Introduction and Neuroimaging predictors of AED resistance in new-onset epilepsies

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Studies on prognosis in epilepsy

the risk factors for an adverse outcome

- a young age at onset,
- more seizures prior to the first visit to a physician,
- presence of a lesion on neuroimaging,
- longer duration of illness

Sillanpaa & Schmidt, 2006, 2009a,b; Geerts et al., 2010
The role of neuroimaging

- The limitations of imaging diagnosis
  - Imaging tool is not the Almighty
  - Needs clinical correlation
    - Semiology, EEG, Scintigraphy studies
- The roles of neuroimaging in epilepsy
  - **Preop evaluation**
    - Locate and define anatomic epileptogenic lesion
    - Surgical resectability
    - Type and extent of epilepsy surgery
    - Assess need for grid insertion or functional mapping of eloquent region of brain
  - **Postop evaluation**
    - Determine adequacy of resection, reasons for op failure
    - Complications, tumor recurrence
Routine vs. Epilepsy Dedicated MRI for Epileptic Brain Lesion

- Routine MRI before 1994
  - In temporal lobe: 55%
  - In extratemporal lobe: 43%
- Epilepsy dedicated MRI for pathologically proven partial seizure
  - 74 to 86% of sensitivity

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Figure 1: An adolescent female presented with complex partial seizures. MRI was suggestive of left mesial temporal sclerosis. High resolution (HR) T2-weighted image (a) showing T2 hyperintensity involving the hippocampal body. Note that signal intensity disturbances are better appreciated on HR T2 and volume loss is better appreciated on 3D FLASH (b).

Chinchure S, et al, Neurol India, 2010
Proton magnetic resonance spectroscopy (MRS) in patients with TLE responded to the first AED (responders) failed to respond to AEDs (failure-group).

Figure 1.
Region of interest (ROI) in both temporal lobes. Each ROI includes part of the hippocampus and adjacent white matter.

Campus BA et al, Epilepsia, 2010
**Figure 2.**
Proton magnetic resonance spectroscopy ($^1$H-MRS) of hippocampal region in control group and ipsilateral to electroencephalography (EEG) in responders and failure groups.

*Epilepsia © ILAE*

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**Figure 3.**
N-Acetylaspartate/creatinine (NAA/Cr) ratios ipsilateral to electroencephalography (EEG) focus. Tukey’s post hoc pairwise comparisons showed differences between the first antiepileptic drug (AED) failure group versus controls ($p < 0.001$) and responders group ($p = 0.001$).

*Epilepsia © ILAE*

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*Campus BA et al, Epilepsia, 2010*
Hippocampal volumetry and T2 relaxometry

- Increased signal and volume loss can be assessed quantitatively or by visual inspection.
- With experience, visual inspection of the hippocampus will reliably detect hippocampal asymmetry of more than 20%.
- A smaller degree of asymmetry is, however, best detected by volumetric quantification.
- Jackson et al. have shown that quantitative evaluation of signal is a sensitive (70%) and reliable method for lateralizing HS.
  
  Jackson GD et al., Neurology, 1993

- Quantitative MR volume studies have increased the detection rate and reliability for diagnosing pathologically proved or probable HS, sensitivity 74-96%, and specificity 73-100%.

  Cascino G et al., Ann Neurol, 1991
T2 relaxometry were strong indicators of seizure recurrence

**Figure 1.** Survival analysis demonstrating a difference in seizure recurrence between patients with or without hippocampal atrophy (HA) (Mantel, p = 0.006). The probability of remaining seizure free 5 years after antiepileptic drug withdrawal was 62% for those without HA and 28% for those with HA. HA− = patients without HA; HA+ = patients with HA.

**Figure 2.** Survival analysis demonstrating a difference in seizure recurrence between patients with normal or abnormal T2 relaxation time (Tarone-Ware, p = 0.013). The estimated probability of remaining seizure free at 5 years after antiepileptic drug withdrawal was 62% for those with normal hippocampal signal and approximately 23% for those with abnormal hippocampal signal.

*Cardoso et al., 2006, Neurology*
Figure 2: A patient with left mesial temporal sclerosis. High-resolution (HR) T2 coronal image (a) showing T2 prolongation and atrophy involving left hippocampal head. There is also evidence of collateral white matter atrophy on the left side. Proton spectroscopy metabolic map (b) shows reduced NAA (N-acetyl aspartate) in left hippocampal head and body. Spectroscopy map (c) from a normal subject shows similar NAA on both sides. (d) A young female patient with temporal lobe epilepsy and right mesial temporal sclerosis. HR T2-weighted image (d) showing signal changes involving right hippocampal head. Atrophy is appreciated in 3D SPGR. (e) Apparent diffusion coefficient (f) in the right hippocampus is high, secondary to gliosis (arrow).

