.
4. Toichi M, Kamio Y. Paradoxical autonomic response to mental tasks in autism. J Aut Dev Disord 2003;33:417-26.
5. Toichi M, Kamio Y, Okada T Sakihama M, Youngstrom EA, Findling RL, et al. A lack of self-consciousness in autism. Am J Psychiatr 2002;159:1422-4.

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Amy BROOKS-KAYAL

Current Position

Professor of Pediatrics
Chief, Pediatric Neurology
University of Colorado at Denver School of Medicine
The Children's Hospital

Education

1984	BA	Cornell University, College of Arts and Sciences
1988	MD	Johns Hopkins University
1988-1991		Resident in Pediatrics, Children's Hospital of Philadelphia
1991-1992		Charles A. Dana Fellow in Neuroscience, Institute of Neuroscience
1992-1995		Resident in Child Neurology, Hospital of the University of Pennsylvania and Children's Hospital of Philadelphia

Academic Appointments

1993-1994	Assistant Instructor in Neurology, Department of Neurology, University of Pennsylvania School of Medicine
1994-1995	Instructor in Neurology, Department of Neurology, University of Pennsylvania School of Medicine
1995-2005	Assistant Professor of Neurology at the Children's Hospital of Philadelphia, University of Pennsylvania School of Medicine
2005-2008	Associate Professor of Neurology at the Children's Hospital of Philadelphia, University of Pennsylvania School of Medicine
2008-present	Professor of Pediatrics, University of Colorado Denver, School of Medicine

Hospital and/or Administrative Appointments

1995-2008	Medical staff, Children's Hospital of Philadelphia and Hospital of the University of Pennsylvania
2008-present	Chief, Division of Neurology, The Children's Hospital, Aurora, CO

Selected publications

1. Brooks-Kayal AR. Rearranging Receptors. Epilepsia 2005;46:29-38.
2. Guojun Zhang, Raol YH, Fu-Chun Hsu, Brooks-Kayal AR. Long-term alterations in glutamate receptor and transporter expression following early-life seizures are associated with increased seizure susceptibility. J Neurochem 2004;88:91-101.
3. Brooks-Kayal AR, Raol YH, Russek SJ. Alteration of Epileptogenesis Genes. Neurotherapeutics 2009 (in press)
4. Eric .Marsh, Brooks-Kayal AR, Porter BE. Seizures and Antiepileptic Drugs:Does Exposure Alter Normal Brain Development? Epilepsia 2006;47:1999-2010.
5. Zhang G, Raol YSH, HSU F-C, Coulter DA, Brooks-Kayal AR. Effects of Status epileptics on hippocampal GABAA receptors are age-dependent. Neuroscience 2004;125:299-303.

Leanne DIBBENS

Present position

MS McLeod Research Fellow, Department of Genetic Medicine, Women's and Children's Hospital, North Adelaide and Affiliate Lecturer, University of Adelaide, Australia

Appointments

1997: Ph.D. Adelaide University, Department of Genetics

1998-1999: Research Officer, Haematology, Hanson Centre for Cancer Research, Adelaide, Australia

2000: Present position

Selected publications

1. Dibbens LM, Tarpey PS, Hynes K, Bayly M, Scheffer IE, Smith R, et al. X-linked protocadherin 19 mutations cause female-limited epilepsy and cognitive impairment. Nat Genet 2008; 40: 776-81.
2. Berkovic SF, Dibbens LM, Oshlack A, Silver JD, Katerelos M, Vears DF, et al. Array based gene discovery with 3 unrelated subjects shows SCARB2/LIMP-2 deficiency causes myoclonus epilepsy and glomerulosclerosis. Am J Hum Genet 2008; 82: 673-84.
3. Dibbens LM, Feng HJ, Richards, MC, Harkin, LA, Hodgson, BL, Scott D, et al. GABRD encoding a protein for extra- or peri-synaptic GABAA receptors is a susceptibility locus for generalized epilepsies. Hum Mol Genet 2004; 13: 1315-19.
4. Harkin LA, McMahon JM, Iona X, Dibbens L, Pelekanos JT, Zuberi SM, et al. The spectrum of SCN1A-related infantile epileptic encephalopathies. Brain 2007; 130: 843-52.
5. Dibbens LM, Ekberg J, Taylor I, Hodgson BL, Conroy S-J, Lensink IL, et al. NEDD4-2 as a potential candidate susceptibility gene for epileptic photosensitivity. Genes, Brain Behav 2007; 6: 750-5.

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Toru TAKUMI

Present position

Professor, Laboratory of Integrative Bioscience,
Graduate School of Biomedical Sciences,
Hiroshima University, Japan

Appointments

1990	Ph.D. Kyoto University
1990-1991	JSPS fellow in Kyoto University Faculty of Medicine
1991-1993	HFSP fellow in Whitehead Insitute for Biomedical Research
1993-1996	Assistant Professor, Osaka University School of Medicine
1996-2001	Associate Professor, Kobe University School of Medicine
2001-2008	Lab Head, Osaka Bioscience Institute
2008	Present position

Selected publications

1. Nakamura W, Yamazaki S, Nakamura TJ, Shirakawa T, Block GD, Takumi T. In vivo real-time monitoring of circadian timing in freely moving mice. Curr Biol. 2008;18:381-5.
2. Yoshimura A, Fujii R, Wanatabe Y, Okabe S, Fukui K, Takumi T. Myosin-Va facilitates the accumulation of mRNA/protein complex in dendritic spines. Curr Biol. 2006;16:2345-51.
3. Fujii R, Okabe S, Urushido T, Inoue K, Yoshimura A, Tachibana T, et al. The RNA binding protein TLS is translocated to the dendritic spines by mGluR5 activation and regulates spine morphogenesis. Curr. Biol. 2005;15: 587-93.
4. Akashi M, Takumi T. The orphan nuclear receptor ROR regulates circadian transcription of the mammalian core-clock Bmal1. Nature Struct Mol Biol. 2005;12:441-8.
5. Takumi T, Ohkubo H, Nakanishi S. Cloning of a membrane protein that induces a slow voltage-gated potassium current. Science 1988;242:1042-5.

Thierry DEONNA

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Present Position

Honorary Professor for Neuropaediatrics, Faculty of Medicine, Lausanne.
Consultant Unite' de Neurologie et de Neurore'habilitation Pe'diatrique, CHUV, Lausanne, Switzerland

Education

Training in Paediatrics, Adult Neurology, Neuropathology and Paediatric Neurology in Boston, USA and in Switzerland
Former Chief and Founder Neuropaediatric Unit, University Children's Hospital, Lausanne, Switzerland

Has held several positions:
Comite' d'Ethique Acade'mie Suissse des Sciences Medicales
Membre du Jury du Prix Bing et Fondation Ott
Comite' Scientifique Association Suisse de Recherche sur Maladies Musculaires
Comite' Editorial: Neuropaediatrics, European Journal of Paediatric Neurology
Occasional Reviewer for Several Journals: Epilepsia, Dev.Med Child Neurology
Board Member of the Societe' de Neurologie Pe'diatrique, Europ. Fed. Child Neurology Societies

Selected publications

1. Deonna T. Annotation: cognitive and behavioural correlates of epileptic activity in children. J Child Psychol Psychiatry 1993;34:611-20.
2. Deonna T. Reflex seizures with somatosensory precipitation. Clinical and electroencephalo graphic patterns and differential diagnosis, with emphasis on reflex myoclonic epilepsy of infancy. Adv Neurol 1998;75:193-206.
3. Deonna T, Zesiger P, Davidoff V, Maeder M, Mayor C, Roulet E. Benign partial epilepsy of childhood: a longitudinal neuropsychological and EEG study of cognitive function. Dev Med Child Neurol 2000;42:595-603.
4. Deonna T, Roulet E. Autistic spectrum disorder: evaluating a possible contributing or causal role of epilepsy. Epilepsia 2006;47 (Suppl 2) : 79-82.
5. Cronel-Ohayon S, Zesiger P, Davidoff V, Boni A, Roulet E, Deonna T. Deficit in memory consolidation (abnormal forgetting rate) in childhood temporal lobe epilepsy. Pre and postoperative long-term observation. Neuropediatrics 2006;37:317-24.