Chinchure S, et al, Neurol India, 2010
### Table 2. MRI features of cortical dysplasias

<table>
<thead>
<tr>
<th>Cortex</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Increased signal on $T_1$</td>
<td></td>
</tr>
<tr>
<td>Increased signal on $T_2$/FLAIR</td>
<td></td>
</tr>
<tr>
<td>Thickened</td>
<td></td>
</tr>
<tr>
<td>Focal hypoplasia/atrophy</td>
<td></td>
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<tr>
<td>Broadening of gyri</td>
<td></td>
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<tr>
<td>Deep sulcus</td>
<td></td>
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<tr>
<td>Gray–white matter junction</td>
<td></td>
</tr>
<tr>
<td>Blurred on $T_2$/FLAIR</td>
<td></td>
</tr>
<tr>
<td>Blurred on $T_1$</td>
<td></td>
</tr>
<tr>
<td>White matter</td>
<td></td>
</tr>
<tr>
<td>Tapering of abnormal white matter signal from cortex to ependymal surface of ventricle ($T_2$/FLAIR hyperintense, sometimes $T_1$ hypointense)</td>
<td></td>
</tr>
<tr>
<td>Diffuse white matter $T_2$/fluid attenuated inversion recovery (FLAIR) hyperintensity</td>
<td></td>
</tr>
<tr>
<td>Atrophy</td>
<td></td>
</tr>
</tbody>
</table>
Fig. 3. MRI features of focal cortical dysplasia

Widdess-Walsh P et al, 2005
Figure 4.
Imaging at 3T and 7T with 32-channel phased array coils in a patient with new-onset seizures, including axial refocussed matted images from a sagittally acquired $T_1$-weighted magnetization prepared rapid gradient echo (MPRAGE) at 3T (A) and at 7T (B), axial $T_2$ turbo spin echo (TSE) images at 3T (C) and 7T (D), and axial $T_2^*$ fast low angle shot (FLASH) (E), demonstrates a cortical dysplasia (arrowheads). Although this lesion is visualized at both 3T and 7T, it is hoped that the improved signal, contrast, and resolution will help identify more subtle cortical dysplasias in the future. The axial $T_2^*$ FLASH image demonstrates radial low-signal cortical vessels (thin arrows) and low-signal subcortical U fibers in normal regions that are disrupted in this area of dysplasia. In some normal regions additional intracortical structure with iron-rich layers can be seen.

Mandan N, grant PE, 2009
Diffusion Tensor Imaging (DTI)

- DTI is a novel technology that can depict orientation and integrity of WM fiber tracts in vivo

Molecular diffusion in white matter

- In CSF (water)
  - Brownian motion
  - Random diffusion
  - Isotropic diffusion

- In white matter
  - Directional motion
  - Restricted diffusion
  - Anisotropic diffusion

Mean diffusivity

0  isotropic  1  anisotropic
Diffusion Tensor Imaging

Fractional Anisotropy

Color Coded Vector Map

CST, MLF, MCP, EC, PLIC, PTR, CC, ALIC, CR, SLF

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Combining DTI data with the computational methods of MR tractography, neuroscientists can estimate the locations and sizes of nerve bundles (white matter pathways) that course through the human brain.

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DTI in Cortical Dysplasia

FLAIR  PET  FA  Tractography

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Figure 5: A young child with complex partial extratemporal seizures. Focal cortical thickening with focally blurred gray-white differentiation is well appreciated on FLAIR (a) T2-weighted (b) T1-weighted inversion recovery (c) Images in the parieto-occipital region. Cortical thickening is also appreciated on 3D spoiled gradient recalled (d) Curved planer reformation (e) Can help in revealing the real extent of the lesion (arrow). It can also help in picking up subtle lesions.