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Jong-Hee CHAE

Present Position

Assistant Professor, Department of Pediatrics
Seoul National University Children's Hospital

Appointments

March 1992 - February 1993	Internship, Seoul National University Hospital
March 1993 - February 1997	Residency, Department of Pediatrics, Seoul National University Children's Hospital
March 1997 - February 1999	Clinical Fellow, Department of Pediatric Neurology, Seoul National University Children's Hospital
March 1999 - February 2000	Research Fellow, Department of Ultrastructural Research, NCNP, Japan
2002:	PhD, Seoul National University, College of Medicine, Seoul Korea
September 2005 - August 2006	Post Doc Fellow, Department of Neurology, College of Physician and Surgeon, Columbia University, NY
2002:	Present position

Selected publications

1. Chae JH, Lee JS, Kim KJ, Hwang YS, Hirano M. Biochemical and mutational analysis of Leigh syndrome in Korea. Brain Dev 2008;30:387-90.
2. Chae JH, Lee JS, Kim KJ, Hwang YS, Park JD. Merosin deficient congenital muscular dystrophy in Korea. Brain Dev 2008. (in press)
3. Chae JH, Lee JS, Kim KJ, Hwang YS, Bonilla E, Tanji K, et al. A Novel ND3 Mitochondrial DNA Mutation in Three Korean Children with Basal Ganglia Lesions and Complex I Deficiency. Ped Res 2007;61: 622-4.
4. Chae JH, Hwang H, Hwang YS, Kim KJ. Influence of MECP2 gene mutation type and X chromosome inactivation on the phenotype of Rett syndrome. J Child Neurol 2004;19:503-8.
5. Chae JH, Hwang YS, Kim KJ. Mutation analysis of MECP2 and clinical characterization in Korean patients with Rett syndrome. J Child Neurol 2002;17:33-6.

Michele ZAPPELLA

Present Position

Consultant in the Centre for Rett syndrome in the Hospital Versilia, Tuscany, Italy
Teaching staff in Child Neuropsychiatry in University of Siena, Viareggio, Italy

Education and Appointments

1960:	Degree in Medicine in the University of Rome with Maxima Cum Laude
1961- 1964:	trained in Child Neurology working in London(UK) in the Fountain Hospital, a Hospital for Children with Brain Damage, subsequently in the EEG Section of the Children's Hospital of the University of Rome
1964- 1965:	Fellow in Neurology, Department of Child Neurology of the Children's Hospital, Washington DC, USA
1966:	Diploma in Child Neuropsychiatry
1968:	Diploma in Mental and Nervous Diseases
1969-1973:	Consultant in Different Psychiatric Hospitals (Volterra, Arezzo, Novara)
1973-2006:	Director of the Division of Child Neuropsychiatry of the General Hospital of Siena, Italy

Selected publications

1. Zappella M. Early-onset Tourette syndrome in children with reversible autistic behaviour: a dysmaturational disorder. Eur Child Adolesc Psychiatry 2002;11:18-23.
2. Canitano R, Zappella M. Autistic epileptiform regression. Funct Neurol 2006;21:97-101.
3. Canitano R, Luchetti A, Zappella M. Epilepsy, electroencephalographic abnormalities, and regression in children with autism. J Child Neurol 2005;20:27-31.
4. Zappella M. The Rett girls with preserved speech. Brain Dev 1992;14:98-101.
5. Renieri A, Mari F, Mencareli MA, Scala E, Ariani F, Longo I, et al. Diagnostic criteria for the Zappella variant of Rett syndrome (preserved speech syndrome) Brain Dev 2009;31:208-16.

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Hitoshi HARA

Present Position:

Director General, Yokohama Central Area Habitation Center for Children Yokohama, JAPAN

Education:

1976: M.D. School of Medicine, Chiba University

1986: Ph.D. Department of Pediatrics, Tokyo Women's Medical College

Appointments

1986-2002: Assistant Professor, Department of Pediatrics, Tokyo Women's Medical College

1986-1994: Section Chief, Department of Developmental Disorders, National Institute of Mental Health, National Center of Neurology and Psychiatry

1994-2002: Director, Department of Education for Children with Health Impairments, National Institute of Special Education

2002-: Present Position

Selected Publications

1. Hara H, Fukuyama Y. Sustained attention during the interictal period of mentally normal children with epilepsy or febrile convulsions, and the influence of anticonvulsants and seizures on attention. Jap J Psychiatr Neurol 1989;43:411- 6.
2. Hara H. Sustained attention in mentally normal children with convulsive disorders. In: Suzuki J, Seino M, Fukuyama Y, Konami S, eds. Art and Science of Epilepsy. Elsevier Science Publishers B.V., Amsterdam. pp 123-126, 1989.
3. Hara H, Fukuyama Y. Partial imitation and partial sensory agnosia in mentally normal children with convulsive disorders. Acta Paediatr Jap 1992;34:416-25.
4. Hara H, Sasaki M. Autistic syndrome and epilepsy: a comparison between the children with epileptic seizures and only with epileptiform EEG abnormalities. In. Naruse H & Ornitz ED eds. Neurobiology of Infantile Autism. Excerpta Medica, Amsterdam. pp.201-202,1992.
5. Hara H. Autism and epilepsy: a retrospective follow-up study. Brain Dev 2007;29:486-90.