Chinchure S, et al, Neurol India, 2010
Advanced imaging techniques

- Advanced imaging techniques, such as tissue classification and segmentation, voxel-based morphometry, texture analysis, cortical and sulcal morphometry, may increase the sensitivity in detection of subtle abnormalities.
- Computer-aided tissue texture detection technique using complex diffusion approach can help to detect focal cortical dysplasia.
VBM – case analysis in lesional epilepsy

patient with right sided polymicrogyria

IS THERE SUCH A THING AS NONLESIONAL EPILEPSY? 
Unveiling epileptogenic lesions: The contribution of image processing

Figure 1.
Example of a 22-year-old patient with drug-resistant new-onset frontal lobe epilepsy. Seizures were clinically characterized by a diffuse numbness sensation, followed by head and eyes turning toward the left, and by elevation of the left arm. During prolonged video-EEG telemetry with scalp electrodes no epileptic spikes were recorded, only intermittent diffuse slow waves. All clinical seizures were associated with diffuse EEG changes, without focal electrographic anomalies. Since the onset of the epileptic disorder 4 years prior to admission, the MRI was repeatedly reported as normal. On the other hand, image processing revealed increased cortical thickness and blurring in the right frontal region (rectangle on images). The stereo-EEG (SEEG) study guided by these results confirmed that all seizures were originated in the lesional area. The patient presently awaits surgery.

Bernasconi A, Bernasconi N, Epilepsia, 52(Suppl. 4):20–24, 2011

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Figure 10: Algorithm for epilepsy imaging

Clinical assessment (semiology) and EEG

- Focal
  - Temporal
  - Extratemporal

- Generalized
  - Symptomatic
  - Idiopathic

MR Sequences:
- T1, T2, PD Axials
- FLAIR Axial, coronal
- DWI, SWI/T2*GRE
- T2 HR Coronal
- T2 Sagittal
- 3D FLASH/SPGR Coronal

MR Sequences:
- T1, T2, PD Axials
- FLAIR Axial
- SWI/T2*GRE
- T2 HR Coronal
- T2 Sagittal
- 3D FLASH/SPGR
- 3D FLAIR
- DTI

Negative / Equivocal
- T2 Relaxometry
- DTI
- MRS
- Volumetry

Positive
- fMRI in presurgical cases

Positive
- Small FOV/ phased array
- Postprocessing:
  - Texture analysis
  - Curvilinear reconstruction
  - Contrast if needed

MR Negative

SPECT/PET coregistered with MR

Chinchure S, et al, Neurol India, 2010
Functional Neuroimaging in Epilepsy

- Positron Emission Tomography
- SPECT
- MR spectroscopic imaging
- Functional MRI
- Magnetoencephalography

Main goals:
1. Identification and delineation of epileptogenic areas
2. Delineation of eloquent areas not to be resected
Blood oxygenation level-dependent functional MRI

Figure 6: An adolescent with focal cortical dysplasia involving precentral gyrus on the right side. (a) Real-time fMRI coregistered on axial, sagittal, and coronal T1-weighted images obtained after left finger tapping vs rest shows activation of primary hand motor area placed close to the lesion. If resection extends to primary hand motor area, the patient is likely to develop postprocedure neurologic deficit.

Chinchure S, et al, Neurol India, 2010
Positron Emission Tomography and Epilepsy

- Has been applied for presurgical evaluation since the 1970s.

- Has now been largely superseded by high-quality MRI - 80%


- 20-25% of pts with refractory focal epilepsy and unremarkable MRI scans
  → Intracranial grid placement
PET and SPECT

- PET studies
  TLE - hypometabolism ipsilateral to ictal onset zone in 87%

- Ictal SPECT.
  sensitivity in 90% for TLE
  81% for ETLE

- PET or ictal SPECT may be a complementary tool in the presurgical evaluation of patients with medically intractable epilepsy, particularly with negative MRI.

- Recently, newer techniques of PET imaging with different neuroreceptor ligands, serotonin, GABA, opioid, and dopamine, are under investigation to evaluate the neurochemical basis of epilepsy and the role of these neurotransmitters in the seizure propagation
Correct localisation: concordance between PET localisation and pathological diagnosis, or pathological diagnosis and the surgical site

Median percentage: 80% (62-100%)

Table 15  Correct lateralisation or localisation of seizure focus by PET, and proportion of PET positive patients with good outcomes in patients with medically refractory epilepsy