International Symposia in Past 5 Years Organized by Infantile Seizuire Society (ISS), Japan

Year	Theme	Invited Faculties (Japanese excluded)	Publications
2004	Neuronal Migration Disorders and Childhood Epilepsies	Battaglia GS, Crino PB, Curatolo P, Golden JA, Granata T, Guerrini R, Mochida GH, Otsubo H, Woermann FG	Jouranal of Child Neurology 2005; 20(4):273-397
2005	Epileptic Syndromes in Infancy and Early Childhood	Andermann F, Berg AT, Capovilla G, Craiu D, De Vivo DC, Engel J Jr, Fejerman N, Hirsch E, Kasteleijin-Nolst Trenite DGA, Kim DW, Lee WL,Lux AL, Moshe SL, Nordli DR Jr, O'Regan ME, Plouin P, Sankar R, Scheffer I, Scher MS, Specchio N, Wolf P	Epilepsy Reserch 2006; 60: Suppl 1: S1-S279
2006	Status Epilepticus in Infants and Young Children	Banu S, Fusco L, Kalra V, Lee JS, Lux AL, Neville B, Otsubo H, Sanker R, Shinnar S, Specchio N, Wasterlain CG	Acta Neurologica Scandinavica Supplementum 2007; vol. 115 Suppl 186
2007	Biology of Seizure Susceptibility	Arzimanoglou A, Baram TZ, Curatolo P, Goldin AL,Mochida GH, Moshe SL, Otsubo H, Prasad A,Sarnat HB, Schridde U, Staley KJ, Swann JW	Takahashi T, Fukuyama, Y eds. Biology of Seizure Susceptibility in Developing Brain. Progress in Epileptic Disorders, Vol 6. Montrouge: John Libbey Eurotext. 2008: 232 pp.
2008	Febrile Seizures and Related Conditions	Vestergaard M, Neville BGR, Kaila K, Nordli D, Shinnar S, Baram TZ, Chang YC, Heida JG, Kubek MJ, Scheffer IE	Brain and Development, 2009 part 1 (in press) part 2 (in preparation)

The 13th Annual Meeting of the Infantile Seizure Society (ISS)
International Symposium on Epilepsy in Neurometabolic Diseases (ISENMD)

Dear Sir/Madam,

Greetings from Taipei. It is our pleasure to tell you that the **International Symposium on Epilepsy in Neurometabolic Diseases (ISENMD)** and **The 13th Annual Meeting of the Infantile Seizure Society (ISS)** will be held in **Howard Plaza Hotel Taipei, Taiwan from March 26-28, 2010.**

Infantile Seizure Society is the world leading organization whose aim is to promote clinical and research on seizures in early life. In the coming symposium, we will invite specialists and researchers to give up-to-date and in-depth discussion in this field. Audience is always a key sector of a conference, thus we also hope to gather those who are interested and dedicate themselves in this field to come together at this platform.

A successful event can not be achieved without your generous help. We are obliged that you can help to spread and post our conference information on the web page of your organization.

International Symposium on Epilepsy in Neurometabolic Diseases (ISENMD)
The 13th Annual Meeting of the Infantile Seizure Society
Theme: Epilepsy in Neurometabolic Diseases
Dates: March 26-28, 2010
Venue: Howard Plaza Hotel, Taipei, Taiwan
Contact E-mail: isenmd2010@knaintl.com.tw
For more information, kindly visit http://www.isenmd2010taipei.org

The 2nd announcement will soon be available, and the secretariat will send it to you for reference. Should you need further information, please contact directly to our conference secretariat at the attention below. Once again we thank you for your kind assistance.

Yours sincerely,

Ein-Yiao Shen,MD
President
Taiwan Child Neurology Society and International Symposium on Epilepsy in Neurometabolic Diseases (ISENMD)

Secretariat of the International Symposium on Epilepsy in Neurometabolic Diseases (ISENMD)
& The 13th Annual Meeting of the Infantile Seizure Society
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