<table>
<thead>
<tr>
<th>Study</th>
<th>n</th>
<th>Epilepsy Type</th>
<th>PET Interpretation</th>
<th>Correct Lateralisation/Localisation % (99% CI)</th>
<th>PET positive with good outcomes % (95% CI)</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tatidil et al 2000</td>
<td>19</td>
<td>Temporal lobe epilepsy</td>
<td>ROI (asymmetry indices)</td>
<td>62.5 (±21.8)</td>
<td>38.5 (±21.9)</td>
<td>Outcomes classified as seizure-free, significantly improved (&lt; 3 seizures per yr and 90% reduction), and not significantly improved.</td>
</tr>
<tr>
<td>Juhasz et al 2003</td>
<td>12</td>
<td>Extratemporal lobe epilepsy</td>
<td>Semi-automated software package</td>
<td>75.0 (±24.5)</td>
<td>85.7 (±19.8)</td>
<td></td>
</tr>
<tr>
<td>Won et al 1999</td>
<td>13</td>
<td>Temporal and extratemporal lobe epilepsy</td>
<td>Visual</td>
<td>84.6 (±19.6)</td>
<td>-</td>
<td>Unable to calculate proportion of PET+ pts with good outcome. Cannot determine numbers of TLE/ETLE pts.</td>
</tr>
<tr>
<td>Murphy et al 2004</td>
<td>10</td>
<td>Partial epilepsy</td>
<td></td>
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<tr>
<td></td>
<td></td>
<td>Temporal lobe</td>
<td></td>
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<tr>
<td></td>
<td>9</td>
<td>Extratemporal</td>
<td></td>
<td></td>
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<td></td>
<td>1</td>
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</tbody>
</table>

## Seizure Outcomes

### Table 16  Positive seizure control outcomes for studies using PET in the pre-surgical evaluation for medically refractory epilepsy patients

<table>
<thead>
<tr>
<th>Study</th>
<th>n</th>
<th>Epilepsy Type</th>
<th>Follow-up</th>
<th>Engel’s I or II % (95% CI)</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Kim et al 2000</td>
<td>14</td>
<td>Temporal and extratemporal lobe epilepsy</td>
<td>≥1 yr</td>
<td>28.6 (±23.7)</td>
<td>Includes 2 patients who did not have PET. Only 2 pts with temporal lobe epilepsy</td>
</tr>
<tr>
<td>Totidlo et al 2000</td>
<td>19</td>
<td>Temporal lobe epilepsy</td>
<td>1-5 yrs</td>
<td>42.1 (±22.2)</td>
<td>“Seizure-free” patients (not reported as Engel’s class outcomes).</td>
</tr>
<tr>
<td>Hwang et al 2001</td>
<td>36</td>
<td>Neocortical epilepsy (temporal and extratemporal)</td>
<td>Mean of 34 mo (range 12-82)</td>
<td>61.1 (±15.9)</td>
<td>Includes 16 temporal and 20 extratemporal (11 frontal, 9 occipital) lobe epilepsy pts.</td>
</tr>
<tr>
<td>Won et al 1996</td>
<td>26</td>
<td>Temporal and extratemporal lobe epilepsy</td>
<td>Mean of 24 mo (range 12-35)</td>
<td>65.5 (±18.3)</td>
<td>Includes 6 patients who did not have PET. Includes 14 temporal and 12 extratemporal (7 frontal, 3 occipital, 2 multifocal) lobe epilepsy pts.</td>
</tr>
<tr>
<td>Juhasz et al 2003</td>
<td>12</td>
<td>Extratemporal lobe epilepsy</td>
<td>Mean of 17.2 mo (range 2-40)</td>
<td>67.0 (±28.8)</td>
<td></td>
</tr>
<tr>
<td>Salanova, Markand, &amp; Worth 2001</td>
<td>6</td>
<td>Temporal lobe epilepsy</td>
<td>Range of 2-8 yrs</td>
<td>67.0 (±37.6)</td>
<td>“Seizure-free” patients (not reported as Engel’s class outcomes). Included as n=18 for localisation results.</td>
</tr>
<tr>
<td>Juhasz et al 2000</td>
<td>12</td>
<td>Neocortical epilepsy (temporal and extratemporal)</td>
<td>Mean of 15.3 mo (±5.3)</td>
<td>75.0 (±24.5)</td>
<td>Includes 3 temporal and 9 extratemporal (5 frontal, 3 central, 1 multifocal) lobe epilepsy pts.</td>
</tr>
<tr>
<td>O’Brien et al 2001</td>
<td>24</td>
<td>Temporal and extratemporal lobe epilepsy</td>
<td>Median of 17 mo (range 6-42)</td>
<td>91.7 (±11.0)</td>
<td>Only 2 pts with extratemporal lobe epilepsy.</td>
</tr>
<tr>
<td>Murphy et al 2004</td>
<td>10</td>
<td>Temporal and extratemporal lobe epilepsy</td>
<td>Mean of 26 mo (range 14-41)</td>
<td>100.0 (±23.7)</td>
<td>Only 1 pt with extratemporal (frontal) lobe epilepsy.</td>
</tr>
</tbody>
</table>

- Median percentage: 67% (29-100%)

Case 6, M/14, CPS

FDG PET: RAT↓, RMT↓  MRI: Normal

Pathology: Rt. hippocampal sclerosis grade 3
KJY, M/8Y8M

- Diagnosis: CPS
- OP: Rt. frontal lobectomy
- Pathology: FCD2B
- Outcome: 1A
The usefulness of FDG PET for presurgical localization in non-lesional pediatric epilepsy patients

- PET detection rate in non-lesional patients: 71% (25/35)
  - ETLE: 72% (13/18)
  - TLE: 70% (5/7)

- Pathologic Matching:
  - Abnormal: 88% (22/25)
    - FCD 14, Microdysgenesis 4, HS 1, Leukomalacia 1, Low grade glioma 1, gliosis 1
  - Normal: 3

Limitations of FDG PET in Presurgical Evaluation

- Does not precisely localize the neocortical focus
- Extent of hypometabolism does not reflect tissue to be resected to achieve seizure freedom
Receptor PET Scanning in Epilepsy

- $\text{GABA}_A$ / central benzodiazepine receptors (Flumazenil)
- Alpha methy tryptophan receptors
- Peripheral benzodiazepine receptors
- Opioid receptors
- Dopamine receptors
- Acetylcholine receptors
- Serotonin and $5$-$\text{HT}_{1A}$ receptors
[\textsuperscript{11}C]Flumazenil PET: Applications in Epilepsy

Decreased flumazenil binding may occur in:

- Medial temporal sclerosis
- Dual pathology
- Seizure onset zone in non-lesional extratemporal lobe epilepsy

Detection at 71-84%, surgically useful at 26-46%

Koepp MJ, Neurology, 2000
<table>
<thead>
<tr>
<th>FDG</th>
<th>FMZ</th>
</tr>
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</table>

Area of decreased flumazenil binding is smaller than glucose hypometabolism.
Focal decrease of cortical flumazenil binding in neocortical epilepsy

14 years old girl
MRI: normal
Ictal EEG: right frontal seizure onset
Use and Limitations of Flumazenil PET

- FMZ PET abnormalities are more specific for epileptogenic areas than FDG PET hypometabolism, thus, normal FMZ binding can occur occasionally.

- None of these tracers are very useful in epilepsies associated with *multifocal lesions*.
AMT PET in Tuberous Sclerosis

EEG: spike and wave activity in the right frontal region.

Detection of cortical dysplasia by alpha\textsuperscript{[11C]}-methyl-L-tryptophan (AMT) PET

MRI

FDG PET

AMT PET

Children’s Hospital of Michigan
Wayne State University, Detroit
Clinical and histopathologic correlates of \(^{11}\text{C-}\alpha\text{-methyl-\text{L-tryptophan}}\) (AMT) PET abnormalities in children with intractable epilepsy

*†Harry T. Chugani, ‡‡Ajay Kumar, §William Kupsky, ‡‡Eishi Asano, ¶Sandeep Sood, and *†Csaba Juhász

Departments of *Pediatrics, †Neurology, ‡Radiology, §Pathology, and ¶Neurosurgery, Children’s Hospital of Michigan, Detroit Medical Center, Wayne State University School of Medicine, Detroit, Michigan, U.S.A.

Table 1. Demographic and clinical profile

<table>
<thead>
<tr>
<th>Characteristics</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total number of children</td>
<td>30</td>
</tr>
<tr>
<td>Gender</td>
<td>17 males; 13 females</td>
</tr>
<tr>
<td>Age at seizure onset (^a)</td>
<td>1.4 ± 1.1 years</td>
</tr>
<tr>
<td>Age at AMT scan (^a)</td>
<td>6.7 ± 4.3 years</td>
</tr>
<tr>
<td>Seizure duration (^a)</td>
<td>5 ± 3.5 years</td>
</tr>
<tr>
<td>Seizure frequency before AMT scan(^b)</td>
<td>≤1/week (21.0%); 2–7/week (12.5%); 1–5/day (45.8%); &gt;5/day (20.8%)</td>
</tr>
<tr>
<td>AMT scan</td>
<td>1–5/day (45.8%); &gt;5/day (20.8%)</td>
</tr>
<tr>
<td>Seizure type</td>
<td>Past or present history of infantile spasms (46.7%); Partial or complex partial seizure (30.1%); Secondary generalization (23.2%)</td>
</tr>
<tr>
<td>Histopathology</td>
<td>MCD 16 (53%) (12 CD, 3 PMGH &amp; 1 SHE)</td>
</tr>
<tr>
<td>Surgery outcome</td>
<td>Engel’s class I–II; II–III; III–IV; IV–V</td>
</tr>
<tr>
<td>Duration of postsurgical follow-up (^e)</td>
<td>8.7 ± 1.8 years</td>
</tr>
</tbody>
</table>

AMT, \(^{11}\text{C-}\alpha\text{-methyl-\text{L-tryptophan}}\); MCD, malformation of cortical development; CD, cortical dysplasia; PMGH, polymicrogyria and heterotopias; SEH, subependymal heterotopias. \(^a\)Values are given as mean ± SD.

Figure 1.
AMT-PET scan (left) showing increased tracer uptake in left parietal lobe in an 8-year-old girl with intractable seizures and normal MRI (middle). Postsurgical histopathology revealed cortical dysplasia type-IIB with balloon cells (right; 40×, H&E). The child is seizure free for 6 years after surgery.

Epilepsia © ILAE

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AMT PET Can Detect “FDG-negative” Epileptic Foci
Abnormal Nicotinic ACh Receptor Binding in ADNFLE (autosomal dominant nocturnal frontal lobe epilepsy)

Picard et al., Brain, 2006

ADNFLE: mutations in the nAChR alpha4 or beta2 subunit, which compose the main cerebral nAChR

PET with $^{18}$F-F-A-85380 (a high affinity agonist at the alpha4beta2 nAChR)

A: Normal control  
B: Increased binding: midbrain, pons, cerebellum (+12-21%) 
Decreased binding: dorso prefrontal cortex
Multimodal approach
Case. M/11 yr 11mo.
Lt. Temporal lobe epilepsy

MRI

FDG - PET

SPECT

SPM

P < 0.001

Department of Pediatric Neurology
Yonsei University College of Medicine
10% asymmetric marked, surface rendered 3D FDG PET
VerbG > REST
FEW
p < 0.001, cluster 50

Language task: Lt. dominancy
Sturge–Weber syndrome: Correlation between clinical course and FDG PET findings

Neurology 2001;57:189
DOI 10.1212/WNL.57.2.189

This information is current as of June 7, 2012
Figure 1. (A) Conceptual illustration of cortical areas classified based on the asymmetries of glucose metabolism. (B) Surface-rendered images of a FDG PET scan show regions that correspond to the areas of hypometabolism marked by the semiautomatic program for detecting cortical asymmetries. Regions of interest (solid black lines) were defined manually on the marked cortical areas according to the asymmetry index.

Figure 2. The frequency of seizures showed a significant correlation with the extent of mildly asymmetric cortical metabolic region (10% to 20% decrease; \( r = 0.63; \ p = 0.027 \)) (A). No similar correlation was found for the severely asymmetric cortical metabolic cortex (\( >20\% \) decrease; \( r = 0.06; \ p = 0.4 \)) (B). Note that the seizure frequency is displayed on a logarithmic scale in this figure, but statistical results are based on the nonparametric rank correlation of Spearman.
Sturge-Weber Syndrome: Rapid Progression of Severe Hypometabolism - Good Cognitive Outcome

Age 5 months  38 months  5 years

Lee JS et al 2001, Neurology
Sturge-Weber Syndrome

Case 1. 20% asymmetry
Frequent seizures,
FSIQ = 48

Case 2. 20% asymmetry
Rare Seizures
FSIQ = 70

Lee JS et al. Neurology 2001

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Conclusion

- Combining imaging modalities (PET, MRI) facilitates an enhanced spatial definition of functional brain abnormalities and may have a great impact to the enhanced delineation of cortical regions to be resected.

- Modern functional neuroimaging techniques advances in our understanding of the basic pathophysiologic process associated with the epilepsies and correct diagnosis in pediatric neurology.

- Combining imaging modalities (PET, MRI) measure may be potential surrogate markers for predicting AED response in near future.
Thank you for your attention!!